Limb Apraxia

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ABSTRACT

Limb apraxia is an impairment in the ability to perform skilled, purposive limb movements as the result of neurological dysfunction. In right-handed individuals, limb apraxia is associated with left hemisphere lesions. This article reviews the subtypes and neuroanatomic correlates of limb apraxia, including limb kinetic apraxia, ideomotor apraxia, ideational apraxia, and conceptual apraxia. The functional impact of limb apraxia on the individual is discussed. Strategies for the assessment and management of this disorder are reviewed.

Keywords: Apraxia, movement planning, praxis

Limb apraxia is an impairment in the ability to perform skilled, purposive limb movements as the result of neurological dysfunction. In defining limb apraxia, we exclude other neurological conditions that may cause impaired skilled motor performance such as weakness, deafferentation, akinesia, abnormalities of tone or posture, and movement disorders such as tremor or chorea.[1] In right-handed individuals, limb apraxia is associated with left hemisphere lesions.[2–4] However, rare cases of limb apraxia in right-handed individuals following right hemisphere lesions (crossed apraxia) have been described.[5–7]

The earliest systematic studies of limb apraxia were recorded by Hugo Liepmann in the early 20th century. Liepmann proposed that for right-handed individuals, the left hemisphere provided the guidance for performing skilled movements with both the right and left arms.[3,4] He suggested that the left hemisphere contained motor representations, or movement formulas, that spec-

Objectives
On completion of this article the reader will be able to recognize and distinguish limb kinetic apraxia, ideomotor apraxia, ideational apraxia, and conceptual apraxia.

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ified the spatial and temporal features of skilled, purposive movements. Liepmann described three forms of limb apraxia: limb kinetic, ideomotor, and ideational. Since Liepmann's time, advances in behavioral neurology, neuropsychology, and neuroimaging have added to our understanding of the nature and manifestations of limb apraxia. However, Liepmann's original terminology and much of his theory about the mechanisms of apraxia remain current today.

In this chapter we will (1) review our current understanding of the subtypes and neuroanatomic correlates of limb apraxia, (2) discuss the functional impact of limb apraxia on the individual, and (3) review strategies for assessment and management of this disorder.

LIMB KINETIC APRAXIA

In limb kinetic or melokinetic apraxia, patients are unable to make fine, precise finger movements of the hand contralateral to a cerebral lesion. The disorder is more apparent when testing distal movements than proximal movements and can be seen in pantomime, gesture imitation, and actual tool or object use. Some consider limb kinetic apraxia to be a movement disorder intermediate between paresis and apraxia.[8] Heilman and Rothi[9] consider limb kinetic apraxia to be more of an elemental motor disorder than a disorder of learned, skilled movement.

Although Liepmann[4] suggested the lesion responsible for limb kinetic apraxia was in the sensory motor cortex, the anatomic location of lesions producing this syndrome has not been firmly established in humans. However, pyramidal lesions in monkeys may lead to contralateral clumsiness that cannot be accounted for by weakness or changes in tone or posture,[10] supporting the notion that limb kinetic apraxia may be related to injury to the corticospinal tract.

IDEOMOTOR APRAXIA

Patients with ideomotor apraxia make several types of errors when performing skilled, purposive limb movements. The most common errors in ideomotor apraxia are spatial errors.[11] One type of spatial error involves the failure to position the hand in an appropriate posture (e.g., closed fist posture for drinking from a cup). A second type of spatial error involves the failure to orient the movement toward an imagined object (e.g., demonstrating the use of a toothbrush at the level of the chest). A third type of spatial error involves the failure to coordinate joint movement (e.g., demonstrating a screwdriver by rotating at the shoulder instead of at the elbow).[12] Another common apraxic error involves the patient using a body part as if it were the imagined tool (e.g., extending the finger to represent the blade of the screwdriver instead of positioning the hand around the handle of the screwdriver). Apraxic patients may also make sequencing errors (e.g., demonstrating key use by rotating the wrist, then extending the arm) and timing errors such as failure to coordinate speed with the spatial aspects of the gesture.[12]

The type of gesture may influence performance in apraxic individuals. Transitive gestures (demonstrating tool/object use) are typically more impaired than intransitive gestures (not involving a tool/object) such as "wave goodbye."

