Deconstructing apraxia: understanding disorders of intentional movement after stroke

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Impairments in praxic functioning are common after stroke, most frequently when the left hemisphere is affected. Several recent studies of apraxia after stroke have made advances in understanding the right hemisphere contribution to praxis, particularly for the performance of novel actions. Moreover, quantitative lesion analysis in stroke patients indicates the importance of cortical regions such as the intraparietal sulcus and the middle frontal gyrus for subserving praxic function. Complex neuropsychological models have been developed to account for the many dissociations observed in the types of errors observed in stroke patients. Relatively lacking, however, are models that attempt to relate the neurological data to what is known about praxis from functional neuroimaging in normal subjects and from physiological studies in the monkey. Moreover, a coherent interpretation of the results of apraxia studies remains hampered by the lack of a standard testing instrument to assess the nature and severity of apraxic impairments in the groups tested. Curr Opin Neurol 15:71–77.

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Introduction

Apraxia is defined as a deficit in the ability to understand an action or to perform an action in response to verbal command or in imitation (e.g. wave goodbye, pantomime use of a hammer), in the absence of basic sensory or motor impairments. This deficit may be conceptualized as a deficit in the mental representation of specific aspects of an action. We will use the term apraxia here, although most patients show only partial effects and might therefore be considered *dyspraxic*, rather than apraxic. Although apraxia is most commonly associated with strokes affecting the left parietal lobe, it may also occur in lesions to the right parietal lobe, the temporal or frontal lobes, and even subcortical regions including white matter and the basal ganglia [1•]. Apraxia and aphasia often occur together, but apraxia can occur in the absence of aphasia and is, therefore, considered a distinct disorder [2].

Here, we present an overview of current models of apraxia followed by a review of the studies of apraxia published in the neurological literature over the past year. We also provide an analysis of the neuroimaging literature from the past year that is relevant to the understanding of normal and impaired praxis.

Models of apraxia

The earliest theories of apraxia formulated by Liepmann postulated that the left parietal cortex is important for the formation of motor programs that specify the spatial and temporal sequence of movements composing an action [3,4]. Ideational apraxia would arise when the motor programming area is destroyed by damage to the supramarginal gyrus, impairing the conceptual representation of an action and leading to deficits in using tools or performing an action to verbal command while imitation is spared. Performance of meaningful action sequences (e.g. lighting a candle) was also used as a test for ideational apraxia. Ideomotor apraxia would arise when the motor programming area is disconnected from the premotor and motor regions, so that the patient can conceptualize but not actually execute the action, demonstrating spared recognition of tools but deficient ability to use them appropriately or to imitate actions.

Since that time, a great number of terms have evolved for classifying different subtypes of apraxia based on subtle dissociations in praxic abilities. The result is confusion in the literature in which the same term may

Table 1. Glossary of apraxia terms

Type of apraxia	Definition
Buccofacial apraxia	Impairment in performing mouth or face actions on verbal command or imitation (see also orofacial apraxia).
Conceptual apraxia	Form of apraxia in which the concept of the action is lost; characterized by impaired ability to use tools and to understand meaningful gestures.
Constructional apraxia	Inability to assemble component parts into a coherent whole
Ideational apraxia	Impairment in the sequential use of multiple objects. Traditionally, also used to refer to impairment in the concept of an action. The term conceptual apraxia (see above) was coined to distinguish between these two dissociable impairments.
Ideomotor apraxia	Impairment in the performance of skilled movements on verbal command or in imitation; most commonly characterized by spatial or temporal errors in movement execution.
Limb apraxia	Usually used to refer to ideomotor apraxia of the limbs; frequently includes impaired performance of actions that also depend on the hands and fingers.
Limb-kinetic apraxia	Slowness and stiffness of movements with a loss of fine and precise movements.
Optical apraxia	Impairment in performing saccadic eye movements on command
Orofacial apraxia	Impairment in performing mouth or face actions on verbal command or imitation (see also buccofacial apraxia).
Speech apraxia	Selective impairment in ability to produce speech sounds
Tactile apraxia	Impairment of hand movements for the use of and interaction with an object, in the presence of preserved intransitive movements.
Unimodal apraxia	Any form of apraxia that is specific to actions demonstrated in a single modality, e.g. visual, but not auditory.

