Apraxia

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The motor systems are capable of directing muscles to make an almost infinite number of movements. In order to perform purposeful skilled movements, the brain must acquire the knowledge to program motor systems appropriately. Apraxia is a cognitive motor disorder that entails the loss or impairment of the ability to program motor systems to perform purposeful movements. Because apraxia is a cognitive motor disorder, abnormal movements should not be called apraxic if they can be attributed to weakness, dyspraxia, tremor, chorea, athetosis, ballismus, seizures, myoclonus, ataxia, defects of sensory feedback, or if they result from non-motor cognitive disorders such as poor comprehension, agnosia, or inattention. Although apraxia is in part defined by exclusion, a variety of errors may characterize the apraxic patient’s performance and these errors help define the variety of apraxia seen in the clinic. This chapter will be limited to descriptions of apraxia that are associated with forelimb and buccofacial movements. The forms of limb apraxia we will discuss include limb-in kinetic, ideomotor, disassociation, conduct, ideational, and conceptual apraxias. We will also briefly discuss buccofacial apraxia. On the basis of these clinical descriptions, we will develop a model of how the brain mediates learned skilled movements. We will not discuss certain disorders that have been called apraxic, because they do not meet our definition of apraxia. Apraxia of eye opening and apraxia of gait are not cognitive motor disorders. Constructional apraxia and dressing apraxia are usually not movement program disorders, but are caused primarily by visuospatial disorders such as neglect.

EXAMINATION AND TESTING

GENERAL NEUROLOGICAL EXAMINATION

Since apraxia is in part defined by excluding the contribution of other disorders that may obscure apraxic errors, a thorough neurological examination is required. Diseases that affect either the basal ganglia or the cerebellum typically do not cause weakness or sensory change but can be associated with nonapractic disorders of movement. Disorders of the basal ganglia and cerebellum are also manifested by changes in posture and tone and by tremors, dysmetria, or stereotypic movements that are evident on neurological examination. If motor, sensory, basal ganglia, or cerebellar signs are limited to one side, the normal side can still be tested for apraxia. If the abnormality is mild
enough to permit use of the affected extremity, it should also be tested. In this case, the examiner should make allowance for the underlying disorder in judging whether apraxia is present.

Many apraxic patients are also aphasics, and aphasic disorders may be confused with apraxic disorders. For example, patients who do not understand commands may be thought to be apraxic when they fail to make an appropriate movement in response to a command. Conversely, apraxic patients with aphasia are occasionally mistakenly thought to have a comprehension disorder when their failure to accurately follow a command results from the inability to produce the correct movement. It is therefore important to test comprehension in other ways, such as with yes/no questions, pointing to objects on command, or by having the patient describe the movement they were asked to perform. Patients who fail to make correct movements but who demonstrate that they have comprehended the command may be apraxic. Patients who fail to demonstrate comprehension, however, may still be apraxic, since aphasia and apraxia frequently coexist.

Apraxic patients may use body parts as objects or make spatial and temporal errors, but often these movements can be recognized as having the correct intent, providing evidence that the command has been understood. Such movement errors should therefore not be attributed to aphasia, even if the patient has a mild deficit in language comprehension.

**TESTING FOR APRAXIA**

Patients rarely complain of apraxia and often appear unaware of their defect. This unawareness may be a form of anosognosia (Roith et al., 1990). Also, apraxic deficits, when recognized, are often explained away. For example, patients with right hemiparesis often think their left hand is clumsy because they are not accustomed to using it. When patients with Alzheimer's disease develop apraxia, it is often attributed to memory loss or to general intellectual decline. Furthermore, apraxia is usually mildest when a patient uses actual tools or objects and is most severe with pantomime. Since patients at home are rarely called upon to use pantomime, they and their families are often not aware of this disorder. Therefore, in diagnosing apraxia, one cannot rely on history but must test patients.

The following are some suggested procedures for apraxia testing. Classification of apraxia is based on relative performance across these tasks and on the type of errors that the patients make. The different forms of apraxia will be discussed in subsequent sections of this chapter.

The examiner should test both hands when possible. If one hand is severely parietic, the nonparietic hand should be tested. In addition to observing a patient's performance, the clinician should ascertain if a patient is disturbed by his or her errors or if the patient can even recognize that he or she has made errors. When testing apraxic patients, there is always the concern that the failure to perform correctly is related to a language comprehension error rather than to an apraxic error. Gesturing to command is most likely to be confounded by impaired language comprehension. However, while apraxic patients make errors, the intent of the gesture is often obvious. Comprehension of movement commands may also be tested by asking patients to describe what they were asked to do or by having patients point to the object (from an array of objects) that they would use to perform a specific action.

**Gesture to command.** This procedure should involve pantomiming both tool use (transitive movement), such as “Show me how you would use a pair of scissors,” and emblems (intransitive movement), which are arbitrarily coded nonverbal communications, such as waving good-bye. Some of the gestures we use are listed in Table 11–1.

Frequently, when patients pantomime, they use a body part as the tool. They may perform in this manner either because they do not understand that they are supposed to pantomime (i.e., they are using the body part as a symbol of the tool) or because they cannot perform the task even though they understand it. If a patient uses a body part as the object, this performance should be corrected: “Do not use your finger as a key. Make believe you are actually holding a key.” If verbal instructions do not help, the examiner should demonstrate the correct pantomime. If the patient still uses body part as tool, the patient is making an apraxic error.

**Gesture to imitation.** Some apraxic patients who cannot gesture to command can imitate gesture. Although some examiners only test imitation if the patient fails to perform the command correctly, there are patients who cannot imitate gestures, but who will perform correctly to command. Therefore, we advocate that imitation be tested in all patients. We also suggest that transitive as well as intransitive movements be tested and that meaningful and meaningless pantomimes also be tested.

**Gesture (pantomime) in response to seeing a tool.** This test may be especially useful when a patient has a language comprehension deficit and it is unclear if the failure to correctly gesture to command results from a language or apraxic disorder.