Finally, the nature of the task may influence gestural performance in apraxic individuals. Performance is usually worse when patients perform pantomime to command. Some individuals may improve with imitation and actual tool/object use, although performance often remains defective.[13] Descriptions of several variants of ideomotor apraxia follow.

CALLOSAL APRAXIA (UNILATERAL LIMB APRAXIA)

Liepmann and Maas[14] described a patient with right hemiplegia who was unable to carry out verbal commands with his left hand. The patient was found to have a lesion of the left basis pontis (accounting for his right hemiplegia) and a callosal lesion. Liepmann and Maas argued that their patient's deficit could not be accounted for by a language-motor disconnection because imitation of skilled movements and actual object use were also impaired. Rather, they concluded that the left hemisphere contained "movement formulas" that store knowledge of how to control skilled movements and that the callosal lesion disrupted the left hemisphere's ability to guide the ipsilateral left hand in the performance of these movements.

Watson and Heilman[15] and Graff-Radford and colleagues[16] described patients with callosal apraxia who demonstrated severe unilateral, left-hand apraxia along with impairments of gesture imitation and tool/object use. However, others have reported callosal apraxia in patients with spared imitation and tool/object use abilities,[17,18] leading some[19] to propose that the "movement formulas" are less lateralized in these cases. The most common natural lesion inducing callosal apraxia results from an infarction of the anterior cerebral artery with involvement of the genu and body of the corpus callosum and sparing of the splenium.[20]

APRAXIA IN POSTERIOR VERSUS ANTERIOR HEMISPHERIC LESIONS

In addition to proposing the dominant role of the left hemisphere in the mediation of skilled, purposive movements, Liepmann[3] described distinct components of the action system. These components included the movement formulas (space-time-form memories of learned, skilled movements) and a system of "innervatory patterns" that adapt these memories to environmental conditions. Liepmann suggested that destruction of the movement formulas was associated with lesions in the posterior portion of the left hemisphere.

Heilman, Rothi, and Valenstein[2] specifically proposed that the movement memories are stored in the left inferior parietal lobe. These authors suggested that the destruction or disconnection of these movement memo-

ries from other areas critical to praxis processing could explain the various disassociations seen in apraxic patients. According to their schema, pantomime to verbal command engages the posterior language areas, then information flows to the inferior parietal lobe where the movement memories are accessed. Information subsequently accesses the premotor and motor areas of the left hemisphere for the control of the right arm. Information crosses the corpus callosum to the motor areas of the right hemisphere for control of the left arm. Visual information may also gain access to the stored movement memories in the left inferior parietal lobe.

Heilman and colleagues[2] tested 20 patients with unilateral left hemisphere lesions using a gesture to verbal command task and a gesture discrimination task. For gesture discrimination, subjects were shown a film of a man performing a pantomime, and they had to choose which of three choices represented the target pantomime named by the examiner. Two groups were distinguished; those who were apraxic and could not discriminate gestures and another group with apraxia but spared gesture discrimination. The subjects with lesions of the left parietal lobe had gesture discrimination problems. That is, they could not recognize gestures because there was damage to the representations for learned, skilled movements (Fig. 1, lesion B). Subjects with anterior lesions not involving the left parietal lobe had production difficulties but not a gesture discrimination problem, suggesting that the movement memories were not destroyed but could no longer interact with anterior areas responsible for motor implementation (Fig. 1, lesion C).