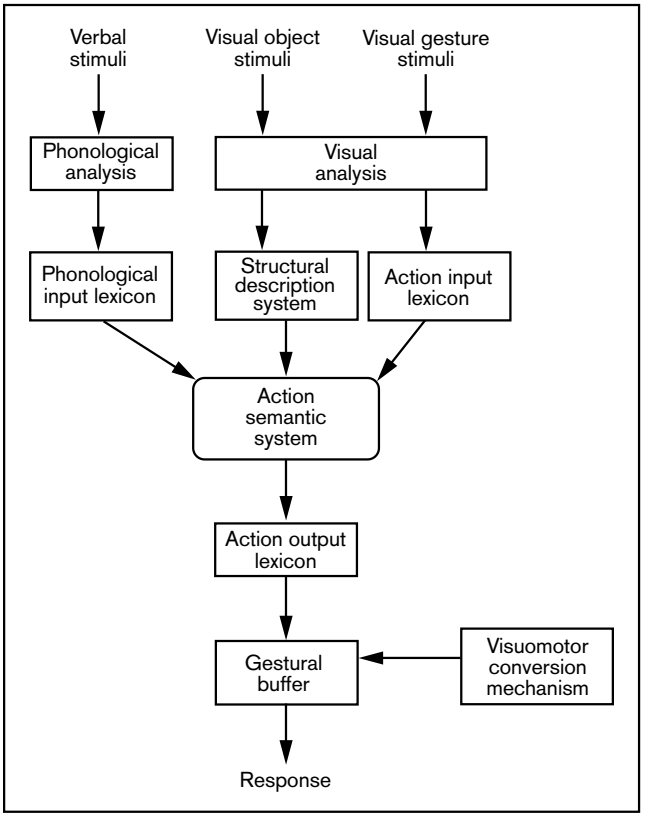
be used differently by different investigators. Table 1 provides the most commonly accepted definitions for a variety of different disorders labelled as apraxia.

The most commonly used term now is ideomotor apraxia. In its strictest sense, ideomotor apraxia refers to the impaired reproduction of meaningful or learned actions, although the ability to perform or imitate meaningless actions is also deficient in some cases. The use of the term ideational apraxia is less common now than when it was first conceived due to difficulties in establishing it as a valid and coherent entity distinct from the symptoms ascribed to ideomotor apraxia or deficits in executive functioning. Researchers soon saw a need to discriminate between sequencing errors, referred to in the traditional way as ideational apraxia, and impairments in action recognition, termed 'conceptual apraxia' [5]. Recent work comparing impairments in single-object and multiple-object use provides another illustration of the difficulties with the traditional concept of ideational apraxia. Tests of multiple-object use (e.g. lighting a candle) were originally designed to elicit sequencing, omission and perseverative errors viewed as characteristic of ideational apraxia, whereas single-object tests (e.g. using a key) were intended to elicit the spatiotemporal errors in tool use that characterized ideomotor apraxia. Correlations and factor analyses of these two tasks within the same group of patients, however, indicate that both appear to involve similar underlying processes [6•]. Moreover, multiple-object actions may be performed more easily than single-object actions by some apraxic patients, and even by normal controls, because the association between related objects may cue correct performance.

A wealth of case reports have demonstrated dissociations in the ability of individual patients to recognize versus use tools, imitate meaningful versus meaningless actions, perform transitive versus intransitive actions, and perform limb versus orofacial actions [7,8•]. These observations are interpreted as reflecting modularity in the praxis system and a number of fairly complex models of apraxia have now been developed (Fig. 1). Separate semantic and non-semantic pathways allow for dissociations in the ability to represent meaningful versus meaningless actions; separate input and output lexicons account for differences in the ability to conceptualize actions and the ability to perform them; and separate input pathways for verbal and visual stimuli explain the dissociation between the ability to perform an action on command versus in imitation.