**Gesture (pantomime) in response to seeing object upon which a tool works.** This test may also be helpful when there is a comprehension disorder. Because seeing a tool may provide cues, this test may be more sensitive. This test is performed by showing the patient an object that receives the action of a tool (e.g., a nail partially driven into block of wood) and having the patient pantomime, using the tool.

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**Actual tool use.** This task is performed by handing the tool to the patient (e.g., hammer) and may be performed with or without the object (e.g., nail) upon which the tool works.

**Imitation of examiner using the tool.** In this task, the patient is asked to imitate the examiner's pantomime of the use of tools or implements.

**Discrimination between correct and incorrect movements pantomimed by the examiner.** To determine if a patient can discriminate between correctly and incorrectly performed pantomime, the examiner performs pantomimes that randomly are either correct or incorrect and asks the patient if the gesture is well performed.

**Pantomime and gesture comprehension.** While the examiner makes a specific gesture, he or she can ask the patient to identify the action, e.g., “What tool am I using?”

**Serial acts.** To learn if a patient can perform a series of acts, in order, that leads to a goal, the examiner asks the patient to perform a multi-step task (e.g., “How would you make a sandwich?”) (see Table 11–1).

**Action-tool associations.** In this test the examiner displays an array of tools (e.g., hammer, screwdriver, knife, pliers) and then pantomimes a target action (e.g., cutting) and asks the pa-
tient to point to the tool in the array that is associated with this action.

Tool-object associations. In this test the examiner displays an object (e.g., partially driven nail) and asks the subject to select from an array of tools the tool used to accomplish this action.

Conceptual knowledge. If the subject performs well on the tool-object association test, the tool is taken away and the patient is asked to select an alternative tool that could accomplish the same goal. For example, if the object is to remove a nail from a piece of wood, and the patient selects a hammer, the hammer is removed, and the patient can select another appropriate tool, such as a pair of pliers.

VARIETIES OF LIMB APRAXIA

Because of the diverse nature of the apraxic disorders, each will be discussed separately. Limb-kinetic, ideomotor, and ideational apraxia are terms used by Liepmann (1920). We also describe three forms of apraxia not discussed by Liepmann: disassociation, conduction, and conceptual apraxia.

LIMB-KINETIC APRAXIA

Clinical

Patients with limb-kinetic apraxia are incapable of making fine, precise movements with the limb contralateral to a central nervous system lesion. The disorder is more obvious when testing distal independent movements (finger movements) than when testing proximal movements, and is especially evident when the patient makes rapid finger movements such as tapping. The movement abnormality can be seen when the patient pantomimes, imitates, or uses objects. In the clinic, we ask patients to pick up a dime from a flat surface. Patients with limb-kinetic apraxia may not be able to perform the necessary pinching movement with their thumb and index finger and instead will slip the dime off the table and grasp the coin between their fingers and their palm. We also ask patients to rotate a quarter between their thumb, index, and middle finger as rapidly as possible. We note if there are any between-hand asymmetries in this task.

Pathophysiology

The neuroanatomic correlates of limb-kinetic apraxia are unclear. Limb-kinetic apraxia is often unilateral (contralateral to the lesioned hemisphere). However, we recently demonstrated that in right-handers limb-kinetic apraxia of the ipsilateral (left) hand is more likely to result from left hemisphere than right hemisphere dysfunction (Heilman et al., 2000). Liepmann (1920) postulated that lesions in the sensory motor cortex may induce this disorder. It has been demonstrated that pyramidal lesions in monkeys can cause clumsiness and a loss of movement fractionation that is not completely accounted for by weakness or by change in tone or posture (Lawrence and Kuypers, 1968). This suggests that the clumsiness seen in patients with limb-kinetic apraxia may be induced by pyramidal or corticofugal tract lesions. The role of the premotor regions in the pathogenesis of limb-kinetic apraxia is also unclear. However, according to Freund and Hummelshim (1985), manual dexterity and the capacity for relatively independent distal movements are unaffected in patients with lesions of the ventromedial premotor cortex. Unfortunately, many patients with lesions of premotor and motor cortex have tone and posture changes that make testing for limb-kinetic apraxia difficult.

IDEOMOTOR APRAXIA

Clinical

Unlike patients with limb-kinetic apraxia, patients with ideomotor apraxia may have normal dexterity (dexterity), especially when their ipsilesional arm is tested. Piccirillo and Vignolo (1967) tested the manual dexterity of 35 patients with lesions of the right hemisphere and 70 patients with lesions of the left hemisphere. The severity of the ideomotor apraxia was independent of manual dexterity. In addition, patients with ideomotor apraxia have greatest difficulty when asked to pretend they are making transitive movements (using a tool or instrument) (Goodglass and Kaplan, 1963). Although they may improve their performance by imitating, gestural imitation is frequently still defective. Similarly, improvement may be noted when the actual object is used, but performance often remains defective (Poizner et al., 1989).

Patients with ideomotor apraxia make several types of errors. Perseverative errors entail execution of previously performed pantomimes when new pantomimes are requested. Sequencing errors involve reversal of the order of movements in a sequence of movements. For example, in pantomiming the use of a key, one needs to extend the arm at the elbow and then rotate the forearm; the patient who makes sequencing errors may reverse this order (rotate first and then extend). Patients with ideomotor apraxia may use body part as object despite reminders to act as if they were actually holding and using the object (Rayner et al., 1997). Spatial errors are most characteristic of ideomotor apraxia. There are three forms of spatial errors: errors of posture, spatial orientation, and spatial movement (Rothi et al., 1988b, Poizner et al., 1990). Purtual errors entail the failure to position the hand to hold the imagined utensil or tool correctly. Body part as object errors are a special kind of postural error. Postural errors are seen primarily with pantomime and imitation and are not usually seen with actual object use, since the actual object constrains hand position. Spatial orientation errors denote hand movements that do not direct the imagined tool toward an imagined object. For example, when asked to pantomime the use of scissors, apraxic patients may move the scissors laterally instead of forward, or when asked to cut a slice of bread with a knife, they may fail to keep the imaginary knife in a constant sagittal plane. Haaland et al. (1999) demonstrated that aiming movement became worse when patients closed their eyes. Spatial movement errors denote movements at incorrect joints. For example, when asked to pantomime the use of a screwdriver, the normal subject will fix the wrist and shoulder and twist the forearm, so that the imaginary screwdriver rotates on its axis. The apraxic patient may fix the wrist and forearm and rotate the shoulder, so that the screwdriver moves incorrectly in an arc. Movement errors can also occur when there is loss of coordination of movements at different joints. For example, normally when cutting a loaf of bread with a knife, the shoulder is flexed in order to bring the arm forward as the elbow is extended, and the shoulder is extended (to bring the arm back) as the elbow is flexed. With each successive cutting movement, the elbow is flexed less so that the knife moves downward. Patients with ideomotor apraxia primarily use one movement, usually the more proximal movement (shoulder), or may fail to coordinate the two movements (Poizner et al., 1990).