In summary, posterior left hemisphere lesions are associated with deficits in both producing and understanding gestures, whereas anterior left hemisphere lesions result in gesture production difficulties in the context of spared gesture comprehension. It should be noted that some individuals have spared gesture comprehension despite damage to the posterior regions of the left hemisphere thought to store the representations for learned, skilled movement. Some have suggested that the representations may be more diffusely, or bilaterally, represented in these individuals.[21,22]

APRAXIA IN SUPPLEMENTARY MOTOR AREA LESIONS

As mentioned previously, Liepmann suggested that in addition to the movement formulas, there is a separate component of the action system, the innervatory pattern system, which is thought to transform the movement representation into a motor plan.[4] It has been posited that the movement formulas are coded in three-dimensional supramodal code.[23] These representations must be transcoded into an innervatory pattern that in turn is fed to the motor cortex. The supplementary motor area (SMA) has connections with the primary motor cortex and the parietal lobe.[24] It is activated prior to motor cortex activation,[25] and it becomes activated during the performance of skilled movements.[26] It therefore may be the site of the transcoding of the praxis representations into motor programs for execution by the motor cortex. Watson, Fleet, Rothi, and Heilman[27] described several patients with SMA lesions who had bilateral ideomotor apraxia but could comprehend and discriminate gestures. Thus, a gesture production deficit in the absence of a gesture reception deficit may occur when the praxis representations can no longer interact with the innervatory patterns (Fig. 1, lesion C), or the innervatory patterns cannot gain access to the motor area (Fig. 1, lesion D).

APRAXIA IN SUBCORTICAL LESIONS

Although rarely reported, ideomotor apraxia may result from lesions in the left basal ganglia and thalamus.[28–32] The apraxia is generally described as "mild" and may be more common in basal ganglia lesions than in thalamic lesions.[33] Error types, when described, appear to be generally similar to those seen in cortical apraxia.[31,32] The role of subcortical structures in praxis processing warrants further study.

PANTOMIME AGNOSIA

Agnosia is a recognition failure that cannot be attributed to an elemental sensory defect, a generalized cognitive defect, a language disorder such as anomia, or a lack of prior knowledge of the stimulus.[34] Rothi, Mack, and Heilman[35] described two patients who could not comprehend or discriminate visually presented gestures, yet could imitate and produce gestures normally. Both patients could recognize faces and objects, sug-

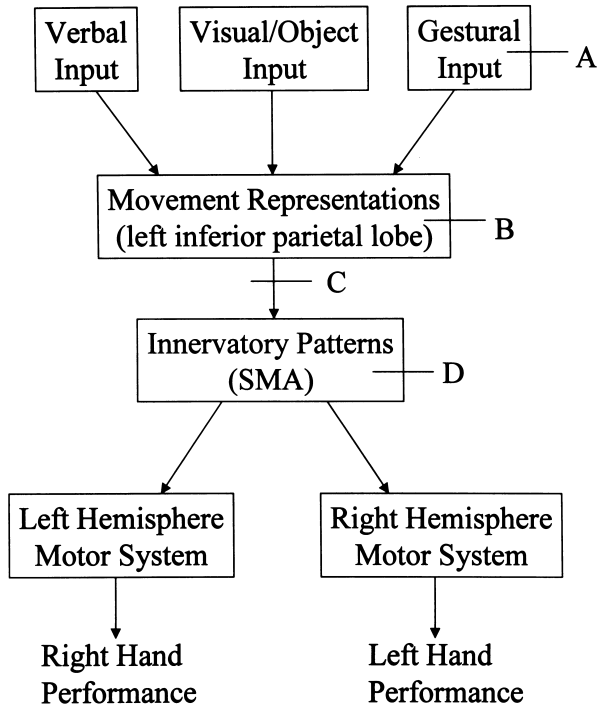

Figure 1. Model depicting possible impairments in ideomotor apraxia.

gesting that their agnosia was limited to pantomime. The lesions in these cases were located in the left, ventral temporo-occipital region. This lesion location presumably prevents visual information from accessing the stored representations for learned, skilled movements (Fig. 1, lesion A).

IDEATIONAL APRAXIA

The term ideational apraxia has been used to describe impairments involving actual tool/object use. Liepmann[3] described a patient who demonstrated inaccurate tool use such as using a razor as a comb and placing eyeglasses on his outstretched tongue. Liepmann noted that these errors were qualitatively different from those made by patients he described as ideomotor apraxics. Liepmann suggested that these errors reflected an impairment in the ideation of tool use and labeled the syndrome ideational apraxia. Liepmann also described this syndrome as the inability to perform a sequence of acts using tools and objects to achieve an intended goal.[4] Subsequent investigators have not agreed on what actually constitutes limb apraxia; a defect in actual tool use[36–38] or failure to perform serial acts involving tools and objects.[39] Because of this controversy, and because a severe production disorder (ideomotor apraxia) may also extend to actual tool use, we prefer the term conceptual apraxia to refer to patients who fail to use tools and objects correctly because of a conceptual disorder.