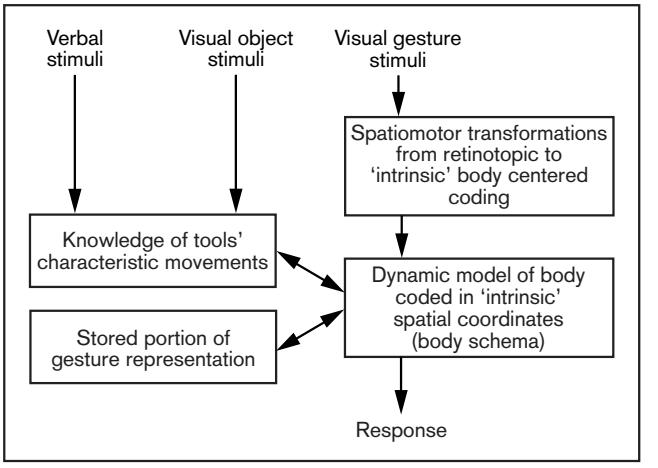
Despite the ability of the model depicted in Fig. 1 to account for the dissociations in apraxic impairments observed in patients, some features deserve further scrutiny. First, the model was developed from the study of ideomotor apraxia of the limb and, thus, emphasizes praxis of meaningful actions. The non-semantic pathway was included in the traditional models because meaningful actions can be imitated without awareness of their meaning. Indeed, the terms 'semantic' and 'non-semantic' were borrowed from the aphasia literature. However, it may be more accurate to refer to meaningful actions as well-practised or learned actions that may be already represented in their entirety in an individual's motor repertoire, and to meaningless actions as actions involving novel motor sequences that must be analyzed and constructed from existing movements. In this context, it is clear that the non-semantic pathway, often termed the 'direct' pathway, is notably underspecified in these models and may be far from direct. In fact, researchers studying apraxia for meaningless actions are discovering that even novel or meaningless actions with no pre-learned representations may be represented at a conceptual level under certain conditions [9,10••]. It is not yet known whether the conceptual representation of meaningless actions would use some of the same pathways as the representation of meaningful actions

Figure 1. Standard model of limb praxis



Derived from Rothi *et al.* 1991 [7] and Cubelli *et al.* 2000 [8•].

Figure 2. Revised model of limb praxis, inwhich dynamic body-schema representations interact with stored representations of learned actions



Derived from Buxbaum *et al.* 2000 [11••].

and whether it would require an expansion of the 'direct' pathway. A recent model of the praxis system proposed by Buxbaum and colleagues (see Fig. 2) provides a very different way of describing the relationship between meaningful and meaningless actions. This improved model proposes an interplay between a dynamic body-centred representation of actions and stored representations of learned actions [11••]. From a physiological perspective, these changes improve the plausibility of the model, although there is still no attempt to explain the possible relationships between the functions represented and the neuropsychological mechanisms that underlie them.

Second, the separation of the input and output lexicons may prove untenable in light of the recent discovery of a so-called mirror system in the ventral premotor cortex and the posterior premotor cortex of the monkey brain. Neurons in area F5 of the premotor cortex tend to alter their pattern of firing during the performance of a specific action, such as precision grip, but also show additional response properties. A subset of neurons in this region, the mirror neurons, also respond to the mere observation of the represented action performed by someone else [12]. The existence of mirror neurons suggests that the brain may represent observed actions by mapping them directly onto the same substrates that are used to execute an action. Recent work using functional magnetic resonance imaging and magneto-encephalography has demonstrated the existence of a similar mirror system in the human brain [13,14].

Lesion correlates of apraxia: interhemispheric

Roughly 30% of patients in the acute phase of stroke show evidence of apraxia [3,15], but the incidence is higher after damage to the left hemisphere (50%) than to the right hemisphere (<10%) [16]. Nevertheless, considerable variability in this estimate is found across studies due to the lack of standardized assessment tools and wide variations in criteria for diagnosing the disorder.

Several recent studies have focused on whether different patterns of apraxic impairment are associated with left hemisphere damage or right hemisphere damage. It appears that comparable levels of impairment may be seen across both groups for certain types of apraxic impairment. In a study of facial apraxia after stroke, patients with left hemisphere damage made more errors than patients with right hemisphere damage when imitating lower face actions, whereas both groups performed similarly for upper face actions [17•]. In a study of limb apraxia, patients with left hemisphere strokes were more likely than patients with right hemisphere strokes to be impaired at pantomiming the use of a tool in response to verbal command, whereas an equal proportion of left hemisphere and right hemisphere patients were impaired when imitating a pantomime demonstrated by the

experimenter [18•]. Although half of the patients with left hemisphere stroke also had aphasia, the correlation with apraxia was similar for the pantomime and imitation conditions.