Patients with apraxia also make timing errors (Poizner et al., 1990). There may be a delay in the initiation of movement or occasional pauses, especially when the spatial trajectory must be changed. Patients may also fail to coordinate the speed of movement with its spatial components. For example, when cutting bread with an imaginary knife, one normally shows the movement when one is about to reverse the direction of the cut; once the direction has been changed, the speed of movement increases. Patients with ideomotor apraxia do not demonstrate this pattern of movement.

Pathophysiology

Callosal Lesions—Lateralized Movement Formula. Liepmann and Maas (1907) studied a patient with right hemiplegia who performed poorly when attempting to carry out verbal commands with his left hand. On postmortem examination he was found to have a lesion in the left basis pontis, which accounted for his right hemiplegia, and a lesion of the anterior four-fifths of the corpus callosum, which Liepmann and Maas thought accounted for his left-hand apraxia. Although it could be postulated that the callosal lesion resulted in apraxia because it disconnected the language areas in the left hemisphere (Wernicke, 1874) from areas of the right hemisphere that control fine move-
imitate and he could use actual tools. He was agraphestic with the left hand and could not type or use anganam letters with the left hand but performed flawlessly with the right hand. He followed commands with his right hand but not with his left. A language–motor disconnection could explain both the left-hand aphasias aphasia and the left-hand apraxia. Similarly, surgical lesions of the corpus callosum (Gazzaniga et al., 1967) were not associated with the type of apraxia proposed by Liepmann and Maas (1907). Subsequently, however, Watson and Heilman (1983) and Graff-Radford et al. (1987) reported patients with acute naturally occurring callosal lesions who, unlike the patient of Geschwind and Kaplan (1962), had severe apraxia with imitation and object usage, thereby providing support for Liepmann’s callosal motor disconnection hypothesis.

Why are some patients with callosal lesions apraxic with imitation and object use, while others are not? Patients with surgical callosal lesions have had prior seizures and brain injury that may have induced brain reorganization, however, Geschwind and Kaplan’s patient did not have long-standing injury, suggesting that the absence of left-hand apraxia cannot be entirely explained by brain reorganization. Extracallosal damage cannot explain the difference, since Geschwind and Kaplan’s patient had considerable extracallosal damage, but he could imitate and use tools, whereas the computer tomographic (CT) scan of Watson and Heilman’s patient (1983) did not show any extracallosal damage. Variability in brain organization may explain the difference. In right-handers, right-hemisphere lesions almost never produce apraxia; however, left hemisphere lesions in areas known to induce both apraxia and apraxia more often induce apraxia. In one study, only 57% (20 of 35) of aphasic patients were also apraxic (Heilman, 1975), suggesting that movement or space-time movement representations are bilaterally represented in a considerable minority of right-handers (Heilman, 1979). It should therefore not be surprising if bilateral damage does not induce apraxia in all patients. The patients of Watson and Heilman and Graff-Radford et al. probably had language and movement representations restricted to the left hemisphere, which might represent the more common pattern. The patients reported by Gazzaniga et al. (1967) and Geschwind and Kaplan (1962) were left hemisphere dominant for language but probably had bilateral movement representations. Geschwind (1965) remarked that the independence of the right hemisphere in nonlanguage skilled motor function manifested by his patient may have been excepted.

The nature of the apraxic deficit seen with callosal lesions depends on the pattern of language and motor dominance in the individual patient. For example, we have seen two left-hand patients who were apraxic but not aphasic following right hemisphere lesions (Heilman et al., 1973; Valenstein and Heilman, 1979). Movement representations in these two patients were stored in the right hemisphere while language was mediated by the left hemisphere. We can speculate that if, prior to their right hemisphere lesion, these patients had a lesion of their corpus callosum, the right hand, deprived of the movement representations, would perform poorly to command, to imitation, and with the use of the actual object. The left hand, deprived of language, should perform poorly to gestural command but perform well with imitation and with an actual object. This pattern of deficits has not yet been reported.

"Left Hemisphere Lesions (Intrahemispheric) Defective symbolization. In right-handed patients, almost all cases of apraxia are associated with left hemisphere lesions (Goodglass and Kaplan, 1963; Hécaen and Ajuriaguerra, 1964; Geschwind, 1965; Hécaen and Saugier, 1971). In right-handers, the left hemisphere is also dominant for language. Apraxia therefore is not commonly associated with aphasia. This has led to the suggestion that apraxia and aphasia may both be manifestations of a primary defect in symbolization: aphasia is a disturbance of verbal symbolization, while apraxia is a defect of nonverbal symbolization (e.g., emblem and pantomime) (Goldstein, 1948). The observation that patients with aphasia perform poorly to command and imitation but improve with the use of the actual object (Goodglass and Kaplan, 1963) lends support to Goldstein’s postulate. In addition, Dee et al. (1970) and Kertesz and Hooper (1982) found a close relationship between language impairment and apraxia.

