REVIEW ARTICLE

The basal ganglia and apraxia

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Summary

Ever since Liepmann’s original descriptions at the beginning of the century, apraxia has usually been attributed to damage confined to the cerebral cortex and/or cortico-cortical connecting pathways. However, there have been suggestions that apraxia can be due to deep subcortical lesions, which raises the question as to whether damage to the basal ganglia or thalamus can cause apraxia. We therefore analysed 82 cases of such ‘deep’ apraxias reported in the literature. These reports consisted of a small number (n = 9) of cases studied neuropathologically, and a much larger group (n = 73) in which CT or MRI was used to identify the size and extent of the lesion. The reports were subdivided into (i) those with small isolated lesions which involved nuclei of the basal ganglia or thalamus only, and not extending to involve periventricular or peristriatal white matter; (ii) those with large lesions which involved two or more of the nuclei, or one or more of these deep structures plus damage to closely adjacent areas including the internal capsule, periventricular or peristriatal white matter; and (iii) lesions sparing basal ganglia and thalamus but involving adjacent white matter. The main conclusions to be drawn from this metanalysis are that lesions confined to the basal ganglia (putamen, caudate nucleus and globus pallidus) rarely, if ever, cause apraxia. Lesions affecting the lenticular nucleus or putamen nearly always intruded into the adjacent lateral white matter to involve association fibres, in particular those of the superior longitudinal fasciculus and frontostriatal connections. Apraxia occurred with deep lesions of the basal ganglia apparently sparing white matter in only eight out of the 82 cases. Apraxia was most commonly seen when there were lesions in the lenticular nucleus or putamen (58 out of 72 cases) with additional involvement of capsular, and particularly of periventricular or peristriatal, white matter. Lesions of the globus pallidus (no cases) or caudate nucleus (three cases) rarely caused apraxia. The caudate lesions also had white matter involvement. Indeed, involvement of periventricular or peristriatal white matter alone caused apraxia. The vast majority of cases described with apraxia associated with deep lesions were in the left, dominant hemisphere. Ideomotor apraxia was described in most reports (72 out of 82 cases). Orofacial apraxia was less common (37 cases), usually with ideomotor apraxia. Ideational apraxia was rare (five cases), all with ideomotor apraxia. Apraxia was either bilateral or involved the left hand if there was a right hemiparesis, in those cases where descriptions were available. Lesions of the thalamus can sometimes cause apraxia (26 cases), even if there is no apparent involvement of white matter (12 cases). Small lesions confined to the thalamus can also sometimes cause apraxia (eight cases). The role of the thalamus in higher order motor control and apraxia remains to be determined. It is suggested that the term limb-kinetic apraxia should be retained to describe motor deficits in planning ‘what to do’, ‘how to do it’ and ‘when to do it’; decisions which appear to involve activation of a complex distributed network of dorsolateral prefrontal cortex, supplementary motor areas, anterior cingulate regions and lateral premotor cortex. Such deficits need to be quantified. If they are present in patients with basal ganglia disease, over and above classical akinesia, bradykinesia and hypokinesia, then such patients could be said to exhibit limb-kinetic apraxia.

Keywords: apraxia; basal ganglia

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**Introduction**

Ever since the original descriptions of Liepmann (1900, 1905, 1906, 1908, 1920) and Liepmann and Maas (1907), apraxia has usually been attributed to damage confined to the cerebral cortex and cortico-cortical connecting pathways. Damage to subcortical structures causes fundamental motor or sensory deficits, or both, which by many definitions exclude the possibility of apraxia. However, here we take the view that apraxia is a legitimate description of disorders of planning and execution of complex movements, irrespective of the site of pathology. The akinesia (with hypokinesia and bradykinesia) and rigidity characteristic of basal ganglia diseases, the weakness and spasticity resulting from corticospinal tract lesions and the ataxia of cerebellar diseases may not be sufficient to explain the full motor deficits in these conditions. In particular, the breakdown of movement in many basal ganglia diseases may involve additional ‘higher order motor disorders’ that could be termed apraxia.

Liepmann himself recognized ‘a-praxia or para-praxia’ as a disorder of complex higher motor behaviour, characterized by the inability to perform purposeful skilled movements in the absence of elementary motor deficits (weakness, akinesia, abnormal posture or tone), abnormalities of sensation, or impaired comprehension or memory. Apractic patients maintain their capacity to contract their muscles and to move. However, they choose the wrong innervatory pattern and the intended motor act is substituted by another ‘inappropriate’ action, or by fragmented and spatially displaced, or perseveratory movements. Thus, from the beginning Liepmann suggested that apraxia was predominantly a motor, indeed a movement, disorder. Liepmann (1900) concluded: ‘...the patient is not apraxic due to impairment of the sensory-receptive functions... the motor part of the action, the control of the motor expression is defective’. Although Liepmann’s observations, even if their main focus is ideomotor apraxia, are cited in the references of virtually all papers on the topic, his interpretation of apraxia as a movement disorder rather than a disorder of symbolic behaviour has not received attention.

If we consider apraxia as a disorder of complex higher motor behaviour, implying not just elementary contraction of muscles but ‘how we put together motor programs and how we run motor plans in response to external influences and our own internal thoughts’ (Marsden, 1984), then some causal relation to the basal ganglia is worth consideration. The introduction of CT in the mid-1970s, and subsequently of MRI, led to the discovery of a hitherto unsuspected role of deep hemispheric structures in the pathogenesis of symptoms that traditionally were deemed to be the province of the neocortex and underlying white matter. The neural substrates subserving ‘higher cortical functions’ (Luria, 1966), a phrase which should no longer be used in a too general and simplistic way, must be enlarged to encompass these basal grey nuclei and/or their connections.

Many higher mental function disorders, such as aphasia (Hier et al., 1977; Alexander and LoVerme, 1980; Damasio et al., 1982; Naeser et al., 1982; Cappa et al., 1983; Puel et al., 1984; Alexander et al., 1987; Basso et al., 1987), aggraphia (Cappa and Vignolo, 1979; Naeser et al., 1982; Tanridag and Kirshner, 1985), constructional impairment (Kirk and Kertesz, 1993) and neglect (Hier et al., 1977; Watson and Heilman, 1979; Heaton et al., 1982; Ferro and Kertesz, 1984) have been described in association with subcortical deep hemispheric lesions. However, little attention has been paid to apractic disturbances and the potential role of the basal ganglia in their pathogenesis.

The existence of subcortical or ‘deep’ apraxias has, with a few exceptions (Agostini et al., 1983; De Renzi et al., 1986; Della Sala et al., 1992), not yet been considered seriously. In general, there has been no reference to apraxia in articles reporting CT or MRI scan evidence of deep hemispheric damage producing aphasia, but such reports exist. These studies often lacked a standardized praxis assessment and a detailed description of apractic behaviour, but they are the only source of reference available at present. Some insight into the problem may be obtained by metaanalysis of these publications. Here we present the data of 82 patients reported in the literature up to April 1994 with deep apraxias associated with damage mainly, or exclusively, in the basal ganglia, thalamus and closely adjacent structures, such as internal capsule and periventricular or peristriatal white matter.

Before reviewing this material we will give a brief historical review of the evolution of the concepts of apraxia. This is an essential prelude to the definition of the ways in which this term has been employed, and how the various subtypes of apraxia have been defined.

**Historical background (Table 1)**

The term apraxia first appeared in the literature in 1871 (Steinthal, 1871). However, it was Hugo Karl Liepmann, a
young German neurologist, who, around the turn of the century, first studied apraxia thoroughly (Liepmann, 1900, 1905, 1906, 1908, 1920; Liepmann and Maas, 1907). His detailed and brilliant clinical description (Liepmann, 1900) of a 48-year-old right-handed Imperial Counsellor (R.T.), admitted to a Berlin Hospital with the diagnosis of 'mixed aphasia and post-stroke dementia', and his work written subsequently, provides the fundamental framework for every neuropsychological model of apraxia.

Apraxia has since been attributed to pathology affecting the parietal and frontal cortical areas of the hemisphere dominant for speech, or to the fibre bundles connecting these areas within the same hemisphere (intrahemispheric) and via transcallosal pathways (interhemispheric) with the motor cortical regions of the ipsilateral and contralateral hemispheres (Liepmann, 1920; Geschwind, 1965, 1975; Faglioni and Basso, 1985; Heilman and Rothi, 1985).

Liepmann and Maas (1907) postulated that the left hemisphere incorporates not only language but also motor engrams which control purposeful, skilled movements. Liepmann (1920) called these motor engrams 'Bewegungsformeln' (movement formulae) containing the 'time–space–form picture of movement' (Kimura, 1979). Heilman (1979) refers to them as 'visuokinaesthetic motor engrams'. Learning a skilled motor behaviour requires the acquisition of both 'movement formulae' and 'innervatory patterns' which convey the information on formulae to the appropriate primary motor areas (Liepmann, 1905).

In Liepmann's praxis-concept, the correct execution of a complex movement depends on the existence of a 'Bewegungsentwurf' by which spatial and temporal arrangement of all the single movements composing the motor action are defined and controlled. Basically, Liepmann distinguished two stages: (i) the evocation of the general plan or the ideation of the movement; (ii) its execution by decoding the series of appropriate innervatory patterns (Liepmann, 1908, 1920). He considered that the idea or plan involved the participation of the whole brain, but with a particular role played by the posterior part of the left hemisphere. He felt that the 'kinetic memory' (kinaesthetic-innervatory engrams) of the innervatory pattern was stored in the (left) 'senso-motorium' (a region corresponding to the primary sensorimotor and the premotor areas). These kinaesthetic-innervatory engrams are aroused and directed by the motor plan.

Liepmann essentially denied the existence of locally confined brain areas ('praxis centres') which could generate complex movements on their own. However, he agreed that there are loci or systems in the brain concerned with the organization of higher order motor behaviour, which are interconnected by important cortico-cortical pathways, and that damage to these discrete structures, which enable sensory association and motor areas to work together, can cause disconnection and thus apraxia. Although, he did not accept the view of praxis centres, he admitted that lesions in different locations could cause different patterns of apraxia. For instance, the more posterior the lesion, the more qualitatively ideational the apractic disturbance. In contrast, the more anterior the lesion site, the more qualitatively limb-kinetic the apractic disturbance. Ideomotor apraxia was considered to be the result of a more intermediate parietal damage (Liepmann, 1920).