These findings suggest an important role for the right hemisphere in praxis, perhaps more particularly for novel actions. The results of studies in which patients imitated meaningless actions may provide a direct test of this hypothesis. A recent investigation of patients with relatively circumscribed lesions to the parietal lobe found that only those with left hemisphere damage are impaired at performing meaningless actions on verbal command or imitation [19]. Unfortunately, the interpretation of this study is problematic since the left hemisphere group was composed primarily of stroke patients whereas the right hemisphere group had tumours, which may have allowed for long-term reorganization of function. Recent studies by Goldenberg and colleagues, however, provide persuasive evidence of the contribution of the right hemisphere to the recognition and imitation of meaningless actions. Patients with left hemisphere strokes were impaired at imitating all meaningless actions and at perceptual matching of meaningless hand-to-head actions [9,20]. In contrast, patients with right hemisphere lesions were impaired at imitating meaningless postures of the fingers of the hand and at perceptual matching of all meaningless actions. Consistent with these findings are the results of a recent study in a patient with callosal resection, in whom the left hemisphere initially had difficulty supporting imitation of finger postures on its own and neither hemisphere could support perceptual matching of finger postures when working on its own. [21]. From this work, it may be concluded that the left hemisphere is important for representing actions in terms of knowledge about the structure of the human body, whereas the right hemisphere participates in the visuospatial analysis of gestures. This suggests that apraxia after right hemisphere strokes results from disruption of a pathway for translating visual input to motor output, which preferentially impairs the representation of novel actions [10••].

Lesion correlates of apraxia: intrahemispheric

Although some studies have not found an association between the locus of the lesion within a hemisphere and the severity of apraxia [22,23] others are now focusing on whether lesions to particular subregions within each hemisphere may be associated with particular subtypes or patterns of apraxic performance. Halsband and colleagues [24•] demonstrated that patients with parietal lesions show the most severe impairments in the recognition and imitation of pantomimed actions and the deficit is particularly severe for left parietal lesions and actions directed toward their own bodies (e.g. brushing hair). In contrast, patients with premotor or precentral lesions were not impaired on the recognition or performance of unimanual pantomimes, but those with damage to medial premotor regions were severely impaired at pantomiming bimanual actions that required different movements from each hand. The importance of medial premotor regions for bimanual actions is consistent with neuroimaging evidence in normal subjects of disproportionately increased activity in supplementary motor area during bimanual compared with unimanual actions [25].

A recent study used quantitative structural image analyses to determine the location of greatest lesion overlap in patients with anterior or posterior strokes. The intraparietal sulcus and the middle frontal gyrus were identified as the regions most important for the imitation of actions [26•]. The use of structural scans to report the extent and overlap of lesions in groups of patients is a relatively new feature of this study that represents an improvement in the analysis of lesion–deficit relationships. The validity of this approach is demonstrated by the similarity between the brain regions identified in this study and those identified in functional neuroimaging studies of imitation in normal subjects [12].

Functional imaging of praxis

Functional neuroimaging studies confirm the importance of the parietal and frontal cortex in the representation of actions under a variety of conditions including action observation, action recognition, observation of graspable objects, imagined action, and imitation [27]. A current goal of such studies is to determine whether the contributions of the parietal and frontal cortex differ, and whether there are specific regions within the parietal or frontal cortex that contribute preferentially to particular aspects of praxis.

To date, the results of neuroimaging studies support the notion that both hemispheres contribute to praxis, although the left hemisphere may be slightly more involved for some tasks. The left hemisphere, particularly the inferior parietal lobule, is more active than the right during perceptual discrimination of meaningless gestures, but when the gestures consist of finger postures, the intraparietal sulcus of the right hemisphere shows greater activity [28•]. Two recent studies found greater activity in the left inferior parietal lobe compared with the right parietal lobe for recognition of transitive actions [29] and tools [30], but these studies used naming tasks and were therefore biased to finding left hemisphere effects. Neuroimaging studies of action imagery, imitation, or observation found increased activity in the left [31], right [13] and bilateral [32•] posterior parietal cortex, respectively. Although increased signal in premotor cortex is more commonly lateralized to the left hemisphere, it has been observed bilaterally in some studies [32•].

Besides providing converging evidence for the neural mechanisms underlying apraxia, neuroimaging studies may also provide new insights into how actions are represented in the brain and how such representations are modulated by specific task demands. A recent functional magnetic resonance imaging study of action observation suggested that actions may be somatotopically represented within the parietal and premotor cortex, with mouth actions being represented ventrally, foot actions more dorsally, and arm–hand actions in between [32•]. Peigneux and colleagues have recently embarked on an ambitious project using positron emission tomography to map each of the components specified by a cognitive model of praxis processing similar to that shown in Fig. 1. Published results to date support the need for elaboration of the 'non-semantic' pathway to involve dorsal parietal regions important for visuospatial processing [33,34].