However, several studies lend support to Liepmann’s hypothesis that the left hemisphere controls skilled movements and that destruction of the movement representations or separation of these representations from the motor areas controlling the extremity causes abnormalities of skilled movement. Goodglass and Kaplan (1965) tested apraxic and non-aphasic apraxic subjects with the Wechsler Adult Intelligence Scale and used the performance-scaled score as a measure of intellectual ability. They also tested their subjects’ ability to gesture and perform simple and complex pantomimes. Although apraxic aphasics performed less well on these motor skills than did their intellectual counterparts in the control groups, no clear relationship emerged between the severity of aphasia and the degree of gestural deficiencies. Apraxic aphasic patients were also less able to imitate than were nonaphasic apraxic controls. Although Goodglass and Kaplan believed that their results supported Liepmann’s hypothesis, they noted that their apraxic subjects did not have any difficulty in handling tools. Liepmann, however, thought apraxic patients were clumsy with tools, and Foiner et al. (1969) observed and quantified motor and spatial errors of apraxic patients’ use of actual objects. Kimura and Archibald (1974) studied the ability of left-hemisphere-impaired aphasics and right-hemisphere-impaired controls to imitate unfamiliar meaningless motor sequences. The performance of aphasic patients with left hemisphere impairment was poorer than that of the controls, again supporting Liepmann’s hypothesis. The strongest support for the postulate that apraxia is a disorder of skilled movement rather than a symbolic defect comes from Liepmann’s own observations that only 14 of 20 apraxic patients were aphasic. Goodglass and Kaplan (1963) and Heilman et al. (1973, 1974) have also described similar patients. In addition, apraxic patients are often not apraxic (Heilman, 1975). In summary, because there is a poor correlation between the severity of symbolic disorders (aphasia) and disorders of skilled movements
and because even nonsymbolic movements are poorly performed by apraxic patients, there is little evidence to support the hypothesis that apraxia is a disorder of symbolic behavior.

Disconnection hypothesis of apraxia. Geschwind (1965) proposed that language elicits motor behavior by using a neural substrate similar to that proposed by Wernicke (1874) to explain language processing (see Fig. 11–1). Auditory stimuli travel along auditory pathways and reach Heschl’s gyrus (primary auditory cortex). From Heschl’s gyrus, the auditory message is relayed to the posterior superior portion of the temporal lobe (auditory association cortex). In the left hemisphere, this is called “Wernicke’s area,” and is important in language comprehension. Wernicke’s area is connected to premotor areas (motor association cortex) by the arcuate fasciculus, and the motor association area on the left is connected to the left primary motor area. When someone is told to carry out a command with the right hand, this pathway is used. To carry out a verbal command with the left hand, information must be carried to the right premotor cortex. Since it is rare to find fibers that run obliquely in the corpus callosum, fibers either cross from Wernicke’s area to the contralateral auditory association cortex or cross from the premotor cortex on the left to the premotor cortex on the right. The information is then conveyed to motor areas on the right side. Geschwind (1965) postulated that the connections between the premotor cortical areas are the active pathways. Support for the hypothesis that the active pathway for praxis crosses the callosum anteriorly comes from the observation that a selective anterior callosal section induced an ideomotor apraxia of the left hand (personal observation by K.M.H.).

Geschwind believed that disruption of cortical-cortical pathways explained most apraxic disturbances. As we have already discussed, according to Geschwind, callosal lesions produce unilateral ideomotor apraxia by disconnecting the left premotor region from the right. Lesions that destroy the left convexity premotor cortex also cause ideomotor apraxia, because the cell bodies of neurons that cross the corpus callosum are destroyed. Therefore, a lesion in the left convexity premotor cortex would cause a defect similar to that induced by a lesion in the body of the corpus callosum (sym pathetic dyspraxia). Lesions of the left convexity premotor cortex are often associated with right hemiplegia, so the right limb frequently cannot be tested. If these patients were not hemiparetic, however, they would probably be apraxic on the right.

According to Geschwind’s schema (1965), lesions of the arcuate fasciculus should disconnect the posterior language areas, important for language comprehension, from the convexity premotor cortex, important for implementing programs. Therefore, patients with parietal (or arcuate fasciculus) lesions that spare convexity premotor cortex should be able to comprehend commands but not perform skilled movements in response to command. More posterior lesions, affecting Heschl’s gyrus, Wernicke’s area, or the connections between them, cause abnormalities in language comprehension, but not apraxia. These patients fail to carry out commands because they cannot understand the command, not because they have difficulty performing skilled movements.

One problem in Geschwind’s interpretation is that patients with arcuate fasciculus lesions should theoretically be able to correctly imitate using their left hand, but often they cannot. Geschwind attempted to explain this discrepancy by noting that the arcuate fasciculus also contains fibers passing from visual association cortex to premotor cortex. He proposed that the arcuate fasciculus of the left hemisphere is dominant for visuomotor connections, but there is no evidence to support this hypothesis. Even if one assumes that the left arcuate fasciculus is dominant for visuomotor connections and interruption of this dominant pathway explains why patients cannot imitate, one could not explain why these patients are clumsy when they use actual tools. One would have to assume that the arcuate fasciculus also carries somesthetic-motor impulses and that the left arcuate fasciculus is also dominant for this function.

Representational hypothesis. After one learns a skilled motor behavior, future behaviors that require that same skill are expedited. In addition, even in the absence of specific instruction or cues, one can pantomime learned skilled behaviors. These observations suggest that the nervous system stores knowledge of motor skills. When this knowledge must be called into use, it is retrieved from storage and implemented rather than constructed de novo. A hypothesis that may explain why patients with parietal lesions cannot properly pantomime, imitate the use of, or use an object postulates that movement formulas or learned time-space movement representations are stored in the dominant parietal cortex (Heilman, 1979; Kimura, 1979). These representations help program the premotor cortex, which in turn implements the required movements by selectively activating the motor cortex. This innervates the specific muscle motor neuron pools needed to carry out the skilled act (Fig. 11–2). We call these movement formulas or time-space motor representations “praxicons.” Theoretically it would be possible to distinguish between dysfunction caused by destruction of parietal areas where praxicons are stored and apraxia resulting from disconnection of this parietal area from motor areas that implement these representations. Although patients with either disorder should experience difficulty in performing a skilled act in response to command, imitation of, or use of an object, patients whose representations for skilled acts are retained but whose premotor areas are disconnected (or whose premotor cortex is destroyed) should be able to differentiate a correctly performed skilled act from an incorrectly performed one because they still have these praxic representations (praxicons) and, therefore, have the information characterizing distinctive features of learned skilled movements. Patients with parietal lesions that have destroyed these representations (praxicons) should not be able to perform this analysis.

