Limb Apraxia

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Synopsis:

Apraxia is a common clinical disorder characterized by an inability to perform complex, familiar actions that cannot be attributed to weakness or elementary sensory loss. The disorder is usually associated with damage to the left parietal or frontal lobe. A variety of different types of apraxia have been identified, ranging from an impairment in dexterity to an inability to construct a motor plan that specifies the sequence of actions required to complete a task such as mailing a letter or making tea. Recent work has demonstrated that apraxia not only has important clinical consequences but also has implications for the understanding of the manner in which action knowledge is represented and motor plans are constructed and executed. Explorations of this disorder promise not only to improve the quality of life of patients with brain lesions but will also contribute to the understanding of neural basis of action.
**Historical Background**

Limb praxis is subserved by a complex multicomponent system that provides a processing advantage to previously experienced, purposive movements. The term ‘apraxia’ was introduced by Steinthal in 1871. While this word, derived from Greek, means *without action*, the term apraxia is used to describe a *decrease or disorder* in the ability to perform skilled movements. Scientific and clinical interest in the disorder dates to the early 20th century, when Liepmann reported patients with cerebral lesions who were unable to gesture to command or, in some instances, to imitation. Subsequently, Liepmann & Maas (1907) described a patient with a lesion of the corpus callosum who was unable to produce gestures with the left hand to verbal command. On the basis of these findings, Liepmann proposed that the left hemisphere was “dominant” for gesture in that it supported the learned “movement formulae” or “time-space-form picture of the movement” which specified the timing, trajectory and content of learned movements.

Liepmann’s ideas were extended by Geschwind (1965), who proposed a specific left hemisphere-based neural circuitry for movement representations. On his account, failure to produce a movement to command was attributable either to a disruption of Wernicke’s area, with resultant failure to understand the command, or to a disconnection of the posterior language areas from motor cortex. A failure to imitate movements was attributed to a lesion involving the arcuate fasciculus, which was assumed to connect the visual association cortex to motor cortices.

**Limb Apraxia Subtypes**

Hugo Liepmann’s description of three major forms of apraxia brought about a ‘paradigmatic shift’ in our understanding of motor control. These three types were “limb kinetic apraxia”, “ideomotor apraxia”, and “ideational apraxia”. To this triad, Gonzalez-Rothi, Heilman and colleagues added another type, termed “conceptual apraxia”. These types of apraxia are described briefly below:
Limb Kinetic Apraxia (LKA). Patients with LKA perform actions with slow, stiff, clumsy movements, and exhibit impairment on tasks requiring rapid independent finger movements, such as rotating a coin between the thumb, index, and middle finger. Errors are more apparent in distal (finger) movements than in proximal movements. LKA is associated with lesions that include the primary motor cortex, premotor cortex or descending corticospinal tract. It frequently occurs in patients with stroke and in degenerative disorders such as progressive supranuclear palsy and corticobasal degeneration.

Ideational Apraxia (IA). Ideational Apraxia (IA) is defined as an impaired ability to carry out a sequence of acts that lead to a goal and that incorporate multiple objects, such as making a sandwich or lighting a candle. For example, a patient with ideational apraxia might attempt to seal an envelope prior to inserting the letter. Another type of error exhibited in this disorder is illustrated in Figure 1.

Figure 1 about here

Ideational apraxia is most frequently induced by bilateral damage and degenerative dementia. Injury to the frontal lobes is often also associated with temporal order processing deficits as well as impaired working memory, and thus one of the critical foci of dysfunction in ideational apraxia may be in frontal-subcortical systems. The strongest predictor of errors in multi-step, naturalistic action is overall severity of cognitive impairment, and not lesion location.

Conceptual Apraxia. Patients with conceptual apraxia make content errors in complex action – that is, they substitute incorrect objects or movements in their actions. For example, patients with conceptual apraxia may eat with a toothbrush. In some cases, underlying deficits in knowledge of specific tools or objects or the association of tools and objects has been demonstrated; these patients may misuse objects because they have lost knowledge regarding the function of the
Conceptual apraxia also frequently co-occurs with ideomotor apraxia, and it has not been established whether these disorders can be reliably disambiguated.