CONCEPTUAL APRAXIA

Roy and Square[40] suggested that praxis processing is mediated by a two-part system involving both conceptual and production components. According to these authors, the praxis conceptual system involves three kinds of knowledge: knowledge of the functions of tools and objects, knowledge of actions independent of tools, and knowledge about the serial organization of single actions into sequences. The praxis production system involves the sensorimotor component of action knowledge, including the information contained in action programs and their translation into action. Within this framework, ideomotor apraxia, with its resultant spatial and temporal errors, would result from a disruption of the praxis production system. Disruption of the praxis conceptual system would result in content errors (e.g., gesturing the use of a comb for a toothbrush).[38,41]

Ochipa, Rothi, and Heilman[38] described a patient who made content errors in gesture production and used actual tools inappropriately in natural contexts. For example, this patient was observed to eat his meal with a toothbrush and brush his teeth with a comb. This behavioral disturbance was not attributed to agnosia, as the patient had adequate naming and name comprehension for the items he misused. He demonstrated poor knowledge of tool function and tool-object association, suggesting impaired conceptual knowledge of tool use.

Ochipa, Rothi, and Heilman[41] suggested that four types of conceptual knowledge may relate to one's ability to correctly use tools/objects in the environment: (1) knowledge of actions associated with tools (e.g., pounding motions are associated with a hammer), (2) knowledge of tool-object relationships (e.g., hammers are used on nails), (3) knowledge about the mechanical advantages afforded by tools (e.g., if a hammer is not available, pliers would pound better than a spoon), and (4) knowledge about tool fabrication (e.g., a wire can be bent to retrieve an object through a narrow opening). Subjects with Alzheimer's disease were found to make all four types of conceptual errors.[41] The conceptual apraxia seen in these subjects was not associated with ideomotor apraxia or semantic language impairment, suggesting that the system that mediates conceptual praxis knowledge may be independent of the gesture production system or verbal semantics.

Patients with ideational and/or conceptual apraxia often have diffuse or bilateral, posterior cerebral involvement.[21] However, the syndrome has been described in focal damage to the posterior left hemisphere.[4,36] The patient described by Ochipa and colleagues[38] was left-handed with a large lesion in the distribution of the right middle cerebral artery.

APRAXIA IN DEGENERATIVE NEUROLOGIC DISEASE

Ideomotor apraxia to both verbal command and imitation has been reported in individuals with Alzheimer's disease.[42,43] Rapcsak and colleagues[43] reported that their Alzheimer's subjects were particularly impaired for transitive movements, whereas intransitive movements were relatively spared. These authors also reported ideational apraxia in this population as measured in a task involving the sequencing of object use to achieve an intended goal. Ochipa and colleagues[41] found conceptual apraxia in a group of Alzheimer's subjects, based on their performance on a variety of tasks designed to assess knowledge of tool use.

Slowly progressive, bilateral ideomotor apraxia has been described in focal degenerative processes involving the posterior regions of the brain.[44–46] Pathological examinations of brains with posterior cortical atrophy have produced variable results.[47]

Unilateral ideomotor apraxia was reported to be among the first cortical signs observed in patients with corticobasal degeneration.[48] The apraxia may progress to both limbs but often remains asymmetrical. Jacobs and colleagues[49] reported spared gesture discrimination ability in their apraxic patients with corticobasal degeneration, suggesting an impairment in praxis production-execution with sparing of the movement representations. Leiguarda and colleagues[50] described bilateral ideomotor apraxia for transitive movements in patients with Parkinson's disease and progressive supranuclear palsy. These authors suggested that apraxia may be related to the amount of cortical involvement in these patients with corticostriatal dysfunction.