To test the postulate that praxicons are stored in the dominant parietal lobe and that destruction of these representations induces a discrimination deficit, we (Heilman et al., 1982; Rotell et al., 1985) gave a gestural recognition and discrimination task to apraxic and nonapraxic patients with anterior lesions or nonfluent aphasia and to patients with poste-
CLINICAL NEUROPSYCHOLOGY

Figure 11-2. Author's schema. View from top of brain. AG, angular gyrus; CC, corpus callosum; LH, left hemisphere; M, motor cortex; PM, premotor area (motor association cortex); RH, right hemisphere; SMG, supramarginal gyrus; VA, primary visual area; VAA, visual association area; W, Wernicke's area. The arrows show major connections of the areas shown.

APRAXIA

a defect in motor learning. The defect appeared to be caused by a combined impairment of acquisition and retention. Wyke (1971), who studied patients with either right or left hemisphere lesions, gave her subjects a motor acquisition test that required binasal coordination. Although patients with left hemisphere disease demonstrated acquisition, it was below the level of skill demonstrated by patients with right hemisphere disease. Since Wyke did not separate her left hemisphere group into apraxic and nonapraxic patients, one could not be certain whether apraxic patients would have demonstrated poorer learning than nonapraxic left hemisphere–damaged patients. Rothi and Heilman (1985) used a modified Bucsko (1975) paradigm to study apraxic subjects' ability to learn a list of gestures. We noted significantly more consolidation errors in the apraxic than control groups, a finding that suggests apraxic patients have a memory consolidation defect. Pistorini and coworkers (1991) also studied gesture and skill learning in patients with ideomotor apraxia and found that these patients were impaired. Lastly, if patients with apraxia have a loss of movement representations (praxicons), they should also demonstrate imagery deficits. Ochspi et al. (1997) demonstrated that ideomotor apraxia may be associated with movement imagery deficits.

Innervatory patterns. Praxicons stored in the inferior parietal lobe are in a three-dimensional supramodal code that has to be translated into a motor plan before the target movement can take place. Although Geschwind (1985) thought that lesions in convexity premotor cortex might induce apraxia, the role of the convexity premotor cortex in praxis is unclear. Many complex movements require the simultaneous movement of multiple joints. Barret et al. (1998) suggested that convexity premotor cortex may be important in binding these movement programs. The convexity premotor cortex may also be important in adapting the motor program to environmental perturbations.

The premotor cortex in the medial frontal lobe is called the supplementary motor area (SMA). Whereas stimulation of the primary motor cortex (Brodmann's area 4) induces simple single movements, SMA stimulation induces complex movements of the fingers, arms, and hands (Penfield and Welch, 1951). The SMA receives projections from parietal neurons and projects to convexity premotor cortex and to primary motor neurons. The SMA neuronal discharge before neurons in the primary motor cortex (Brinkman and Porter, 1978). Studies of cerebral blood flow, an indicator of cerebral metabolism, reveal that a simple repetitive movement increases activation of the contralateral motor cortex. However, complex movements increase flow in contralateral motor cortex and bilaterally in the SMA. When subjects think about making complex movements but do not move, blood flow is increased to the SMA but not to primary motor cortex (Orgogozo and Larsen, 1979).

Watson et al. (1986) reported several patients with left-sided medial frontal lesions that included the SMA who had bilateral ideomotor apraxia. However, unlike patients with parietal lesions, these patients could both comprehend and discriminate pantomimes. Because the SMA has connections with the primary motor cortex and the parietal lobe, it is activated before motor cortex, becomes activated with complex learned movements, and when ablated induces apraxia, we believe the SMA is the site where praxicon representations are translated into motor programs or innervatory patterns that activate motor cortex.

Basal ganglia. Whereas lesions and dysfunction of the basal ganglia are thought to induce alterations of muscle tone, abnormal movement, decreased spontaneous movements, and slowing of movements, the role of basal ganglia dysfunction in apraxia remains unclear. There have been several reports of patients who demonstrated ideomotor apraxia from lesions that involved the basal ganglia and/or thalamus (Basso et al., 1990; Agostini et al., 1983). Rothi et al. (1988a) described two patients with left-sided lenticular infarctions that did not involve cerebral cortex or associative pathways. Both patients had spatial movement errors that were similar to errors seen in patients with cortical lesions. However, both patients also showed frequent perseverative errors.
Alexander et al. (1986) described five discrete cortical-striatal–palidode-thalamic-cortical circuits. We have already provided evidence that lesions of left SMA are associated with apraxia. The SMA is a part of the motor circuit that projects to the putamen. The putamen projects to the globus pallidus and the globus pallidus projects to the ventrolateral nucleus of the thalamus. Finally, the ventrolateral nucleus projects back to the SMA. This discrete “motor loop” may control the flow of information into the SMA. Therefore, lesions of the loop may cause SMA dysfunction and SMA dysfunction may lead to apraxia.

Support for the observation that the basal ganglia are important in apraxia also comes from reports of patients with degenerative basal ganglia diseases. For example, Shelton and Kerpelman (1991) reported that apraxia was associated with Huntington’s disease and Leiguarda et al. (1997) reported that 27% of patients with Parkinson’s disease have ideomotor apraxia. Ideomotor apraxia has also been reported in the Parkinson-plus syndromes of progressive supranuclear palsy (PSP) (Leiguarda et al., 1997) and corticobasal degeneration (Jacob et al., 1999; Merians et al., 1999). In addition, patients with basal ganglia disease such as Parkinson’s disease may be impaired at learning new skills. However, even nondemented patients with basal ganglia disease may have cortical dysfunction. Pramstaller and Marsden (1996) performed a detailed review and meta-analysis of the relationships between basal ganglia injury and apraxia and concluded that diseases confined to the basal ganglia “rarely, if ever, cause apraxia.”