Although Liepmann did not encourage the notion of praxis centres in the brain, the concept is of value in theoretical 'black box' models of the organization of movement. Thus, the first stage of motor action involves the organization of a motor plan or idea of the action required, in what might be termed 'planning centres'. The motor plan must then be translated into the appropriate motor programmes required to activate the muscular actions necessary to achieve the overall objective. Such translation of the motor plan into the necessary motor programs can be conceived as taking place in 'executive centres'. 'Planning centres' and 'executive centres' do not imply specific anatomical locations.

Any disruption in the transmission of information from 'planning centres', where external stimuli evoking the movement and internal thought are integrated and processed into the motor plan, to the 'executive centres', where the plan of motor behaviour must be programmed in order to activate the appropriate motor neuron pools spatially and temporally, produces ideomotor apraxia. If the 'senso-motorium' is isolated, limb-kinetics are conserved but disconnected from the ideation or conceptualization of movement. On imitation some movements may well be restored but on the whole performance is still defective. The term 'ideomotor apraxia' includes difficulties in the purposeful manipulation of real objects as well as actions with pretend objects. The apractic patient knows exactly what he has to do, but is incapable of calling on the innervatory pattern needed to execute the movement.

In contrast, inadequate elaboration of Liepmann's 'Bewegungsentwurf', results in ideational apraxia (Ideatorische Apraxie). Liepmann's original definition of this disorder was the failure to carry out sequential motor activity, when each step could be performed separately. Motor behaviour is most affected when the subject must create and programme the motor action. Its severity is proportional to the complexity of movement, and errors are concerned mainly with the correct sequence of movements.

According to Liepmann (1920), loss of the kinaesthetic-innervatory engrams or kinetic memory as a result of damage to the 'senso-motorium' causes limb-kinetic apraxia (Gliedkinetische Apraxie). This kind of apractic disturbance affects all types of gesture without relation to their complexity, independent of whether the patient must create or only imitate the action. Movements are characterized as clumsy or awkward and are preceded by pauses during which 'unsuccesful' movement patterns may be carried out.

Geschwind (1965, 1975) substantially accepted Liepmann's interpretations, and proposed that verbal and written commands elicit motor behaviour using a neural pathway similar to that proposed by Wernicke (1874) for language.
Table 1  Apraxia: evolution and definitions

Liepmann, H.
Definition: higher order motor behaviour (praxis): continuous interaction between ‘movement formulae’, ‘kinetic memory’ (where learned movements are stored), and transcoding of the formulae into an adequate scheme of ‘innervations’; disruption of this interaction results in an inability to perform purposeful skilled movements, in the absence of any elementary motor (weakness, akinesia, abnormal posture or tone) or sensory deficits, or impaired comprehension or memory.

Ideomotor apraxia (Ideokinetische Apraxie): disconnection between the (left) posterior areas (auditory, visual, and tactile cortical areas) and the (left) ‘senso-motorium’; the limb kinetics are maintained but detached from the ideation of movement.

Ideational apraxia (Ideatorische Apraxie): failure of elaboration of the motor plan (‘Bewegungsentwurf’) associated with inability to conceive how the gesture must be organized; failure to carry out a sequential motor activity, when each component step could be performed separately.

Melo- or limb-kinetic apraxia (Gliedkinetische Apraxie): loss of kinaesthetic-innervatory engrams or kinetic memories due to damage of the (left) ‘senso-motorium’.

Summary: The left hemisphere (inferior parietal lobule) programmes and directs higher order motor behaviour, and controls the motor activity of the right (subordinate) hemisphere. Motor planning is the province of the the cortex and adjacent white matter.

Geschwind, N.
Definition: disconnection between interacting cortical areas leads to a disorder of the execution of learned movement which cannot be accounted for either by weakness, or sensory loss, or by incomprehension of or inattention to commands.

Parietal apraxias (apraxias of the supramarginal gyrus): unilateral lesions penetrating deeply in this region may lead to an apraxia which is either equal bilaterally or worse in the right.

Sympathetic apraxia: inability of the nonpathologic hand to carry out commanded movements.

Callosal apraxia: inability of one limb (usually left) to perform on command, even though the other limb performs easily.

Summary: ‘disconnexion’ of motor regions from the speech area and from other sensory inputs.

Heilman, K. M.
Definition: disorder of skilled movement, not caused by weakness, akinesia, deafferentation, abnormal posture, movement disorders (such as tremor or chorea), intellectual deterioration, poor comprehension or uncooperativeness.

Ideomotor apraxia: impairment of selection, sequencing and spatial orientation of movements involved in gestures including emblems and pantomimes; two forms of ideomotor apraxia:
(i) posterior type: induced by lesions of the left parietal cortex (supramarginal or angular gyrus) which contains the visuokinesthetic motor engrams;
(ii) anterior type or ideomotor disconnection apraxia: induced by lesions anterior to the supramarginal gyrus that disconnect the visuokinesthetic motor engrams from premotor and motor areas.

Disconnection and disassociation apraxias:
(i) verbal–motor disassociation apraxia: defective ability to elicit the correct motor sequence in response to language;
(ii) visuomotor and tactile–motor disassociation apraxia: disconnection between modality-specific pathways and the centre where movements are programmed.

Ideational apraxia: difficulty in sequencing a series of acts.

Limb-kinetic apraxia: inability of making fine precise movements with the limb contralateral to a CNS lesion; although the precision of movement is lacking, selection, sequencing and spatial orientation are intact.

Summary: visuokinesthetic motor engrams, stored in the dominant parietal cortex (inferior parietal lobule), not only programme motor areas for gesture production but also play a crucial role for gesture comprehension and discrimination.

De Renzi, E.
Definition: inability to select the correct motor innervation.

Ideomotor apraxia: impairment in selecting the appropriate pattern of innervation needed to implement the idea of the movement (inability to implement the general idea of the movement into a proper sequence of specific muscle innervations).

Ideational apraxia: impairment of the ability to evoke the general shape of movement; evocative or ideational deficit: either due to the disruption of the engram storage, or its unaccessibility (lack of access to specific aspects of the semantic store); faulty manipulation of actual objects.

Summary: inability to deliberately make a choice amongst the repertoire of motor innervations; apraxia model should be enlarged to include the deep nuclei and the pathways running through them.
Table 1 Continued:

Poeck, K.
Definition: disruption of ‘higher order’ motor processes causing ‘parapractic’ errors (i.e. inappropriate movements or inappropriate elements within a movement).
Ideomotor apraxia: impairment in the proper selection of the motor elements which constitute a movement and the correct ordering of these elements in a motor sequence.
Ideational apraxia: disturbance in the associative elaboration of various inputs with motor programmes; disturbance in the conceptual organization of movements.
Summary: disruption of ‘higher order’ motor processes:
(i) impaired selection of elements which constitute a movement;
(ii) impaired sequencing; both are of equal importance.

Freund, H. J.
Definition: higher order motor disturbance which can affect almost any aspect of motor behaviour.

Posterior apraxias
Unimodal apraxias: (disturbances of higher order motor behaviour in relation to somatosensory or visual functions):
(i) tactile apraxia;
(ii) visuomotor apraxia;
(iii) apraxia of speech (location: posterior parietal lobe or its junctional zones with the temporal and occipital lobes).
Supramodal apraxias: (the disturbed motor behaviour is not restricted to a particular modality but affects the ideation, and conception of the intended motor acts at a global level):
(i) Ideational apraxia: deficit of the conception of movement;
(ii) Ideokinetic (ideomotor) apraxia: the action is conceptually determined but faulty in the execution of its parts; complex acts can be executed properly, whereas their constituent elements may be disturbed.

Frontal apraxias
Executional apraxias:
(i) limb-kinetic apraxia;
(ii) disorders of motor learning and rhythm production.
Summary: prominent role of unimodal and polymodal sensory association areas for motor control.

processing. Furthermore, he emphasized the ‘disconnexion’ of the motor centres from the stimuli-processing cortical association areas (auditory association cortex for verbal commands, visual association cortex for movement carried out to written commands or on imitation, and somaesthetic association areas for movements performed under tactile control). Considering the anatomical pathways by which movements with the right hand are carried out on verbal command, Geschwind postulated that the order has to be transmitted from Wernicke’s area (usually involved in the comprehension of spoken language) through the lower parietal lobe to the left premotor region, which controls the precentral motor cortex that gives rise to the pyramidal tract. When a command to carry out an action with the left hand is given to the subject, the order must also pass through Wernicke’s area in the left hemisphere, but from there two alternative routes may be taken: (i) the anterior route goes from Wernicke’s area to the left premotor cortex, from there via the corpus callosum to the premotor region of the right hemisphere, and on to the right precentral motor cortex, which controls the left limb; or (ii) the posterior (alternative) route goes from Wernicke’s area to the corresponding region in the right hemisphere and on to the right premotor cortex and precentral motor cortex. The posterior route is probably not used primarily, as damage to this pathway when the more anterior pathway is spared does not lead to apraxia in the left arm (Geschwind, 1975).

Liepmann’s proposal (1905, 1908) that the hemisphere dominant for handedness is a storehouse for the learning involved in the acquisition of motor skills, is supported by Geschwind: ‘... when we use our left limbs, the left hemisphere—the repository of detailed information concerning movements—is likely either to direct completely the right hemisphere or at least to contribute to the significantly smaller store of learning of the right’ (Geschwind, 1975). This view implies that the side of the brain governing the most dexterous hand must also be dominant in motor planning. However, one need not assume that in all cases only the left hemisphere learns motor skills. The right ‘subordinate’ hemisphere may have a store of motor learning that is released only when given much more information (Geschwind, 1975). Geschwind also emphasized the concept of ‘multiple motor systems’ (pyramidal and nonpyramidal) which might explain the preservation of certain types of movements. Such systems could also constitute the alternative routes by which patients attempt to respond in spite of disconnecting lesions (Geschwind, 1975).