Deficits in conceptual action knowledge have been associated with the dominant posterior parietal lobe and/or temporal parietal junction. On the other hand, errors apparently attributable to conceptual deficits frequently occur in patients whose lesions entirely spare brain regions typically associated with conceptual action knowledge (e.g., right parietal cortex).

**Ideomotor Apraxia (IMA).** Ideomotor apraxia (IMA) is a common disorder of complex skilled action not attributable to weakness, incoordination, or other elemental sensory or motor impairments. It is typically observed in individuals who have suffered left hemisphere strokes; IMA is observed in the actions of the “unimpaired” left hand of approximately 50% of patients with left hemisphere stroke, and commonly persists for at least 1 year after stroke. IMA is also common in Alzheimer Disease and in Cortico-basal Degeneration. In stroke, it is usually a consequence of damage to the left inferior parietal lobe (and on occasion, adjacent intraparietal sulcus and superior temporal gyrus), but has also been observed following left dorsolateral prefrontal, callosal, and subcortical damage (see Figure 2).

IMA is usually diagnosed on the basis of spatiotemporal errors in the production of gesture pantomime both to sight of objects and upon imitation of others. That is, IMA is typically seen when a patient is asked to show how an object (e.g., scissors) would be used or when the patient is asked to copy a gesture produced by the examiner. Kinematic analyses have revealed that IMA patients pantomime skilled tool-use movements with abnormal joint angles and limb trajectories, and uncoupling of the spatial and temporal aspects of movement (see Figure 3).
Spatiotemporal errors persist to a lesser degree with actual tool use. The deficit is not restricted to meaningful movements, and has also been observed in meaningless postures and sequences. IMA is also associated with cognitive deficits in declarative knowledge of the manipulation actions appropriate to objects, impairments in mechanical problem-solving, deficits in motor planning, and difficulty learning new gestures.

The disorder may be attributed either to damage to stored spatio-temporal gesture representations in the left parietal lobe, sometimes called “visuokinesthetic engrams”, or to disconnection of intact movement representations from motor output. The integrity of gesture representations is thought by many investigators to bear upon the integrity of gesture recognition. In the case of damage to the representations, patients have impaired knowledge of the appropriate motor action to perform, as evidenced by deficits in gesture recognition (Representational IMA). In the case of disconnection of intact engrams, patients have unimpaired knowledge of appropriate gestures, as evidenced by intact gesture recognition and ability to discriminate correct from incorrect gestures, but nevertheless perform with spatiotemporal errors (Dynamic IMA).

In Representational IMA, inability to discriminate correctly from incorrectly performed meaningful object-related hand movements correlates strongly with ability to produce the same movements, suggesting that the same representations may underlie both. Additionally, Representational IMA patients are significantly more impaired when producing object-related than symbolic, non-object related movements. This in turn suggests that the damaged system underlying Representational IMA is specialized for movements related to skilled object use.

Disconnection and Dissociation Apraxias. Several apraxia patterns indicate that aspects of input to and output from the skilled action system are dissociable. Verbal-motor dissociation apraxia
refers to a pattern of impairment in which patients are unable to gesture in response to command, despite adequate comprehension and unimpaired ability to gesture to imitation. Heilman and colleagues posited that the lesion responsible for this apraxia subtype was in the angular gyrus, but were unable to obtain neuroimaging data. Another reported pattern are the tactile-motor and visuomotor dissociation apraxias, in which patients fail to gesture appropriately when holding tools or viewing tools, respectively, despite unimpaired object recognition and better gesture performance in the unaffected modality. In response to these and other patterns of dissociation, Gonzalez-Rothi, Heilman and colleagues proposed a detailed diagrammatic model of IMA (see Figure 4). Theoretical “lesions” at various loci in the model appear to explain many of the observed dissociations.