**CONDUCTION APRAXIA**

*Clinical*

Although most patients with ideomotor apraxia imitate transitive gestures better than they can pantomime transitive gestures to command, Ochita et al. (1990) described a patient whose imitation of learned transitive and symbolic movements was worse than his pantomime of these same movements to command. This patient had no difficulty comprehending the examiner’s pantomimes and gestures.

Pathophysiology

Unfortunately, the praxis model we have developed thus far (Fig. 11–3) cannot account for these findings. Such findings suggest that there are two independent sets of movement representations or praxicons—one for processing gestural input (input praxoncon) and one for processing movement output (output praxoncon) (Rothi et al., 1991) (see Fig. 11–4). A preserved ability to comprehend gesture in conjunction with impaired imitation would suggest that the input praxoncon remains intact and that the impairment occurs after the input praxoncon. The observation that the patient was better able to pantomime to command than imitate would suggest that verbal language is capable of activating the output praxoncon (by bypassing processing by the input praxoncon) (see Fig. 11–4) and that this patient’s deficit was a disassociation between the input and output praxoncon. Unfortunately, the localization of lesions that cause conduction apraxia is unknown.

**DISCONNECTION AND DISASSOCIATION APRAXIAS**

**Verbal-Motor Disassociation Apraxia**

Hospital. Heilman (1973) described three patients who, when asked to gesture, hesitated to make any movements and often appeared as if they did not understand the command. They could, however, demonstrate both verbally and by picking out the correct act from several performed by the examiner that they understood the command. Unlike patients with ideomotor apraxia, these patients were able to imitate and use actual tools flawlessly. Because imitation and actual object use were performed well, it would seem that their representations of motor skills (praxicons) were intact. What seemed to be defective in these patients was the ability to elicit the correct motor sequences in response to language.

**Pathophysiology** Although we hypothesized that the lesions were in, or deep to, the parietal region (angular gyrus), we never learned the exact locations of the left hemisphere lesions that induce this apraxia. The patients with callosal lesions described by Cestowid and Kaplan (1962) and Gazaniga et al. (1967) could not perform with their left hand in response to command, but they could imitate and use tools. Performance with the left hand of these patients with callosal disconnection was similar to the performance with both hands of patients with the left hemisphere lesions described above (Heilman, 1973). If normal performance on imitation and use-of-object tasks suggests that movement representations (praxicons) are intact and connected to premotor and primary motor areas, then patients with callosal lesions and patients with angular gyrus or subcortical lesions deep in the parietal lobe must have a disassociation between language areas and the area where motor representations (praxicons) are stored. In patients with callosal lesions, these movement representations are presumed to be in both hemispheres, whereas comprehension of command is mediated by the left hemisphere. In patients with left hemisphere le-
sions, both speech comprehension and the learned motor skills are being mediated by the left hemisphere, and the lesions disassociate language areas from praxias such that language is not able to activate the appropriate praxis (see Fig. 11–4). An alternative hypothesis is that in patients with left hemisphere lesions, the right hemisphere is mediating language comprehension, the left hemisphere contains the praxic representations, and the lesions disconnect the language areas from these representations.

Neuromotor and Tactile–Motor Dissociation Apraxia

Clinical. De Renzi et al. (1982) replicated Heilman's (1973) observations of a verbal–motor dissociation apraxia and also described a patient who performed in the opposite manner. The patient failed to correctly perform gestures with visual stimuli but performed well to verbal command. Although most patients perform skilled actions better with vocal stimuli than to command, the authors described two patients who performed better with visual and verbal stimuli than with tactile stimuli.

Pathophysiology. The mechanism proposed by De Renzi et al. to explain these modality-specific apraxias was similar to that proposed by Heilman (1973); namely, there is a disconnection between modality-specific pathways and the center where movements are programmed.

Pantomime Agnosia

Clinical. Agnosia is a failure of recognition that cannot be attributed to deafferentation or a naming disorder. Disorders of discrimination and recognition associated with ideomotor apraxia from posterior lesions (described above) may not be considered to be a form of agnosia because of the associated production deficits. Rothi et al. (1986) reported two patients who could not comprehend or discriminate visually presented gestures, but who performed gestures normally. These patients could, however, recognize tools. These patients could be considered to have pantomime agnosia without object agnosia. Both patients could imitate better than they could comprehend or discriminate gestures. Because they could imitate, their inability to discriminate or comprehend gestures could not be accounted for by a defect in vision or perception. The patient had left-sided temporal–occipital lesions that may have disconnected visual input from the input praxic system. Schwartz et al. (1998) and Larabee et al. (1985) reported patients who had the opposite dissociation. They could not recognize tools but could recognize pantomimes.

Pathophysiology. Studies of patients with object agnosia suggest that these patients have injury to their ventral temporal–occipital "what" system. Whereas Rothi et al. (1986) thought that the ventral "what" visual stream is also important for gesture comprehension, the loss of the ability to name tools with a preserved ability to comprehend gestures reported by Schwartz et al. (1998) in a patient with a ventral lesion suggests that it may be the dorsal "what" visual stream that is important for gesture recognition. In regard to the neuropsychological mechanism, patients with Wernicke's aphasia and pure word deafness can neither comprehend spoken language nor repeat. Whereas the former is thought to be related to destruction of the lexicon (representations of learned word sounds), the latter disorder is thought to be related to an inability of auditory input to gain access to the phonological lexicon. However, patients with transcortical sensory aphasia can repeat (i.e., imitate) in spite of being unable to comprehend, demonstrating that comprehension and imitation are dissociable. This comprehension–imitation dissociation suggests that these two processes are at least in part divergent and are mediated by different parts of the brain. Lichtheim (1855) suggested that while repetition is mediated by a phonological–lexical system that is still functional in transcortical sensory aphasia, comprehension unlike repetition requires semantic processing. Thus, in transcortical sensory aphasia the systems that mediate semantic processing of lan-

Aphasia are impared, or auditory input cannot gain access to these semantic systems (Heilman et al., 1976). The neuropsychological mechanisms underlying impaired gesture comprehension with spared imitation reported by Rothi et al. could be similar. These patients can gain access to the input praxic but the activated praxic representations could not access semantics (see Fig. 11–4).