Heilman (1979) introduced the concept of visuokinaesthetic motor engrams using the biological term for memory (engram) in reference to Liepmann’s movement formula. These ‘motor representations’ or ‘time–space motor engrams’ which ‘... program sequences of movement needed to perform skilled acts’ are thought to be stored in the dominant parietal cortex (inferior parietal lobule) (Heilman, 1979;
Kimura, 1979; Rothi and Heilman, 1985), but may also have a bilateral representation (Heilman, 1979; Kertesz et al., 1984). Lesions which destroy these engrams should be dissociable behaviourally from lesions that spare but disconnect the engrams from primary motor areas. Destruction of such engrams would cause ideational apraxia. Disconnection of the engrams from the motor areas would cause ideomotor apraxia.

Heilman et al. (1982, 1991) and Heilman and Rothi (1985) distinguished two forms of ideomotor apraxia. The first, the posterior type of ideomotor apraxia, is due to lesions of the left parietal cortex (supramarginal or angular gyrus) which contains the visuokinaesthetic motor engrams. Destruction of these engrams, which are thought to support the gestural discrimination function, causes disruption of input processing as well as performance deficits. Patients with this form of ideomotor apraxia perform badly to command and imitation, and also cannot discriminate between poorly and well-performed acts carried out by the examiner. The second, the anterior type of ideomotor apraxia, is induced by lesions anterior to the supramarginal gyrus that disconnect the visuokinaesthetic motor engrams from premotor and motor areas. Patients with this ideomotor disconnection apraxia also cannot perform well to command or imitation, but are able to comprehend and discriminate pantomimes flawlessly because they still have the information (motor representations or time-space engrams) characterizing distinctive features of movement or movement formula (Heilman et al., 1991). Patients with damage to the dominant parietal lobe that destroys these time-space motor engrams cannot perform this motor analysis (Heilman et al., 1982; Rothi et al., 1985). The anterior disconnection form of ideomotor apraxia may be further subdivided into two types: (i) that induced by callosal lesions (patients fail to perform correctly with the hand ipsilateral to the hemisphere containing the engrams, but perform normally with the hand contralateral to these engrams; (ii) that induced by lesions of the hemisphere containing these visuokinaesthetic motor engrams (patients, if not hemiparetic, should fail to perform correctly with either hand) (Heilman et al., 1982, 1991). Apraxia associated with recognition and discrimination disorders caused by posterior lesions was considered by some as a form of 'agnosia of gesture'. Heilman et al. (1991) stated that there may be some patients with disconnection between the visual areas and these motor engrams who should use objects correctly and perform to command but not recognize gestures; only such patients might be considered agnosic for gestures. Rothi et al. (1986) described two patients with left occipitotemporal lesions who were agnosic for pantomimes and gesture (pantomime agnosia). They proposed that these lesions deprived the time-space movement representations, stored in the parietal lobe, of visual input.

Heilman's definition of ideational apraxia (Heilman, 1973) was subsequently termed verbal–motor dissociation apraxia. Originally, Heilman described three patients who, when asked to gesture, performed differently from patients with ideomotor apraxia. They offered no motor response when asked to perform, but were able to describe the desired act, imitate the examiner and use actual objects flawlessly. Because imitation and actual object use were performed well, Heilman concluded that their engrams for motor skills were intact. He postulated a disconnection between Wernicke's area and the proposed parietal centre for the time-space motor engrams. If the information in the command could reach this parietal centre by some other way (imitation or presentation of the actual object), transmission to the frontal lobe for motor execution could proceed normally. This definition has not been accepted by other authors. In summary, Heilman's motor representations or visuokinaesthetic motor engrams in the proposed parietal 'centre' not only programme motor areas for gesture production, but also play a crucial role for gesture comprehension and discrimination.

Although De Renzi (De Renzi et al., 1983; De Renzi, 1986) basically shared Heilman's view that the brain region most frequently associated with apraxia is the left inferior parietal lobule, he considered a potential role of deep brain structures (De Renzi et al., 1986). He stressed that the essence of the apractic deficit appears in the patient's inability to deliberately make a choice amongst his repertoire of motor innervations, when his motor performance is not assisted by a facilitating set of circumstances. The crucial factor in determining apraxia is not the quality of movement, but rather the artificial condition of its evocation and planning (De Renzi, 1985). Therefore, according to De Renzi et al. (1982), apraxia is not a unitary disturbance of movement execution, but variously affects gestures in relation to their nature and the modality through which the instructions eliciting the appropriate motor response are conveyed.

Poeck (1985) emphasized that the defining feature of apraxia is not that the patient performs an action in a clumsy way but rather that the examiner notes 'parapractic' errors (i.e. inappropriate movements or inappropriate elements within a movement). Poeck's view is that the two varieties of motor apraxia, ideomotor and ideational, are caused by disruption of 'higher order motor processing'. This disruption may occur in two ways: (i) impaired selection of elements which constitute a movement; (ii) impaired sequencing (Poeck, 1985). Both aspects are of equal importance. In a study stimulated by the research of Birdwhistell (1970) into the units of movement ('kinemes and kinemorphs'), Poeck and Kerschensteiner (1975) developed a method permitting the quantitative and qualitative assessment of the single components constituting apractic behaviour. Based on the results of this study, these authors concluded that apraxia cannot be viewed as a mere deficit in sequential activity, but that inappropriate selection of motor elements within a motor sequence must be of equal importance. Lehmkühl and Poeck (1981) and Poeck (1982) suggested that ideational apraxia is due to disturbance in the associative elaboration of various inputs with motor programmes, and could be viewed as disturbance in the conceptual organization of movements.

De Renzi and Luchelli (1988) and Poeck (1985) support
the view that ideational apraxia is not a very severe form of ideomotor apraxia, but represents an autonomous syndrome. De Renzi et al. (1968) defined ideational apraxia as a faulty manipulation of actual objects, with its basic deficit residing in the lack of access to a specific aspect of the semantic store (De Renzi and Luchelli, 1988). Associated unilateral brain damage is frequently found in the left posterior temporoparietal junction (De Renzi and Luchelli, 1988).

Freund (1992) has attempted to link the traditional concepts and other motor dysfunctions that can be designated as apractic on the basis of the disordered motor behaviour and the underlying pathophysiological changes of disturbed sensorimotor integration. He has subdivided apraxia into two main groups: (i) posterior apraxias and (ii) frontal apraxias. The posterior apraxias are of the unimodal type (disturbances of higher order motor behaviour in relation to somatosensory, auditory or visual functions), or supramodal type (the disturbed motor behaviour is not restricted to a particular modality but affects the ideation and concept of the intended motor act at a global level). Unimodal apraxias comprise (i) tactile apraxia (deficit of active touch and manipulation), (ii) visuo-motor apraxia (disorder of visually guided motor behaviour) and (iii) apraxia of speech. By contrast, supramodal apraxias are composed of (i) ideational apraxia (deficit of the conception of movement) which interferes with everyday motor activities and is experienced as disability, and (ii) ideokinetic (ideomotor) apraxia (the action is conceptually determined but faulty in the execution of its parts). The frontal apraxias are executional apraxias and comprise (i) limb-kinetic apraxia and (ii) disorders of motor learning and rhythm production. Freund concludes '...what emerges from the observation of apractic behaviour and the corresponding lesions is the prominent role of unimodal and polymodal sensory association areas for motor control...these areas subserve the processing of sensory information not only for perceptive and cognitive purposes but also for elaboration of motor behaviour that in turn selects and shapes sensory input' (Freund, 1992).

Melo- or limb-kinetic apraxia is rarely described in the literature, and the term is rarely used at present. It has never acquired the same popularity enjoyed by ideomotor apraxia and to a lesser extent by ideational apraxia. This relative neglect may be partly due to the fact that Liepmann (1908) himself seemed to be somewhat uncertain about the nature of this type of apraxia, and used as an example not a case of his own, but rather one published by Westphal (1908). Geschwind (1965) stated: '...my feeling had been that 'limb-kinetic' apraxia has not been defined clearly enough to separate it from mild pyramidal disturbance'. Brown (1972), in his paper on the apraxias, emphasized its similarity with paresis.

The descriptions of the motor disturbances in limb-kinetic apraxia range from slowness, clumsiness and awkwardness of movement to loss of the kinetic melody, temporal disordering and decomposition of movement. According to Luria (1966): '...the disintegration of the dynamics of the motor act and of complex skilled movements' constitutes the central symptom of motor disturbances arising from lesions of the premotor cortex. Heilman and Rothi (1985) describe patients with limb-kinetic disturbances as incapable of making fine, precise finger movements with the limb contralateral to the lesion. Characteristically, the patient produces movements that, although lacking precision, are accurately selected sequences correctly orientated in space. In contrast, patients with ideomotor apraxia have difficulty in selection, sequencing and spatial orientation. Poeck (1985) considers that the classical definition of motor apraxia excludes limb-kinetic apraxia as described by Kleist (1934), which is just impairment of fine distal movements indicating a functional disturbance in primary motor pathways.

De Renzi (1986) emphasized the dearth of well-documented case reports of apractic disturbances, which could be termed limb-kinetic apraxia, associated with damage to the frontal lobe not extending behind the Rolandic fissure. In an extensive review of the literature, Faglioni and Basso (1985) found only seven cases that fit the criteria of classic limb-kinetic apraxia. Freund and Hummelsheim (1985), in a very interesting study on lesions of premotor cortex in man, reported proximal paresis and bibrachial limb-kinetic apraxia for coordinated movements (the dysfunction was most pronounced when the patient was asked to produce a 'windmill' movement), and concluded that both aspects have one common feature: the disturbance of the temporal sequencing of muscle activation. In general, many investigators renounce limb-kinetic apraxia as a 'true' apraxia, and regard it as a mild pyramidal or extrapyramidal disorder. Nevertheless, this deserves further investigation.

Despite the fact that isolated patients with apraxia arising from 'deep' lesions have been reported, no studies, with the exception of those of De Renzi et al. (1986) and Della Sala et al. (1992), have systematically explored apraxia following subcortical deep lesions. This general lack of interest in errors of movement formation and even more in their 'non-classical' localization aspects, compared with aphasia and other neuropsychological disorders, may have two possible explanations. First, apraxia, except the ideational type (Poock, 1983, 1985; Freund, 1992), seldom disrupts everyday activities and may therefore escape both the patient's (who may also be anosognosic) and examiner's attention, unless it is deliberately looked for. In contrast, aphasia is immediately perceived by the patient or others around him. Secondly, the different pattern or clinical pictures of apractic disorders still defy a definition. The paucity of reports of 'deep apraxias' may be due to the relative rarity of these cases, but it probably depends more on a lack of specific praxis investigation.