FUNCTIONAL IMPACT OF LIMB APRAXIA

Reports of the incidence of limb apraxia among individuals with left hemisphere lesions range from 50%[51] to 80%,[52] with variability likely due to subject selection and testing differences across investigators. Nevertheless, apraxia appears to be a common consequence of left hemisphere brain damage. It also appears to be enduring. Kertesz, Ferro, and Shewan[53] reported that 40% of 118 patients still had apraxia three or more months after the onset of a left hemisphere stroke. Studies indicate some improvement in limb praxis performance occurs over time (e.g., improvements in gesture recognizability), although significant impairments in the spatial and temporal aspects of gesture production remain.[54]

Although limb apraxia is a common and enduring consequence of left hemisphere lesions, many consider the syndrome to have little practical significance. One reason may be that patients rarely complain of the disorder. Rothi, Mack, and Heilman[55] reported that apraxic patients are often unaware of their apraxic deficits. Their lack of awareness may be related to the fact that they often have a right hemiparesis and attribute their clumsiness to inexperience in using their left, nondominant hand. Patients may also be limited in their ability to express concern because of the common cooccurrence of aphasia.

Another reason clinicians consider apraxia to have little practical significance is that the disorder is most easily identified in the context of the pantomimed use of tools/objects, a task generally limited to the artificial test environment. Individuals with apraxia often improve their performance during actual tool use. However, studies have shown that apraxic subjects make the same types of errors in actual tool use as they do in pantomime to command.[13,56] Studies of the ability of patients with limb apraxia to use tools and objects in natural contexts have confirmed the negative impact of apraxia on a patient's everyday activities.[38,57,58] Sundet, Finset, and Reinvang[59] evaluated the dependence of patients with left or right hemisphere strokes on caregiver assistance in activities of daily living 6 months after the stroke onset. They reported that measures of apraxia were highly correlated with levels of dependence estimated by caregivers. Moreover, they reported that patients with limb apraxia required more assistance with activities of daily living than patients with other neuropsychological deficits. In a study of stroke patients 1 year after onset, apraxia was a highly significant predictor of low subjective well-being.[60]

Finally, the impact of apraxia on communicative gesture must be considered. Gesture is often used as an alternative or augmentative communication for aphasic adults.[61,62] Unfortunately, limb apraxia and aphasia often cooccur. The presence of limb apraxia negatively affects the quality of communicative gesture[63] and the ease of gesture acquisition,[64] thereby complicating rehabilitation efforts for communicatively impaired individuals.

In summary, limb apraxia is not only of theoretical interest. It is a common and enduring disorder with potential for significant negative impact on an individual's functional independence.

ASSESSMENT OF LIMB APRAXIA

This section will review some of the assessment techniques that have been used in our clinical practice and in our research with neurologically impaired adults. For a more comprehensive review of limb praxis assessment, see Rothi and colleagues[65,66] or De Renzi[67] for another view.

We have found that both our normal and our apraxic subjects often produce inadequate gestural responses, not appreciating the precision and elaboration expected when performing pantomimes. For this reason, we have found it helpful to spend time beforehand instructing the subjects to produce the gesture as though they are actually holding the imagined tool (e.g., hammer) and stressing the need to imagine the object (e.g., nail) they are acting upon. When apraxic individuals pantomime, they often gesture with a body part as if it were the tool (body part as tool errors). Some clinicians question the significance of body part as tool errors because individuals without brain damage produce these responses as well. If a patient makes a body part as tool response, we suggest reinstructing the patient (e.g., " Do not use your finger as the key. Make believe you are really holding a key."). Apraxic subjects are generally unable to improve their performance after reinstruction or cuing by the examiner.[68]

When possible, we test the dominant hand. Because hemiparesis often prevents this, the nondominant, ipsilesional hand is tested. We use a screening instrument, the Florida Apraxia Screening Test-Revised[69] (see Table 1). The items include 20 transitive and 10 intransitive gestures that can be performed with one hand. We use the same items to assess gesture production and gesture reception. Of course, task modifications may be necessary for patients with concomitant expressive or receptive language impairment. Finally, although the following tasks are useful in identifying apraxia at bedside or in the clinical setting, the importance of observation of object use in natural contexts should not be overlooked.