Several authors have suggested that speech repetition (i.e., imitation) may be performed by using either stored word representations (lexical) or a nonlexical route (McCarthy and Warrington, 1984; Coslett et al., 1987). Just as we can repeat words we have not heard, we can also mimic movements we have never seen or previously engaged in. Perhaps imitation, like repetition, can take place without having to access stored memories of previously learned movements (Mehler, 1987). Because there are no memory stores for unfamiliar movements, the praxic could not be accessed and the patient had to rely on a nonrepresentational route that was impaired. Perhaps the patients with ideomotor apraxia who improve with imitation can use this nonrepresentational route, whereas those who do not improve may have an additional deficit in this nonrepresentational route.

Identiational Apraxia

Clinical. There has been much confusion about the term ideational apraxia. The dissociation apraxias discussed above were unfortunately originally called "ideational apraxia" (Heilman, 1973). The inability to carry out a series of acts, an ideational plan, has also been called "ideational apraxia" (Marcuse, 1960; Pick, 1960). These patients have difficulty sequencing acts in the proper order. For example, instead of cleaning the pipe, putting tobacco in, lighting it, and smoking it, the patient with ideational apraxia may first put tobacco in the pipe and then clean it. As noted by Pick (1905), most of the patients with this type of ideational apraxia have had dementia, illusions or confusional states. Most patients with ideomotor apraxia improve with actual object use; however, De Renzi et al. (1965) reported patients who made gross errors with the use of actual tools. De Renzi et al. considered the inability to use actual tools a sign of ideational apraxia. While the inability to use actual tools may be induced by a conceptual disorder, Zangwill (1960) noted that failure to use actual tools may be related to a severe production disorder, ideomotor apraxia. However, as we will discuss in the next section, the nature of the error may reveal if the patient is suffering from a production or conceptual disorder. To avoid confusion between those patients who cannot sequence acts and are said to have ideational apraxia and those who have a conceptual disorder, we term this latter disorder conceptual apraxia.

Pathophysiology. Most patients with ideational apraxia suffer with some form of dementia. Both lesion (Mateer, 1990) and functional imaging studies (Wildgruber et al., 1999) suggest that the frontal lobes are critical for sequencing. Vascular or arteriosclerotic dementia is more often associated with frontal dysfunction that is early Alzheimer's disease and vascular dementia is more often associated with sequenc- ing deficits (Starkstein et al., 1996); however, no studies have attempted to determine which forms of dementia are most frequently associated with ideational apraxia or if ideational apraxia can be observed with isolated lesions.

Conceputal Apraxia

Clinical. Whereas patients with ideomotor apraxia make production errors (e.g., spatial and temporal errors) when pantomiming, imitating, or even using actual tools, patients with conceptual apraxia may make content errors resulting from an inability to select the actions associated with the use of specific tools, utensils, or objects (tool–object action knowledge). Therefore, patients with this type of conceptual error make
content errors (De Renzi and Lucchelli, 1988; Ochins et al., 1989). For example, when asked to pantomime how to use a screwdriver, the patient may pantomime a hammering movement or use the screwdriver as if it were a hammer. Patients with ideomotor apraxia may make arcs rather than twisting on fixed axis, but do demonstrate the knowledge of the screw-turning action of screwdrivers. Making content errors (i.e., using a tool as if it were another tool) can also result from object agnosia. However, Ochins et al. (1989) reported a patient who could name tools but used them inappropriately (e.g., used a tube of toothpaste as a toothbrush and a toothbrush as a fork). Because this patient could name the tool he was not agnostic. However, because he could not associate the correct action with the tool he was considered to have a conceptual apraxia.

Patients with conceptual apraxia may be unable to recall which tool is associated with an object (tool-object association knowledge). For example, when shown a nail that has been partially nailed into a piece of wood, they may be unable to select a hammer from an array of tools. Instead, they may select a screwdriver. This conceptual defect may also be in the verbal domain: patients may be unable to name or point to a tool when its function is discussed, even though they can name the actual tool and point to it when named by the examiner. They may also be unable to describe verbally the function of a particular tool.

Patients with conceptual apraxia may also be unaware of the mechanical advantage afforded by tools (mechanical knowledge). Therefore, when they are presented with a partially driven-in nail and are expected to complete the task without an available hammer, these patients may not select an alternate tool that is hard, rigid, and heavy, such as a wrench or pliers. Instead, they may select an alternate that is flexible and lightweight, such as a handsaw. Lastly, patients with conceptual apraxia may also be unable to construct tools (tool fabrication).

Because patients with Alzheimer’s disease may have impaired semantic memory even early in the course of the disease, Ochins et al. (1990) studied patients with degenerative dementias of the Alzheimer type for these four components of conceptual apraxia. They found that patients with Alzheimer’s disease often have conceptual apraxia. When compared to controls, they were impaired on all four levels discussed above. It was also learned that some elements of conceptual apraxia may even be seen in patients who do not have either ideomotor apraxia or semantic language impairment.

Heilman et al. (1987) also demonstrated that conceptual apraxia may be associated with focal cerebral diseases such as cerebral infarction. In right-handers, it was primarily injury to the left hemisphere that induced conceptual apraxia. Further evidence that these representations are lateralized comes from the observation of a patient who had a callosal disconnection and demonstrated conceptual apraxia of the nonpreferred (left) hand (Watson and Heilman, 1983). However, the subject investigated by Ochins et al. (1989) was left-handed and rendered conceptually apraxia by a lesion in the right hemisphere, suggesting that both production and conceptual knowledge have lateralized representations and that such representations are usually contralateral to the preferred hand.