Clinicoanatomical correlations

Material

The literature was reviewed from 1914 up to April 1994 for descriptions of apractic disorders produced by lesions
Neuropathological studies

Liepmann (1906) first approached apraxia from a clinicanoanatomical point of view. However, the major neuropathological work on the topographical aspects of apraxia was carried out by von Monakow (1914). He and other authors challenged Liepmann's 'associationist' schema, and claimed that apraxia was the result of diaschisis (von Monakow, 1914) or mass effect (Lashley, 1929), rather than damage of specific cortical areas. In von Monakow's view (1914) apraxia was a disorder of automatic movement resulting from a change of excitability in cortical areas provoking interference between certain reflex arcs. He was also the first to compare cases with focal or diffuse lesions causing apraxia in life, with those who had lesions in sites thought to cause apraxia but with no such clinical deficit. He reviewed the literature until 1914 and reported the topographical and pathological features of 24 apractic patients (von Monakow, 1914, p. 537). Eight patients had a lesion in the inferior parietal lobule, eight exclusively or predominantly in the corpus callosum, one in the temporal and three in the frontal lobe. In four cases the lesions were multifocal or diffuse. In seven of the 24 cases the lesion was confined to left hemisphere, in two to the right and in seven it spanned both hemispheres (in the remaining eight cases the exact location was not described). As far as aetiology was concerned, adding 10 cases of his own, von Monakow concluded that 17 cases had encephalomalacic lesions presumably due to infarction, 13 had a tumour, one had a haemorrhage and three had diffuse arteriosclerotic cerebral atrophy.

Considering his own necropsy series of more than 14 cases von Monakow noted that amongst his positive cases there was a group of four patients with marked bilateral, mainly ideomotor apraxia, which did not fit the 'usual' anatomical concept of apraxia. The lesions in this 'atypical' group characteristically involved basal structures, such as the lenticular nucleus, thalamus and posterior limb of the internal capsule.

Case reports

Case V (von Monakow, 1914, pp. 520–2, 538, 544, 554). U.B.: a 47-year-old man with marked difficulty in eating and dressing. Later in the course receptive aphasia. Constant feature was a marked bilateral ideomotor, later in the course also ideational apraxia. Necropsy: a tubercle destroying the left posteroverentral part of the lenticular nucleus, the posterior limb of internal capsule and the subthalamic region was found. However, there was also some brain swelling and hydrocephalus.

Case XII (von Monakow, 1914, pp. 532–5, 539–40, 544, 554). Widmer: a 38-year-old teacher with a history of right mastectomy for breast cancer. On examination there was no manifest hemiplegia but marked abasia and astasia, no aphasia or agnosia but remarkable alexia and agaphia. She was bilaterally apractic (ideomotor apraxia) and could not use her hands purposefully (with major difficulties in eating, using a towel, etc.). Necropsy: multiple bitemporal metastatic lesions, left more than right. The main localization was in both the thalami, predominantly on the left (posterior and anteromedial) with invasion of the third ventricle, the ventral part of the left lenticular nucleus and the retrolenticular capsular region. On the right there was involvement of the splenium and hippocampal gyrus. One lesion was in the tectum mesencephali.

Vmar (von Monakow, 1914, pp. 544, 554). This patient also presented with bilateral apraxia as the main and, for a long time, the only symptom. Necropsy: glioma originating in both thalami, lenticular nuclei and adjacent internal capsules invading the corona radiata.

Case VI (von Monakow, 1914, pp. 522–3, 554). Heuss: a 27-year-old man with bilateral apraxia as early feature. Necropsy: huge cystic sarcoma originating from the anterior part of the corpus callosum invading both hemispheres, mainly the dorsal thalamus and centrum ovale on the left and with marked compressive effect on the lenticular nucleus, internal capsule and thalamus bilaterally. von Monakow concluded that this is certainly not a 'pure' case of basal ganglia lesions and apraxia, but could be illustrative for aspects of mass or neighbourhood effect.

Involving the basal ganglia (caudate nucleus, putamen, globus pallidus) and the thalamus. The reports were classified into two main groups based upon (i) the anatomical investigation carried out (neuropathology or neuroimaging) and (ii) the reported extent of the lesions. The lesions were subdivided into (i) small isolated lesions involving nuclei of the basal ganglia or thalamus only and not extending to involve the internal capsule (anterior limb internal capsule, posterior limb internal capsule) or periventricular or peristriatal white matter; and (ii) large lesions with involvement of two or more nuclei of the basal ganglia and/or thalamus, or one or more of those deep structures plus damage to closely adjacent structures (such as internal capsule, periventricular or peristriatal white matter, or both), or lesions affecting the internal capsule, periventricular or peristriatal white matter, or both, without basal ganglia or thalamus involvement.

We started from the early neuropathological studies at the beginning of the century, and ended with the neuroimaging studies reported in recent years. In almost all cases the aetiology was deep vascular damage causing infarction or haemorrhage. Rarer cases were due to tumour or encephalomalacia. A list of the reports we have reviewed for this metanalysis is given in the Appendix. Ideomotor, ideational and orofacial apraxia were considered. Ideomotor apraxia was by far the most frequently reported and investigated, while limb-kinetic apraxia was scarcely mentioned.
There are two other case reports which refer to basal ganglia lesions producing apraxia. Forster (1913; cited in Kleist, 1922) described ideomotor apraxia in a patient with a tumour whose necropsy showed involvement of the left putamen, globus pallidus and caudate nucleus. Noethe (1913; cited in Kleist, 1922) described a patient with ideomotor apraxia in whom the left putamen and globus pallidus were involved by softening; callosal structures were also reported to be involved. von Stauffenberg (1918) reported one patient, and Kleist (1922) two other cases with softenings in the basal ganglia and apractic disturbances, but in all three cases parietal or frontal lobe involvement was also mentioned.

**Neuroimaging studies**

Cappa and Vignolo (1979) reported limb apraxia following left thalamic haemorrhage in one of their patients. Apractic difficulties were described in imitating limb movements and in demonstrating the use of actual objects. Alexander and LoVerme (1980) described 15 patients with left putaminal or thalamic haemorrhage. Praxis was impaired in six of the 15 patients (mild, \( n = 5 \); moderate, \( n = 1 \)) and deficits were seen in both orofacial and limb tasks to verbal command. For limbs the deficits consisted only of the use of a body part as an object. Perseveration was also noted. Basso and Delia Sala (1986) found four patients with ideomotor limb apraxia in a group of 26 vascular patients with lesions confined to deep structures (left thalamus and nucleus lenticularis; cited in Faglioni and Basso, 1985).

Naeser et al. (1982) investigated nine patients: eight patients with infarct, and one patient with haemorrhage, in the left capsular–putaminal region. She divided them into three main groups. In the first group there were three patients (all occlusive-vascular) with capsular-putaminal lesions extending anteriorly and superiorly on CT brain scan. Two of these cases had mild, and one had moderate, orofacial apraxia; one had moderate left limb apraxia. All three patients had a lesion in the putamen and part of the anterior limb of the internal capsule, with anterior extension into the periventricular white matter deep to Broca’s area and a large superior extension into the periventricular white matter and corona radiata. Damage was also present in varying amounts in the posterior limb of the internal capsule, the globus pallidus (mostly spared), parts of the caudate nucleus, and the area of the external capsule, claustrum and insula. In the second group there were three patients (occlusive-vascular, \( n = 2 \); haemorrhage, \( n = 1 \)) with capsular-putaminal lesions extending posteriorly. One had mild orofacial apraxia; left limb apraxia was mild in two and moderate in one. All had a lesion in the putamen, anterior limb and part of posterior limb of the internal capsule, with posterior extension across most of the auditory radiations in the temporal isthmus. Lesions were also present in parts of the globus pallidus, caudate nucleus, in the internal capsule, claustrum, external capsule and insula. In the third group there were three cases (all occlusive-vascular) with capsular-putaminal lesions extending both anterior–superiorly and posteriorly. All had severe orofacial and left limb apraxia. The lesions were centred in the putamen, globus pallidus and anterior and posterior limb of internal capsule with extension anterior–superiorly and posteriorly into the adjacent white matter.

Damasio et al. (1982) studied six cases with circumscribed non-haemorrhagic deep infarctions of the anterior limb of the internal capsule and of the striatum in the dominant hemisphere. All but one case had normal gestural praxis tested only on verbal command. CT scans showed an area of decreased density in the depth of the left hemisphere involving the white matter lateral to the body of the caudate nucleus and probably part of the body itself. There was an extension anteriorly and inferiorly into the area lateral to the tip of the frontal horn of the lateral ventricle. Bogliun et al. (1982), studying 14 cases with circumscribed vascular thalamic lesions, described the presence of ideomotor apraxia in one non-aphasic patient.

Agostini et al. (1983) reported seven patients with cerebrovascular lesions (ischaemic, \( n = 3 \); haemorrhagic, \( n = 4 \)) located in the basal ganglia and/or thalamus, without concomitant involvement of the cerebral cortex. Unfortunately, no detailed information about the exact anatomical lesion site in the basal ganglia was given. However, analysis of their schematic representation of the lesions reveals not only involvement of the basal ganglia and/or thalamus but also capsular involvement. Ideomotor apraxia, which was always mild, was present in all seven patients; one also had orofacial apraxia. Graff-Radford et al. (1984) described limb apraxia in three patients with left thalamic non-haemorrhagic infarction.

Kertesz and Ferro (1984), in a study addressing lesion size and localization in ideomotor apraxia, examined 177 adult right-handed patients who had had a single ischaemic stroke in the left hemisphere. They found nine patients with small lesions causing moderate to severe apraxia. Basal ganglia lesions were described in three of them involving the lenticular nucleus (\( n = 3 \)), caudate nucleus (\( n = 2 \)) and anterior limb of internal capsule (\( n = 2 \)). On higher cuts the lesions appeared lateral to, and in close relationship to, the body of the lateral ventricles, involving the anterior half of the periventricular white matter.