Outstanding Issues in Diagnosis of Apraxia Subtype

Relevance of recognition and imitation deficits for diagnosis of IMA vs. IA/Conceptual Apraxia

Historically, gesture recognition and imitation have both been used to distinguish between IMA and IA/Conceptual Apraxia. On Liepman’s account, patients with IA fail to reliably activate gesture engrams. Consequently, they perform normally when provided with the ‘idea’ of the movement; that is, when they are asked to imitate the movement of another person. Liepman believed that by contrast, patients with IMA suffered a disconnection of an intact idea (time-space-form picture of the movement) from motor innervatory patterns. Thus, on Liepman’s account, providing IMA patients with the ‘idea’ in the form of a gesture to imitate would not be of benefit.

On many contemporary accounts, it is Representational IMA patients who fail to reliably
activate gesture representations and who therefore may be able to imitate gestures. Recent evidence indicates that imitation may be accomplished either via gesture engrams (the so called “indirect” or “semantic” route), or by way of a “direct route” to action that enables imitation without access to meaning. (The direct route may bear a relationship to the putative “mirror neuron” system to be discussed below). Therefore, the ability to imitate may depend upon the integrity of each of these routes. On the other hand, there is also evidence that the direct route is not used for meaningful gestures even when it is intact, suggesting that there may be obligatory activation of the semantic route whenever familiar gestures are viewed.

Disagreement persists on whether gesture recognition problems signify IMA or IA. However, recent evidence from monkeys and humans indicates that the same representations are likely used for action recognition and production. In the macaque, cells in the inferior parietal lobule and in a sector of premotor cortex corresponding to Brodmann’s Areas 44 and 45 in humans respond both when the monkeys produce actions and when they observe the same specific actions performed by others (“mirror neurons”). In humans, there are strong correlations between action production and recognition for the same items. This suggests that gesture recognition problems may reflect degraded or inaccessible sensorimotor representations, a characteristic of IMA.

**Relationship of object knowledge to gesture representations.** Continued work is required to clarify the relationship of knowledge of appropriate object-oriented actions to the gesture engram system. In an influential paper on IA, De Renzi and Lucchelli proposed that the problem underlying deficient object use was a loss of knowledge of the manner in which objects are to be used, which they characterized as a semantic deficit. This emphasis on “manner” of manipulation raises questions about the role gesture engrams might play in object use knowledge. In contrast, other investigators view deficient recognition of the gestures associated with objects (that is, the manner of use) to be a symptom of the Representational type of IM, and not IA.
Recent evidence suggests that different types of object knowledge may bear different relationships to apraxia. For example, knowledge of object function and knowledge of manner of object manipulation are dissociable. One might have knowledge of the function of an object (e.g., a knife is for cutting things) without knowledge that knives are often used with a back-and-forth, saw-like gesture. Patients with IMA tend to have of the latter type, but not necessarily the former. Some reports indicate that patients with IMA may also be impaired in mechanical problem-solving or the ability to infer function from structure.

Evidence for a relationship between function knowledge and performance on tasks involving multiple objects is equivocal. One potential source of confusion is that the relationship between functional knowledge and object use is sometimes assessed with single objects and sometimes with tasks involving multiple objects. In several studies using single object tests in patients with semantic dementia, a disorder with a predilection for the temporal lobes, a strong relationship has been reported. In other investigations no relationship between single object use and functional knowledge has been found. There is stronger evidence that function knowledge is not well correlated with performance on tasks involving multiple objects. There are patients who make “conceptual” errors on these tasks, but who perform normally on semantic tests of functional and associative object knowledge, and others who perform nearly normally on tasks involving multiple objects or in real-life action, despite considerable semantic deficits.

**Functional Implications of Limb Apraxia**

Historically, most clinicians and researchers regarded IMA as a clinical oddity that had little significance in the real world. It appears that this view was derived from the notion that IMA was present when gestures to command and imitation were tested, but improved when actions with actual objects were examined. A number of recent studies, however, have suggested that IMA is associated with deficits in activities of daily living. At least in some studies, subjects with IMA
are more likely to be impaired in object use, particularly in complex tasks, than non-apraxic subjects who have suffered a stroke.