Little is known about the neural functioning of patients with apraxia, although two case studies showed hypometabolism of the left parietal lobe [35,36]. A recent electrophysiological study measured patterns of slow cortical potentials and of event-related desynchonization of activity in the alpha and beta bands in groups of stroke patients with apraxia or other movement disorders during the execution of self-paced voluntary movements [37•]. Compared with a group of normal controls, two patients with ideomotor apraxia showed electroencephalograph patterns suggestive of decreased activity in left parietal and medial frontal cortex. Unfortunately, no structural images of the lesion site were available for these patients so it is not known whether the actual lesion encompassed one or both of these sites. Analyzing electroencephalographs in combination with structural imaging data would be a powerful tool permitting one to test the effects of a lesion in one brain region on activity in another during tasks relevant to understanding apraxia.

Treatment of apraxia

It should be obvious that the presence of apraxia in the acute phase after stroke is an impediment to rehabilitative therapies aimed at improving ambulation and self-care, since the process of learning new motor sequences and skills usually depends heavily on imitative learning and on mental representation of an action. Moreover, in aphasic patients, the presence of apraxia prevents the teaching of gestural communication as part of therapeutic interventions. Although apraxia usually improves over time following an acute lesion, spatiotemporal errors in imitation or tool use may persist [38]. The presence of limb apraxia, more than any other neuropsychological disorder, correlates with the level of caregiver assistance required six months after a stroke [39], whereas the absence of apraxia is a significant predictor of return to work after a stroke [40]. Therefore, a behavioural training programme focused on the imitation of actions has been developed that appears to produce improvements specific to tests of apraxia in left hemisphere stroke patients [41].

Another therapeutic approach is to teach strategies to compensate for continued apraxic deficits. Using such an approach improvements were reported in activities of daily living, but not on tests of apraxia [42,43]. Unfortunately, these studies did not include a control group for pre- and post-treatment assessment so it is impossible to determine whether the improvements observed were the result of the intervention or part of the natural course of recovery.

Conclusion

There appears to be a consensus within the literature that the left parietal cortex subserves a particularly important component of the praxis system, especially concerned with the knowledge or representation of overlearned actions. It is recognized, however, that damage to cortical and/or subcortical regions outside the left parietal cortex, including the right hemisphere, have also been associated with apraxia and it is assumed that each of these different neural regions makes its own distinct contribution to the representation of action. Progress in describing the unique contribution of each region through the study of brain-damaged patients has been limited by two factors: (a) the variability in the size, location and structures affected by the lesion and (b) testing of a limited range of praxic functions, usually focusing solely on the performance of meaningful gestures. Although meaningful actions are of greater interest to the apraxic patient who must be able to perform activities of daily living, relearning these actions would appear to require complex abilities in visuospatial analysis and the ability to reform action sequences from preserved movement abilities.

Research on apraxia after stroke may continue to advance our current level of understanding of the neural substrates of normal and impaired praxis. We make the following recommendations for future studies: adequate matching of groups with similar brain lesions in terms of aetiology and onset; quantitative analyses of lesion location and extent; assessment of the full range of praxic functions, including imitation of novel actions; separate assessment of different effectors, including mouth, face and foot; and exploration of patterns of neural activity associated with action representation in normal controls and in apraxic patients.

Furthermore, it is now essential to begin the task of relating what is known about apraxia from the study of patients, and about normal praxis from functional

neuroimaging in normal controls, with the wealth of literature describing the physiological mechanisms of action knowledge and representation as observed in non-human primates. Studies of anatomic and functional connectivity will help to constrain our theories of which brain regions may work together systematically to subserve praxis. Lesions studies in non-human primates can be carefully controlled by the experimenter to allow assessment of the effects of very focal unilateral or bilateral damage on action representation. Finally, there is a vast literature describing how single-cell recording techniques have been used to define the specific response properties of neurons throughout the cortex during reaching and grasping, during the presentation of graspable objects and during the observation of actions performed by others [44–46].

The implications of such research for understanding apraxia are self-evident and indeed some physiologists have turned to the neurological research in patients in interpreting their findings. Unfortunately, the neurological literature contains few references to the physiological literature as a framework for understanding the impairments observed in patients. A notable exception is the recent review by Leiguarda and Marsden [47], which contains an impressive attempt at bringing together the literature from both camps. Clearly, the time has come to build a model of praxic functioning in which neurological and physiological data may be understood within a coherent framework. One approach would be to test the validity of neuropsychological models in terms of their ability to map onto plausible physiological substrates. We believe that a multidisciplinary approach will ultimately be most fruitful in terms of understanding the nature, prognosis, and rehabilitation of apraxic impairments seen after stroke.

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