Limb apraxia in three out of 21 patients with left hemisphere vascular damage was reported by Basso et al. (1985). One patient had a thalamic lesion, another had a lesion in the lenticular nucleus and the anterior limb of the internal capsule, and the third patient had a haematoma encroaching upon the lenticular nucleus, internal capsule and periventricular white matter. Basso and Della Sala (1986) presented a single case report of a severe ideomotor apraxia associated with a peculiar difference between single movements and sequences. The lesion, haemorrhagic in nature, was apparently restricted to the head of the left caudate nucleus, the anterior limb of the internal capsule and the medial part of the lenticular nucleus.

A study which systematically addressed limb apraxia in
lesions confined to the left basal ganglia or thalamus, or both, apparently without any involvement of the cortex and the adjacent white matter, was carried out by De Renzi et al. (1986). In 14 patients (infarct, n = 10; haemorrhage, n = 4) limb praxis was investigated with standardized tests for ideomotor apraxia. Five patients were severely impaired regardless of the manner in which gesture organization was tested, whether by imitation, pantomimes or with the use of actual objects. In one of these cases there was also evidence of an ideational component, mainly in the form of omission and mislocation. In two patients the lesion was primarily thalamic, and in three it was mainly in the lenticular nucleus and posterior limb of the internal capsule. Another patient with a left lenticular haematoma and ideational apraxia was reported by De Renzi and Luchelli (1988). The errors consisted mainly in omissions (n = 6), perplexity (n = 2) and misuse and sequence errors (n = 1). The same patient also had evidence of ideomotor apraxia. Sanguineti et al. (1989) reported a case with an ischaemic lesion in the left periventricular white matter lateral to the putamen associated with ideomotor apraxia.

In two similar studies, Basso et al. (1987) and Della Sala et al. (1992) investigated apraxia in patients with purely deep lesions. The first study (Basso et al., 1987) of 37 cases, primarily addressed language. The lesions involved the basal ganglia, thalamus, internal capsule and/or periventricular white matter. Concomitant apractic disturbances were discovered in 10 patients. Nine cases had ischaemic lesions and one a haemorrhage. Five patients had orofacial apraxia only, one had ideomotor apraxia only and four had a combination of orofacial and ideomotor apraxia. In the more recent study, Della Sala et al. (1992) investigated 35 vascular patients, addressing apraxia. Ten patients (ischaemic, n = 8; haemorrhagic, n = 2) with deep lesions located mainly in lenticulo-thalamo-capsular structures, and always associated with periventricular white matter involvement, had some form of apraxia. Four patients had orofacial apraxia only, one had ideomotor apraxia only and five had ideomotor and orofacial apraxia.

Donnan et al. (1991) described 50 patients with striatocapsular infarction; 70% showed ‘cortical’ signs in the acute phase but none had CT evidence of cortical infarction. One of these patients with a left-sided deep lesion had ideomotor apraxia and aphasia. Papagno et al. (1993), in a study aiming to verify the existence of a double aphasia/apraxia dissociation, described two non aphasic patients with severe ideomotor apraxia and basal ganglia involvement. One patient had a left ischaemic lesion in the head of the caudate nucleus with no white matter involvement on that side. The second patient had an ischaemic and haemorrhagic lesion in the left hemisphere. The infarction involved the head of the caudate nucleus, the haemorrhage encroached upon the parietal cortex. In a prospective study, Godefroy et al. (1994) looked at the neuropsychological changes in 11 patients with unilateral lenticulostrate infarcts diagnosed by CT. Three of their patients showed gestural apraxia. MRI in these patients revealed the presence of cortical involvement not seen on the CT scan.

There have been other reports of ideomotor apraxia attributed to deep or entirely deep vascular damage (Basso et al., 1987) but no association with a specific deep structure was identified; these cases were not considered in our metaanalysis.

Results (Table 2)

Eighty-two cases with apraxia and associated damage to deep nuclei have been reported. The aetiology in the neuro-pathology group (n = 9) was softening in four cases, tumour (either primary or secondary) in four cases, and a tubercle in one case. The aetiology in the neuroimaging group (n = 73) was in almost all cases deep vascular damage causing either infarction (n = 46; 63%) or haemorrhage (n = 19; 26%) or both (n = 1; 1.4%). In seven cases (9.6%) the exact nature of the deep vascular damage was not specified. In 72 cases (88%) the damage was confined to the left basal ganglia region, and in five cases (6%) to the right basal ganglia region. Five patients (6%) had bilateral lesions.

Small, isolated lesions

There were 12 reported cases with small, isolated lesions. Most were vascular in nature (haemorrhagic, n = 7; infarction, n = 3, two were not specified). All were unilateral, in the left hemisphere. All were investigated only by CT scan. The time interval between the onset of the cerebrovascular insult and the CT scan was very variable, as were the methods of anatomical CT-localization of the lesions. It is well known that the sizes of lesions on CT scan images, in particular of cerebral haemorrhages, differ considerably in form and dimension according to when they were taken after the acute insult. Furthermore, CT scanning underestimates the full extent of anatomical damage.

Four cases were reported to have isolated lesions in the putamen. All were haemorrhagic and all had ideomotor and orofacial apraxia. Eight cases had lesions confined to the thalamus; three were haemorrhagic and three were ischaemic; two were not specified. Two of the eight cases had ideomotor apraxia only and six had ideomotor and orofacial apraxia. Ten of the 12 cases (83%) with isolated, left putaminal or thalamic lesions revealed a combination of ideomotor and orofacial apraxia. None showed oral apraxia only. No isolated lesions causing apraxia were reported to involve the caudate nucleus or globus pallidus.

Large lesions

Large lesions involving two or more basal ganglia structures and/or thalamus without involvement of the internal capsule and periventricular white matter

There were eight such cases reported. All were unilateral and confined to the left hemisphere. The lenticular nucleus
Table 2  Metanalysis of deep nuclei lesions and apraxia in 82 cases: 77 unilateral (72, left; five, right), five bilateral

<table>
<thead>
<tr>
<th>Small isolated lesions (n = 12) (unilateral, left)</th>
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<tbody>
<tr>
<td>PUT (n = 4)</td>
<td>TH (n = 8)</td>
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<tr>
<td>IMA/OA (n = 4)</td>
<td>CN (n = 0)</td>
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<td></td>
<td>GP (n = 0)</td>
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<th>Large lesions (n = 70) (unilateral, n = 65; bilateral, n = 5)</th>
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<tr>
<td>Large lesions without IC-PV/PSWM (n = 8) (unilateral, left)</td>
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<tr>
<td>TH plus LN without IC-PV/PSWM (n = 4)</td>
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<tr>
<td>LN without IC-PV/PSWM (n = 3)</td>
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<tr>
<td>LN plus CN without IC-PV/PSWM (n = 1)</td>
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<tr>
<td>IMA (n = 4)</td>
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<tr>
<td>IMA (n = 2), IMA/IA (n = 1)</td>
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<td>IMA (n = 1)</td>
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<tr>
<th>Large lesions with IC-PV/PSWM (n = 62) (unilateral, n = 57, left, 52; right, 5; bilateral (n = 5)</th>
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<tr>
<td>Basal ganglia and/or thalamus with IC-PV/PSWM (n = 52)</td>
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<tr>
<td>IC-PV/PSWM plus LN (n = 16)</td>
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<tr>
<td>IC-PV/PSWM plus LN/CN (n = 12)</td>
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<td>IC-PV/PSWM plus LN/TH (n = 11)</td>
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<td>IC-PV/PSWM plus CN (n = 3)</td>
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<tr>
<td>IC-PV/PSWM plus PUT (n = 0)</td>
</tr>
<tr>
<td>IC-PV/PSWM plus PUT/CN (n = 7)</td>
</tr>
<tr>
<td>IC-PV/PSWM plus TH (n = 3)</td>
</tr>
<tr>
<td>IMA (n = 8), OA (n = 3), IMA/OA (n = 5)</td>
</tr>
<tr>
<td>IMA (n = 7), OA (n = 1), IMA/OA (n = 3), IMA/IA (n = 1)</td>
</tr>
<tr>
<td>IMA (n = 8), OA (n = 1), IMA/OA (n = 1), IMA/OA/IA(n = 1)</td>
</tr>
<tr>
<td>IMA (n = 3)</td>
</tr>
<tr>
<td>IMA (n = 2), OA (n = 2), IMA/OA (n = 1), IMA/IA (n = 2)</td>
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<tr>
<td>IMA (n = 3)</td>
</tr>
<tr>
<td>IMA (n = 1), OA (n = 1), IMA/OA (n = 6)</td>
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<td>OA (n = 1)</td>
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n = number of cases; PUT = putamen; TH = thalamus; CN = caudate nucleus; GP = globus pallidus; IMA = ideomotor apraxia; OA = oral apraxia; IA = ideational apraxia; BG = basal ganglia; LN = lenticular nucleus; IC = internal capsule; PV/PSWM = periventricular/peristriatal white matter.

and the thalamus were affected in four patients; all had ideomotor apraxia. Lesions in the lenticular and caudate nucleus were reported in one patient who had ideomotor apraxia only. The lenticular nucleus only was involved in three cases; two patients had ideomotor apraxia, and one showed ideomotor and ideational apraxia.

Large lesions involving one or more basal ganglia structures and/or thalamus with involvement of the internal capsule and/or periventricular white matter

There were 52 such cases reported; 47 were unilateral (left, n = 42; right, n = 5) and five were bilateral. All but one of the patients with deep right-brain damage were right-handed. In one patient, handedness was not mentioned. In the only right-brain damaged patient for whom apraxia testing was reported as possibly bilateral, the motor performance was identical in both hands.