**Ideomotor Apraxia in View of Recent Developments in the Motor Control Literature**

**Imitation.**

With the discovery of mirror neurons in the macaque premotor and parietal cortex that respond both to observed and performed actions, action imitation has emerged as an area of considerable interest in the neuroscience community. One important question concerns the degree to which imitation failure in IMA reflects damage to the mirror neuron system. Indeed, the neuroanatomic loci of lesions leading to IMA overlaps considerably with the localization of mirror neurons. Additionally, as noted, imitation and recognition impairments show a strong correspondence in IMA. On the other hand, IMA due to left parietal lesions frequently disrupts object-related (transitive) imitation far more than non-object related, symbolic (intransitive) imitation. Additionally, there is evidence of body-part specificity in IMA imitation disruption that is not easily accommodated by putative damage to a mirror neuron system. Left hemisphere IMA patients tend to be significantly more impaired in imitation of hand postures than finger positions, and in general, IMA appears to affect arm more than leg imitation. These dissociations could be accommodated by positing that effector-specific populations of mirror neurons might reside in different cortical regions in each hemisphere, but to this point there is little evidence for this possibility. Future investigations addressing these issues are required.

**Object-Related Action.**

Recent evidence from single cell recordings in monkeys indicates that populations of neurons in the inferior premotor cortex (in an area with probable homologue of Areas 44 and 45 in humans) as well as in the anterior intraparietal sulcus (AIP) are active in response to objects that are graspable by the monkey observer. These have been termed “canonical” neurons.
Complementary studies using fMRI and TMS in humans are consistent in suggesting that similar regions in the human brain are responsive to the structural properties (i.e., shape and size) of graspable objects. These populations appear to encode hand movement parameters (e.g., finger thumb aperture) for object grasping. In this context, there is considerable recent evidence that IMA patients, while intact in their ability to position the hand in response to object structure, are disproportionately impaired in hand shaping for functional object manipulation. The relationship of this pattern of performance to the “canonical” neuron system is an additional area of interest for future investigation.

**Spatiomotor Frames of Reference for Action.**

At least two different frames or reference or coordinate systems have been proposed for action. Many investigators have proposed that action may be planned in workspace-specified extrinsic coordinates. On this account, movements are planned with respect to a target that is coded in external space. Reaching to grasp a target would entail the creation of a spatial vector describing a desired movement’s direction and amplitude. An alternative hypothesis proposes that movement control may occur in body-specified intrinsic coordinates; on this account, a movement plan would specify the positions of the shoulder, elbow and wrist that would be needed to get the hand to the target. Extensive evidence for both types of control has led to a third group of accounts proposing that control is an interactive process that uses both extrinsic and intrinsic coordinate frames, depending in part upon the demands of the task.

Recent evidence from IMA patients indicates that movements that may putatively rely strongly upon extrinsic control (i.e., object-directed movements) are accurate, whereas movements not having external referents (i.e., body-directed movements) are characterized by spatial errors in hand configuration, wrist angle, hand orientation, and hand location. The possibility that IMA may in part reflect deficient coding of action in a body-centered framework is an area of active investigation in several laboratories.
Feedforward and Feedback-driven Processes in IMA.

The process of motor control is commonly subdivided into planning and online correction components. Planning is the preparation of a movement before movement initiation, whereas online correction refers to the adjustment of the movement plan during movement execution. There is evidence that IMA may be attributable in part to deficits in planning actions with relatively intact online correction. IMA patients are impaired in motor imagery, thought by several investigators to be a proxy for motor planning stages of action. They are also abnormally disrupted when visual feedback of movement is unavailable. This suggests that such patients may rely abnormally on visual feedback in the performance of skilled action.

Treatment of Limb Apraxia

The current literature on apraxia treatment is sparse. Approximately 10 treatment efforts have been reported; in many cases, there is but a single study devoted to each treatment approach. The studies uniformly fall into the category of Phase I studies in which feasibility is assessed in small number of subjects. Thus, it is difficult at this stage to draw conclusions about treatments that may hold particular promise.