GESTURE PRODUCTION

Gesture production tasks may include gesture to verbal command (e.g., "Show me how to use a comb to fix your hair.") and gesture to visual presentation (e.g., the examiner shows the subject a tool and says, "Show me how you use this."). Testing different input modalities (verbal command versus visual presentation) allows one to determine whether deficits are related to sensory-specific input processing mechanisms.[35,70]

GESTURE IMITATION

Gesture imitation involves the subject viewing the examiner during the performance of a gesture and dupli-

Table 1. Stimuli Used in the Florida Apraxia Screening Test-Revised (FAST-R)*

Show me:

1. how you salute
2. how to use scissors to cut a piece of paper out in front of you
3. how to use a saw to cut a piece of wood out in front of you
4. how you hitchhike
5. how to use a bottle opener to remove a cap on a bottle out in front of you
6. how to use wire cutters to snip a wire out in front of you
7. stop
8. how to use a salt shaker to salt food on a table out in front of you
9. go away
10. how to use a glass to drink water
11. how to use a spoon to stir coffee on a table out in front of you
12. how to wave goodbye
13. how to use a hammer to pound a nail into a wall in front of you
14. how to use a comb to fix your hair
15. how to use a knife to carve a turkey on a table in front of you
16. how to use a brush to paint a wall out in front of you
17. come here
18. how to use a screwdriver to turn a screw into a wall out in front of you
19. how to use a pencil to write on a paper on a table out in front of you
20. someone is crazy
21. how to use a key to unlock a doorknob on a door out in front of you
22. be quiet
23. how to use an iron to press a shirt out in front of you
24. how to use a razor to shave your face
25. OK
26. how to use an eraser to clean a chalkboard out in front of you
27. how to use a vegetable peeler to shred a carrot
28. how to make a fist
29. how to use an ice pick to chop ice out in front of you
30. how to use a scoop to serve ice cream

*"Cutoff" score for "normal" performance is 15 out of 30 trials correct.[69]

From Rothi LJG, Raymer AM, Ochipa C, et al. Florida Apraxia Battery, experimental edition (unpublished), 1992.

cating that performance. Often, imitation of gestures is superior to gesture production to verbal command. However, it is possible for imitation performance to be worse.[71]

GESTURE RECEPTION

Reception tasks may include discrimination between correct and incorrect pantomimes (e.g., "Is this the correct way to use a hammer?"), gesture naming (e.g., "What tool am I using?"), and gesture comprehension (e.g., the examiner pantomimes, and the patient selects the correct tool from an array of choices). Impaired gesture reception in the absence of gesture production difficulty may indicate a problem in accessing the movement representations through the visual modality.[35] Combined production and reception deficits may indicate destruction of the movement representations, implicating damage to the left inferior parietal lobe. Spared gesture comprehension in the context of impaired gesture production may suggest an anterior left hemisphere lesion.

MANAGEMENT OF LIMB APRAXIA

Few studies have reported the direct treatment of apraxic deficits.[72–74] These studies have indicated that apraxia does respond to treatment (i.e., apraxic subjects can learn gestures). However, there is generally poor generalization of learned gestures to other communicative contexts and little generalization of gestural improvement to gestures not specifically targeted in treatment.[73–75] These findings should be interpreted not as implying that treatment should not be performed but that items targeted in treatment should be carefully chosen based on their functional salience for the individual patient.

When direct treatment of limb apraxia cannot be accomplished, management of the functional disability is possible. Management of limb apraxia would involve altering the environment to reduce the risk of injury and reduce the negative impact of apraxia on activities of daily living.[76] Strategies may include removing dangerous implements from the patient's access, limiting the available selection of tools for a particular task, replacing tasks that may require tools with those that can be performed without tools, and avoiding tasks that require the use of multiple tools. Patients may be referred to rehabilitation specialists for training in alternative strategies for task performance.

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