All subjects showed mainly ideomotor apraxia; one patient had concomitant orofacial apraxia. The periventricular/peristriatal white matter and/or internal capsule plus the lenticular nucleus were involved in 16 cases. Eight of the 16 cases had ideomotor apraxia, three orofacial apraxia, and five ideomotor and orofacial apraxia. Lesions of the periventricular/peristriatal white matter and/or internal capsule plus lenticular nucleus and caudate nucleus were found in 12 patients. Seven of the 12 patients had ideomotor apraxia only, one had orofacial apraxia only, three had ideomotor and orofacial apraxia, and one had ideomotor and ideational apraxia. Involvement of periventricular/peristriatal white matter and/or internal capsule plus lenticular nucleus and thalamus were reported in 11 cases. Eight had ideomotor, one had orofacial, one had ideomotor and orofacial, and one had ideomotor, orofacial and ideational apraxia. Lesions affecting periventricular/peristriatal white matter and/or internal capsule plus caudate nucleus caused apraxia were reported in three patients; all had ideomotor apraxia. Periventricular/peristriatal white matter and/or internal capsule damage plus putamen and caudate nucleus damage was found in seven cases. Two had ideomotor, two had orofacial, one had ideomotor and orofacial, and two had ideomotor and ideational apraxia. Lesions involving the periventricular/peristriatal white matter and/or internal capsule plus the thalamus only were reported in three cases; all had ideomotor apraxia. There were no cases of apraxia associated with lesions of the periventricular/peristriatal white matter and/or internal capsule plus putaminal damage only.

Lesions involving internal capsule and/or periventricular white matter without basal ganglia and/or thalamus involvement

There was a group of 10 patients with deep lesions (all unilateral, and all left hemispheric) in the the periventricular/peristriatal white matter or internal capsule, or both, but
without basal ganglia and/or thalamus involvement. The periventricular/peristriatal white matter was affected in nine of the 10 patients. A capsular lesion only caused oral apraxia in one patient. Lesions involving the periventricular, particularly peristriatal white matter alone caused apraxia in eight cases. Six had ideomotor and orofacial apraxia, one had ideomotor, and one had only orofacial apraxia. Lesions affecting both the periventricular/peristriatal white matter and the internal capsule only were found in one apractic patient who had oral apraxia.

**Overall findings**

Considering lesions involving the deep nuclei but sparing the white matter, there were 20 cases (12 small, isolated; eight large). Twelve of these involved the lenticular nucleus. The putamen alone was involved in four of these cases. Twelve of the 20 cases involved the thalamus. The thalamus was involved alone in eight. The caudate was specifically involved in only one case, with additional lenticular nucleus damage. The globus pallidus was never involved alone, but was included along with the putamen in the eight cases of lenticular nucleus lesions.

Of the 62 cases with white matter involvement, the lenticular nucleus was involved in 46 cases, of which only the putamen was affected in seven. The caudate nucleus was involved in 22 cases, but in only three of these was the lesion confined to the caudate not involving the lenticular nucleus. The thalamus was involved in 14 cases, along with the lenticular nucleus in 11 of these, and alone in three cases.

In the 62 cases of deep nuclei lesions also with white matter involvement, the periventricular/peristriatal white matter was involved in 38 cases, with additional involvement of the internal capsule in 21 of these patients. In 24 cases of deep nuclei lesions the internal capsule alone was involved. Of the 52 cases with involvement of white matter and deep nuclei, the majority had damage to the lentiform nucleus (39 cases) or putamen (seven cases).

Considering the type of apraxia described in these 82 cases, ideomotor apraxia was described in the majority (72 cases). Orofacial apraxia was described less often (37 cases), and ideational apraxia was rarely mentioned (five cases). Limb-kinetic apraxia was not referred to in any publication. Orofacial apraxia was described in association with ideomotor apraxia in 27 cases, and alone in only 10 cases. Ideational apraxia was described with ideomotor apraxia in all five cases.

In those cases where there were adequate descriptions of the laterality of apraxia, apraxia was described as bilateral in nine cases, or involved the left hand if there was a right hemiparesis in 35 cases.

With regard to the coexistence of aphasia with apraxia, the neuropathological reports do not provide adequate descriptions. In the 73 patients with apraxia due to lesions identified by neuroimaging, aphasia was described in 46 cases, was said to be absent in four cases and was not specified in 23 cases. Of the 46 cases with aphasia and apraxia, the type of aphasia was not categorized in 19 cases. It was described as 'subcortical' in nine, 'transcortical' in seven, 'trans-subcortical' in six, global in four and Broca's type in one case.

**Discussion**

The main conclusions which we can draw from this metaanalysis are as follows. (i) The vast majority of cases described with apraxia associated with deep lesions were in the left, dominant hemisphere. (ii) Ideomotor apraxia was described in the vast majority of reports (72 out of 82 cases). Orofacial apraxia was less common (37 cases), usually with ideomotor apraxia. Ideational apraxia was rare (five cases), all with ideomotor apraxia. (iii) Apraxia was bilateral or involved the left hand if there was a right hemiparesis in those cases where descriptions were available. (iv) Deep nuclei lesions confined to the basal ganglia or thalamus alone are rarely associated with apraxia. Apraxia occurred with deep lesions apparently sparing white matter in only 20 out of 82 cases. (v) Additional involvement of capsular, and particularly of periventricular or peristriatal, white matter seems to play an important role in subcortical or 'deep' apraxia. Indeed, involvement of periventricular or peristriatal white matter alone can cause apraxia. (vi) Where deep nuclei are involved, apraxia is most commonly seen when there are lesions in the lenticular nucleus or putamen (58 out of 72 cases). (vii) Lesions of the globus pallidus (no cases) or caudate nucleus (three cases) scarcely ever cause apraxia. The caudate lesions also had white matter involvement. (viii) Lesions of the thalamus sometimes cause apraxia (26 cases), even if there is no apparent involvement of white matter (12 cases). Even small lesions confined to the thalamus sometimes can cause apraxia (eight cases).

**Subcortical apraxia: the significance of damage to peristriatal white matter including the superior longitudinal fasciculus**

The most parsimonious explanation of these findings is that lesions of the basal ganglia (putamen, caudate nucleus and globus pallidus) do not cause apraxia. Apraxia seen with large lesions involving the lenticular nucleus or putamen usually involve the periventricular, or more specifically the peristriatal, white matter. It is here that cortico-cortical fibre pathways thought to be important for speech and motor control are passing (Fig. 1). Deep nuclei lesions could well encroach upon these long fibre bundles. From a study of patients with lesions in the territory of the lenticulostriate and deep penetrating arteries, von Cramon (cited in Della Sala et al., 1992) stated that there is no relationship between ideomotor apraxia and basal ganglia, but if the lesions go only 1–3 mm beyond the outer edge of the neostriatum into the periventricular white matter, then ideomotor apraxia occurs.

This explanation does not immediately account for the 12
from frontal motor regions. Damage to the superior fasciculus and therefore disconnecting parietal motor areas especially of the putamen, may cause apraxia, especially ideomotor apraxia, by damaging the superior longitudinal frontal region. Accordingly, lesions of the basal ganglia, operculum and courses through the insula, to reach the lower posterior end of the sylvan fissure, through the parietal originates in the supratemporal plane, arches around the margin of the putamen, in relation to the anterior limb of the internal capsule. The superior longitudinal fasciculus lies very close to the lateral superior margin of the putamen, in relation to the anterior limb of the internal capsule. The superior longitudinal fasciculus originates in the supratemporal plane, arches around the posterior end of the sylvan fissure, through the parietal operculum and courses through the insula, to reach the lower frontal region. Accordingly, lesions of the basal ganglia, especially of the putamen, may cause apraxia, especially ideomotor apraxia, by damaging the superior longitudinal fasciculus and therefore disconnecting parietal motor areas from frontal motor regions. Damage to the superior longitudinal fasciculus, which connects Wernicke’s area in the temporal lobe to Broca’s area in the frontal lobe, has also been implicated in classical concepts of conduction aphasia (see Geschwind, 1965; Damasio and Damasio 1980). It is therefore important to discuss the relationship of conduction aphasia to the subcortical aphasias described with basal ganglia lesions. Indeed, of the 50 patients with apraxia described here in whom aphasia was mentioned, no less than 46 cases were aphasic.

**Subcortical aphasias**

Conduction aphasia ('Leitungsaphasie') was originally proposed as a distinct form of aphasia by Wernicke (1874). Geschwind (1965) described the clinical picture as ‘... the patient usually shows no or little hemiplegia. His spontaneous speech is often, but not always, copious. Dysarthria tends to be absent or mild, and whatever phrases are produced tend to be fluent. While articulation may be normal, the speech is obviously and often severely aphasic, usually highly circumlocutory and often grossly paraphasic with a tendency particularly to literal paraphasias. There is marked difficulty in naming. Writing suffers along with spontaneous speech. Facial apraxia to command is often marked and may also be present on imitation. The notable feature of these cases is the marked discrepancy between comprehension and repetition ...’. Benson et al. (1973) defined conduction aphasia as a disorder characterized by fluency and paraphasia in spontaneous speech with normal or nearly normal comprehension of spoken language, but markedly impaired repetition of words and phrases, usually with some difficulty in naming.

Lesions involving the capsulostriatal regions or the periventricular white matter may cause aphasia (Damasio et al., 1982; Naeser et al., 1982, 1989; Schiff et al., 1983; Tanridag and Kirshner, 1985; Alexander et al., 1987; Mega and Alexander, 1994). A number of different syndromes have been described among those with subcortical aphasia. Naeser et al. (1982), in an analysis of nine patients, found that the character of the language disturbance varied with the lesion site. Alexander et al. (1987) have categorized 19 of their own cases of subcortical aphasia and 61 cases described in the literature into several subsyndromes associated with specific subcortical lesions. Cases involving the extreme capsule and insula exhibited conduction aphasia without dysarthria. Cases with lesions confined to the putamen or head of caudate nucleus caused no language disturbance, or only mild word finding difficulty; large lesions were associated with hypophonia. Extension of the lesions into the anterior limb of the internal capsule or into the superior periventricular white matter also did not usually compromise language or speech. However, extension into the anterior or anterior–superior periventricular white matter produced a transcortical motor aphasia, and if lesions were more extensive anteriorly they caused a global or non-fluent aphasia with dysarthria. More posterior extension of damage to

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**Fig. 1** Coronal brain section. Deep nuclei and fibre bundles said to be important in motor control. (Della Sala S et al., 1992; with permission of Oxford University Press.)
periventricular white matter caused a fluent aphasia or a Wernicke's aphasia, often with dysarthria. Alexander et al. (1987) attributed these various patterns of disturbances of language and speech to subtle variations in damage to multiple white matter pathways, including the superior longitudinal fasciculus, frontostriatal reciprocal connections, cortico-bulbar pathways, those from supplementary motor area to Broca's area, and anterior callosal pathways. They concluded that there is little evidence that striatal structures themselves are important in the aphasia syndromes (Alexander et al., 1987).