In general, the few reported treatment approaches can be grouped into three categories: 1) Studies that attempt to directly ameliorate deficient object-related gesture production with a variety of visual and tactile cues and feedback, 2) studies focusing on providing corrective feedback for errors in naturalistic multi-step action, 3) studies that attempt to prevent error from occurring (errorless learning approach). All of the studies report at least some treatment benefit, but several difficulties obscure the interpretation of results. For example, apraxia type is frequently poorly characterized. Although gesture recognition is clearly an important index of the integrity of gesture representations (which in turn, may have important implications for rehabilitation strategies), recognition testing is usually not performed. Only a few studies report
generalization to untreated stimuli (or behaviors), maintenance of treatment effects, or impact upon daily activities.

There is preliminary evidence based on these few studies that limb apraxia is amenable to treatment. The purpose of Phase I research, however, is to develop hypotheses, protocols, and methods, establish safety and activity, determine the best outcome measures, identify responders vs. non-responders, determine optimal intensity and duration, and determine why the treatment is producing an effect. This suggests that further systematic inquiry is required to satisfy the objectives of Phase I research.

**Testing for Limb Apraxia**

Apraxia cannot be assessed in subjects whose comprehension or cognitive deficits prevent them from understanding the task or whose visual deficits preclude identification of an object or gesture; before testing for apraxia, these disorders must, therefore, be excluded. In order to identify apraxic subjects and distinguish between the different types of apraxia described above, a testing battery should include at least the following components:

1. Assessment of manual dexterity (e.g., rotation of coin between fingertips).

2. Testing of gesture to command and to sight of object.

3. Imitation of meaningful and meaningless gestures.

4. Assessment of intrinsic egocentric spatial coding – that is, the ability to imitate meaningless static positions of the body such as holding the dorsum of the left hand against the right cheek

5. Assessment of extrinsic egocentric spatial coding by reaching to touch or grasp objects.

6. Tests of functional semantic knowledge (e.g., which two of three pictured objects - paper clip, rubber band, door lock - are used for the same purpose).

7. Tests of manipulation knowledge (e.g., which two of three pictured objects - saw, clothes iron, and watering can - is used with the same or similar gesture).
8. Perform a familiar multi-step task such as preparing a cup of instant coffee.

9. Recognize gestures by naming a gesture or selecting which of 2 gestures is correctly performed.

Conclusions

Apraxia is a complex and heterogeneous disorder that has important clinical and scientific implications. Recent investigations of the disorder that are motivated by emerging accounts of motor control and planning are beginning to explicate the processing impairments underlying the apraxic disorders. The accumulating knowledge offers promise not only for the development of treatments of apraxia but also for the understanding of the procedures by which actions are generated and their underlying neural basis.
Suggested Readings


Figure Legends:

Figure 1. Photographs of a patient with conceptual apraxia making a sandwich with meat and mustard. She correctly places meat on a slice of bread, closes the sandwich, and opens a mustard jar. She replaces the mustard jar, reaches into a package of marking pens, retrieves a yellow marker, and proceeds to color the meat yellow.

Figure 2. Maximal lesion overlap from 17 apraxic patients is shown in dorsolateral and inferior parietal regions (reproduced with permission from Haaland, Harrington, & Knight, 2000).

Figure 3. Typical errors in ideomotor apraxia. TOP: Three still photographs from a videotape showing a sequence of postures produced by an apraxic patient in imitating a sawing movement. Note the typical hand posture error comprised of repeated hand opening with arm extension, despite the fact that the model maintained a closed grip throughout. BOTTOM LEFT: Typical “body-part-as-object” error in a toothbrushing pantomime. BOTTOM CENTER: Typical arm posture error in imitating a scissoring movement. The model’s movement was produced perpendicular to the body wall (from near to far) whereas the patient’s movement proceeded left to right. BOTTOM RIGHT: Typical amplitude error in imitation of a hammering gesture. The model to be imitated demonstrated a large swing with peak amplitude at shoulder height, whereas the patient’s maximal amplitude was at elbow height.

Figure 4. A slightly modified version of the cognitive neuropsychological model of limb apraxia proposed by Gonzalez-Rothi, Ochipa, & Heilman (1997).