Mega and Alexander (1994) have subsequently characterized the disturbance of language caused by capsulostriatal vascular lesions in 14 cases as impairment of generating language with anomia, despite a generally grammatical and fluent conversational or responsive language output. Repetition, reading and comprehension are generally intact. Many patients also have dysarthria and hypophonia. Many of the components of the core subcortical aphasia profile are reminiscent of those encountered in conduction aphasia, and may be attributed to damage to the superior longitudinal fasciculus. Thus, fluent speech with preserved comprehension is a feature of conduction aphasia, but preserved repetition, dysarthria and hypophonia are not. Therefore, Mega and Alexander (1994) concluded that damage to frontostriatal circuits also plays a critical role in such 'deep aphasias'. Such damage may account for the difficulty in generating language, the dysarthria and hypophonia.

Some of the features of the subcortical aphasia profile may also be found in transcortical motor aphasia, which is most often due to left anterior cerebral artery infarctions, left frontal lobe lesions anterior to the precentral gyrus, or to deep lesions of the left hemisphere involving the basal ganglia (Wallesch, 1985). The striking preservation of repetition also distinguishes transcortical motor aphasia from conduction aphasia. Presumably, repetition remains normal because the causative lesions do not disconnect the perisylvian language circuit from Wernicke's area via the superior longitudinal fasciculus to Broca's area, but they disrupt connections from other cortical areas into the language circuit. Indeed, Alexander et al. (1987) suggest that damage to the anterior–superior periventricular white matter may reduce language output by destruction of pathways from the supplementary motor area to Broca's area. More extensive lesions result in classical transcortical motor aphasia.

In conclusion, as far as 'deep aphasia' is concerned, lesions of the basal ganglia and adjacent white matter rarely cause a classical conduction aphasia even if they involve the superior longitudinal fasciculus. More often, especially with anterior lesions, there is paucity of spontaneous speech, preservation of repetition, dysarthria and hypophonia. Nevertheless, such lesions often intrude into the peristriatal white matter and must damage the superior longitudinal fasciculus.

**The superior longitudinal fasciculus and apraxia**

The superior longitudinal fasciculus is also implicated in apraxia. Indeed, classical concepts have considered that damage to the superior longitudinal fasciculus causes not only conduction aphasia, but also bilateral ideomotor apraxia (see Geschwind, 1965; For. 2). According to Geschwind, verbal commands must first be heard (Heschl's gyrus), then transcoded into language (left Wernicke area), and then transmitted via the same superior longitudinal fasciculus as is used in language to the left premotor cortex which is dominant for learned movements, in the same way as the left Broca's area is dominant for motor speech. If the motor act involves the right extremities then the left premotor area activates the appropriate motor neuron pool in the left motor cortex. If, however, the left extremities are involved, the information has to be transmitted via transcallosal pathways to the right premotor area, and subsequently to the right motor cortex. Lesions involving the superior longitudinal fasciculus are usually found in its course in the parietal operculum.

Benson et al. (1973) described three patients with conduction aphasia, two of which had severe ideomotor apraxia involving both the right and left extremities and the face, in addition to their language disorder. The other patient did not show any signs of apraxia. Post-mortem examination revealed that the two patients with apraxia had entirely suprasylvian lesions involving the white matter deep to the parietal operculum, while the non-aparctic subject had lesions confined to Wernicke's area. Benson et al. (1973) therefore concluded that the presence of ideomotor apraxia in conduction aphasia implicates suprasylvian white matter pathology. Tanabe et al. (1987) reported another three cases of conduction aphasia and apraxia (Cases 2 and 3) caused by a small infarct almost exclusively confined to the superior longitudinal fasciculus in the parietal operculum between the
The relationship between orofacial and limb apraxia. The first model considers apraxia as a unitary motor disorder, and they pointed out that there are at least two possible models depicting this relationship. Raade et al. (1991) revealed no significant association between orofacial and limb apraxia. Furthermore, there was clear evidence that they were differentially influenced by the nature of the movement (intransitive versus transitive), that they exhibited different proportions of error types and that they demonstrated different neuroanatomy. Raade et al. (1991) concluded that these results support the non-unitary model and suggest that the underlying mechanisms of orofacial and limb apraxia are, at least in part, functionally independent.

**Orofacial apraxia**

Ideomotor apraxia was the most common type of apraxia described with basal ganglia lesions, but orofacial apraxia was also reported frequently. Orofacial apraxia, a term used as a shorthand for ‘apraxia of the cranial musculature’ (Geschwind, 1965), is probably the most common of all apraxias. It may occur with lesions present in the inferior frontal gyrus, perisylvian-central area, the insula and the striatum (Raade et al., 1991). This specific involvement of frontal cortical and subcortical grey matter structures has been emphasized in previous studies (Tognola and Vignolo, 1980). Geschwind (1965) stated that the pathway for facial movements to verbal command probably runs from the posterior speech area via the superior longitudinal fasciculus to the association cortex lying anterior to the face area. The pathway for facial movements in response to visual input also passes from parieto-occipital areas through the inferior parietal region to an area anterior to the Rolandic face region. Lesions of the supramarginal gyrus region thus lead to facial apraxia. Orofacial apraxia, particularly to verbal and visual stimuli, is often associated with conduction aphasia. According to Geschwind (1965) the orofacial apraxia accompanying conduction aphasia results from the same lesion probably low in the superior longitudinal fasciculus. He further stated: ‘... the fact that limb apraxia may be absent suggests that the fibres intended for facial ‘association cortex’ run lower down than those going to the motor association cortex for the limbs’.

The co-occurrence of orofacial and limb apraxia has been investigated by Raade et al. (1991). They pointed out that there are at least two possible models depicting the relationship between orofacial and limb apraxia. The first model considers apraxia as a unitary motor disorder that transcends the output modalities of both orofacial and limb praxis. Indeed, Poeck (1985, p. 103) explicitly stated that ‘... the traditional distinction between oral and limb apraxia appears quite artificial’. In the second model, orofacial and limb apraxia are thought to be distinct. Two separate praxis systems involved in planning and controlling orofacial and limb movements, respectively, are presumed. A high degree of concordance and similarity between the two apractic disturbances would support the unitary model, whereas the presence of specific qualitative and quantitative differences would favour the non-unitary model. The study of Raade et al. (1991) revealed no significant association between orofacial and limb apraxia. Furthermore, there was clear evidence that they were differentially influenced by the nature of the movement (intransitive versus transitive), that they exhibited different proportions of error types and that they demonstrated different neuroanatomy. Raade et al. (1991) concluded that these results support the non-unitary model and suggest that the underlying mechanisms of orofacial and limb apraxia are, at least in part, functionally independent.

**Apraxia and aphasia are related but independent**

The common occurrence of aphasia and apraxia, orofacial and limb ideomotor, has been an area of interest since Liepmann’s historical descriptions. Again, it was Geschwind (1965) who stated that the association of orofacial apraxia with lesions near to Broca’s area is readily understood. Facial movements cannot be carried out to command or visual stimuli, because the lesion has cut off connections to the left face area and cut off the origin of the callosal fibres to the right face area (Geschwind 1965). Impairment in the performance of orofacial movements is generally more common than limb apraxia in patients with aphasia. Lesions producing aphasia usually destroy the association areas and callosal fibres involved in face movements but will often spare those associative connections running more superiority which are involved in limb movements (Geschwind, 1965). As De Renzi et al. (1980) pointed out, the close association between apraxia and aphasia raises the question of whether the language disorder per se plays a specific role in the impairment of motor behaviour or whether this relationship is due only to the anatomical contiguity of the neural substrates subserving the two performances. The first hypothesis seems rather unlikely. However, a lesion producing apraxia may encroach upon cerebral areas known to be important for language. Descriptions of patients who are only mildly aphasic but severely impaired in their motor performances or vice versa, strengthen the belief that the two functions may be separately impaired.

Kertesz et al. (1984) studied the functional and anatomical relationship between aphasia and apraxia in 177 patients with CT evidence of left hemisphere stroke. They found that apraxia was strongly associated with aphasia, but correlation...
of apraxia with aphasia varied with the type of aphasia. They also found six cases of severe aphasia in whom praxis was spared, one of whom had a small temporal lesion associated with severe Wernicke’s aphasia and the others had large frontoparietal lesions. It was suggested that praxis sparing could be due to the bilateral representation of the visuokinaesthetic motor engrams and functionally active right parietofrontal connections. Selnes et al. (1982) described the opposite, namely a case with severe apraxia and mild, recovering aphasia. They postulated bilateral representation of language but unilateral visuokinaesthetic motor engrams.

In conclusion, it seems likely that the common association between impaired language and motor behaviour is because the neuroanatomical systems involved with aphasia and apraxia are separate but closely related. Both occur most frequently with a middle cerebral artery occlusion that damages both language and praxis areas. Cerebrovascular disease therefore impairs both language and praxis networks, whereas neurodegenerative conditions such as Alzheimer’s disease seem more selective for the praxis network (Kertesz et al., 1984).

**Thalamic apraxia**

There remains the issue of thalamic lesions causing apraxia. We encountered 12 reports of thalamic lesions that did not apparently involve white matter, and in eight cases apparently confined to the thalamus, associated with apraxia, nearly always ideomotor apraxia. There are good reasons to consider that this is a true ‘subcortical apraxia’. Shuren et al. (1994) reported a patient with ideomotor apraxia following a left posterior cerebral infarct with lesions in the left medial occipital lobe, inferoposterior temporal lobe, and the pulvinar nucleus of the thalamus. The authors pointed out that limb ideomotor apraxia is not usually found after infarction in the territory of the posterior cerebral artery, or after damage to the thalamus. Shuren et al. (1994) note that the pulvinar has connections with both the inferior parietal cortex and the lateral prefrontal cortex, cortical regions known to be important for praxis. They therefore suggested that ideomotor apraxia in their case was due to the damage in the pulvinar.

The same authors also analysed the three cases of limb apraxia associated with infarcts in the distribution of the tuberothalamic artery described by Graff-Radford et al. (1984). Shuren et al. (1994) suggested that apraxia in these latter cases may have been due to damage to the frontal lobe—inferior thalamic peduncle—nucleus reticulararis—centromedian system. Nadeau et al. (1994) have also recently described a case of severe ideomotor apraxia with extensive infarction of the left thalamus involving the ventrolateral, ventral posterolateral, and lateral posterior nuclei, with some involvement of the pulvinar.

**Limb-kinetic apraxia**

If basal ganglia lesions alone do not cause classical ideomotor or ideational apraxia, there remains the issue as to whether some of the motor deficits seen with basal ganglia diseases could be considered as apractic.

As stated in the historical section, Liepmann postulated that limb-kinetic apraxia is due to damage to the contralateral anterior and posterior area of the central fissure, a region known as ‘sensomotorium’. In contrast to ideomotor and ideational apraxia, limb-kinetic apraxia is characterized by a marked breakdown of previously skilful movements (Liepmann, 1920; Yamadori, 1982). Movements are described as slow, clumsy, awkward, temporally disordered and decomposed. The apractic disorder is more obvious when making precise and fine finger movements, but can also be seen when making gestures, imitating postures and using objects (Heilman and Rothi, 1985). This limb-kinetic apraxia pattern can be observed after localized lesions to both the precentral and the postcentral cortical area, in both humans and monkeys. Shiota and Kawamura (1994) analysed 10 cases of limb-kinetic apraxia caused by lesions in the central region. All patients showed clumsiness of intentional movements, imitation and object use in the hand contralateral to the lesion. Patients who had damage confined to the precentral gyrus revealed the same motor-behavioural symptoms as those with damage to the postcentral gyrus. However, patients with lesions in the postcentral gyrus showed also sensory disturbances and presented with greater difficulty in their hand movements when there were no visual cues. Similar motor behaviour has been seen in awake monkeys after injecting muscimol (a potent gamma-aminobutyric acid agonist) in their precentral or postcentral gyrus (Iwamura, 1994). The animals adopted motor strategies for using their limbs that looked similar to untrained motor actions. It was hypothesized that the somatosensory cortex sends organized information to the premotor region through cortico-cortical fibre connections. This information is thought to be essential in order to achieve precise and complex motor behaviour (Shiota and Kawamura, 1994). Okuda et al. (1992) reported two patients with slowly progressive limb-kinetic apraxia. One patient with mainly right-sided limb-kinetic apraxia had decreased cerebral blood flow in the left central region between frontal and parietal cortices; the other patient with left-sided apraxia had decreased cerebral blood flow in the right parietal cortex.

The described characteristics of limb-kinetic apraxia, including slowness, clumsiness and awkwardness of movement, along with loss of kinetic melody, disturbances of timing and lack of precision, could be applied to the limb motor deficits of parkinsonism. Indeed, the description of lack of precision of movement, despite accurate selection of sequences correctly orientated in space used by Heilman and Rothi (1985) to describe limb-kinetic apraxia also describes some of the deficits of movement seen in Parkinson’s disease. Patients with the latter condition also exhibit slowness (bradykinesia), reduced amplitude and fatigue on repetition (hypokinesia), and freezing or motor blocks (akinesia). However, patients with Parkinson’s disease select the correct individual movements, and can put together sequences of
movements, albeit with distorted timing (Marsden, 1984, 1987).

Likewise some of the deficits due to lesions of the supplementary motor area and premotor cortex, to which the basal ganglia project, are reminiscent of those seen in basal ganglia disease. Thus, bilateral lesions of the supplementary motor area causes an akinetic–abulic syndrome (Foerster, 1936), while unilateral lesions produce a contralateral hemiakinetik syndrome (Laplane and Degos, 1983). Rothi et al. (1991) and Watson et al. (1986) consider that the supplementary motor area plays an important role in trans-coding time–space representations of the action–output lexicon holding visuokinaesthetics motor engrams into correct innervatory patterns. Disturbances of the correct innervatory patterns may cause limb-kinetic apraxia. Lesions of the basal ganglia, or of its connections with the supplementary motor areas, may therefore be expected to cause dysfunction of the latter regions, expressed as limb-kinetic apraxia.

There is obviously some confusion over the use of terms to describe the motor deficits seen after basal ganglia and motor cortical area lesions. The patient with Parkinson’s disease is said to be akinetic (subsuming bradykinesia and hypokinesia); the patient with supplementary motor area damage is said to exhibit limb-kinetic apraxia. Yet the two deficits have much in common, although they are not identical. Furthermore, pathology affecting both the basal ganglia and motor cortical regions, as in corticobasal degeneration (Rinne et al., 1994), produces a motor deficit greater than that seen in Parkinson’s disease or motor cortical damage alone. Such patients with corticobasal degeneration have not only a profoundly akinetic rigid limb, but also the inability to execute correct movements spontaneously or to command, or to copy postures which are readily recognized, as well as other phenomena, such as alien limb behaviour.

Perhaps the term limb-kinetic apraxia needs to be retained, but what it defines must be characterized by exact measurement of motor parameters. Akinnesia, bradykinesia and hypokinesia can be measured. The selection of the correct motor response (‘what to do’), the correct movements to achieve the chosen response (‘how to do it’) and the correct timing of action (‘when to do it’) can also be measured. Neurophysiological and PET activation studies have shown that ‘what to do’, ‘how to do it’, and ‘when to do it’ involve a distributed system including the network of the dorsolateral prefrontal cortex, the supplementary motor areas, the anterior cingulate regions and the lateral premotor cortex (M. Jahanshahi, H.J. Jenkins, R.G. Brown, R.E. Passingham, D.J. Brooks and C.D. Marsden, unpublished results).

Of these structures, the supplementary motor area (Brodmann’s area 6), localized in front of the primary motor area (Brodmann’s area 4) on the mesial surface, is thought to play a distinct role in higher order motor control. Penfield and Welch (1951) showed that stimulation of the supplementary motor area in monkeys produced contralateral movements of the distal upper limb that were far more complex than the localized movements of individual body parts induced by stimulation of the primary motor area. Fulton (cited in Foerster, 1936), on the basis of his animal work, distinguished between area 4 and area 6 syndromes. The former was characterized by the fact that after recovery of an initial contralateral flaccid paresis the performance of even complex finger and hand movement sequences was preserved. In contrast, monkeys with isolated lesions of area 6 showed after an initial contralateral spastic paresis, forced grasping and a permanent or long-lasting impairment of all fine, precise, and sequential hand and finger movements. Kleist (1934), although not referring at all to Fulton’s experimental data, described a special form of praxis disturbance that closely resembled Fulton’s ‘loss of skilled movements’. This kind of apraxia, which he named ‘gliedkinetische innervatorische Apraxie’ (limbkinetic innervatory apraxia), was found in association with lesions confined to area 6a. Kleist’s descriptions of the movements being clumsy and awkward, as well as being spatially and temporally disordered, closely resemble the descriptions above. A major feature of this kind of motor behaviour was that the correct intent or concept of motor action was clearly identifiable at all times and in all movements. The patients knew what to do, they just could not do it correctly. The more complex the motor action the more evident the praxis deficit. Kleist (1934) was convinced that even if lesions in area 6 are associated with damage to area 4, the motor impairment seen in these patients resulted from damage to a hierarchically higher motor cortical area, such as area 6aa or its connections.

In conclusion, damage to the distributed frontal system comprising supplementary motor area, dorsolateral prefrontal cortex, the anterior cingulate regions and the lateral premotor cortex may cause the specific deficits of motor choice, action and timing, to which the term limb-kinetic apraxia may be applied. If similar defects occur in patients with basal ganglia disease, then they too may be said to exhibit elements of limb-kinetic apraxia in addition to their classical akinesthesia, bradykinesia and hypokinesia.

**Conclusions**

This analysis of apraxia due to deep subcortical lesions highlights the conclusion that damage to basal ganglia *per se* (putamen, caudate nucleus and globus pallidus) does not cause ideomotor or ideational apraxia. Additional involvement of periventricular, especially peristriatal white matter, plays a crucial role in the development of ideomotor apraxia after such deep lesions. Where basal ganglia are involved, ideomotor apraxia is seen most often in lesions associated with the lenticular nucleus or putamen. Lesions affecting the putamen nearly always intrude into the adjacent lateral white matter to involve association fibres, in particular those of the superior longitudinal fasciculus. Damage to such association fibres would explain why lesions in the region of the basal ganglia causing apraxia are nearly always in the left hemisphere and predominantly cause ideomotor apraxia,
which is often bilateral or involves the left hand where there is a right hemiparesis. The special role of the thalamus in disturbances of higher order motor behaviour remains to be determined. Finally, it is suggested that damage to motor cortical regions may cause specific deficits in the choice of motor action, its execution and its timing to which the term limb-kinetic apraxia might be applied. If similar deficits also occur in patients with disease of the basal ganglia, which project to frontal motor cortical areas, over and above akinesia, bradykinesia and hypokinesia, then such patients may also be said to exhibit limb-kinetic apraxia.

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Basal ganglia and apraxia


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### Appendix: authors and cases included in the metanalysis

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*Continued on next page*
**Appendix: continued**

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Nc = no. of cases; C = case in the original paper; a = anterior; p = posterior; v = ventral; m = medial; pa = parietal; oc = occipital; fr = frontal; BG = basal ganglia; LN = lenticular nucleus; CN = caudate nucleus; PUT = putamen; GP = globus pallidus; IC = internal capsule; ALIC = anterior limb of IC; PLIC = posterior limb of IC; TH = thalamus; ST = subthalamic nucleus; PVWM = periventricular white matter; AR = auditory radiation; TI = temporal isthmus; CC = corpus callosum; EC = external capsule; S = splenium; HG = hippocampal gyrus; IMA = ideomotor apraxia; OA = oral apraxia; IA = ideational apraxia; GPe = globus pallidus pars externa; In = insula. *Patient 6 was severely impaired in imitating oral and limb movements the first 10 days of the disease (isch., left CN, ALIC, LN).