Pathophysiology

The lesion associated with conceptual apraxia when testing tool use (tool-object action and tool-object association knowledge) has been localized to the posterior regions of the left hemisphere by Liepmann (1920), who thought that this knowledge in stored in the caudal parietal lobe. De Renzi and Lucchelli (1988) thought that the critical area was in the temporoparietal junction. Although Heilman et al. (1997) found that in right-handers conceptual apraxia was associated with left hemisphere lesions, they did not find a critical hemispheric locus.

Ideomotor and conceptual apraxia often occur, but the finding that there are patients with ideomotor apraxia who do not demonstrate conceptual apraxia and patients with conceptual apraxia who do not demonstrate ideomotor apraxia provides evidence for the postulate that the conceptual and production systems are independent (Rapcsak et al., 1995; Heilman et al., 1997). However, to perform

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skilled acts to command or when presented visually with tools or objects, these conceptual and production systems must interact. The recognition and naming of gestures also require that these systems interact. This is diagrammatically depicted in Figure 11–4 (Rodhi et al., 1991).

BUCCOFACIAL APRAXIA (ORAL APRAXIA)

Clinical

Hugheinus Jackson (cited by Taylor, 1932) was the first to describe buccofacial or oral apraxia (nonprotrusion of the tongue). Patients with oral apraxia have difficulty performing learned skilled movements with the face, lips, tongue, cheeks, larynx, and pharynx on command. For example, when they are asked to pretend to blow out a match, suck on a straw, lick a rubber, or blow a kiss, they will make incorrect movements. Poock and Kerschensteiner (1975) found several types of errors. Verbal descriptions may be substituted for the movement: the oral apraxic asked to pantomime blowing out a match may respond by saying “blow.” Other errors include movement substitutions and perseverations. Raade et al. (1991) noted that patients with buccofacial apraxia make content, spatial, and temporal errors. Mateer and Kimura (1977) demonstrated that imitation of meaningless movements was also impaired, providing evidence that oral apraxia is not a form of ataxia. Although many of these patients do not improve with imitation, they consistently improve dramatically when seeing or using an actual object (e.g., a lighted match). Raade et al. (personal communication) also demonstrated that some patients with buccofacial apraxia have impaired comprehension of buccofacial gestures.

Pathophysiology

In order to learn if impairment of the same system could account for both buccofacial and limb ideomotor apraxia, Raade et al. (1991) studied the co-occurrence of these apraxias, the type of errors made by apraxic patients, and lesion sites. Forty percent of their subjects had only one type of apraxia. Baso et al. (1980) reported dissociations in 23% of their subjects, and De Renzi et al. (1968), in 25% of theirs. Raade et al. (1991) also found different error types. Whereas patients with limb apraxia made more errors with transitive than with intransitive movements, Raade and colleagues found no difference in errors between transitive and intransitive movements in patients with buccofacial apraxia. Lastly, lesion sites were found to be different.

Tognola and Vignolo (1980) studied patients who were unable to imitate oral gestures. The critical areas for lesion included the frontal and central opercula, anterior insula, and a small area of the first temporal gyrus (adjacent to the frontal and central opercula). Tognola and Vignolo (1980) and Kolb and Milner (1981) found that parietal lesions were not associated with oral apraxia, but they did not test performance to command. Benson et al. (1973), however, described patients with parietal lesions who exhibited oral apraxia to command.

Some authors have classified the phonological selection and sequencing deficit of nonflu- ent aphasia as “apraxia of speech” (Johns and Daley, 1975; Brown and Daley, 1972). Although Picciniro and Vignolo (1967) noted that 90% of patients with Broca’s aphasia have oral apraxia, it would seem unlikely that buccofacial apraxia causes this phonological disturbance because there are patients with nonflu- ent aphasia who do not have oral apraxia. It can be argued, however, that oral and verbal apraxias are points along a continuum, sharing a common underlying mechanism. It could be hypothesized that speech requires finer coordination than does response to a command such as “blow out a match”; therefore, the effortful, phonologically inaccurate speech of the nonfluent aphasic may still be caused by an apractic disturbance affecting speech more than oral, nonverbal movement. Oral apraxia and the speech production deficits associated with Broca’s aphasia often coexist, but they can also be completely dissociated (Heilman et al., 1974), suggesting that, at least in part, the anatomic system that mediates facial praxis is not the same as those that mediate the movements used in speech. Furthermore, because patients may have conduction aphasia with or without oral apraxia (Benson et al., 1973), oral apraxia...
May coexist with fluent speech. If one attributes the non fluent disorders of speech in patients with Broca’s aphasia to a generalized oral motor programming deficit, one cannot explain how oral apraxia may be associated with the fluent speech seen in conduction aphasia. In addition, we have examined a patient with aprasia (non fluent speech with intact writing skills) who did not have oral apraxia. If the speech deficits exhibited by left hemisphere impaired patients is induced by a motor deficit, this motor programming defect is strongly linked to the language and phonological systems and is not a generalized oral motor programming deficit.

**DISEASES THAT MAY INDUCE APRAAXA**

Any disease that destroys or injures portions of the cerebral cortex or portions of the thalamus (Nadeau et al., 1984) may induce apraxia, including stroke (infarctions and hemorrhages), trauma, and tumors. Apraxia may also be associated with degenerative diseases. For example, ideomotor, ideational, and conceptual apraxias are often seen in patients with Alzheimer’s disease (Ochipa et al., 1988; Paposca et al., 1985). In cortico basal degeneration, ideomotor and limb kinetic apraxia may be one of the first symptoms or signs. Usually the ideomotor apraxia is of the innervatory-executive type (Jacobs et al., 1999) Often this apraxia is asymmetrical or even unilateral. Although Leiguarda et al. (1987) reported that apraxia may be associated with Parkinson’s disease, other laboratories have been unable to replicate this finding. Apraxia has also been reported to be associated with Huntington’s disease (Shelton and Knoiman, 1991) Finally, apraxia may also be sign and symptom of a developmental disorder.

**RECOVERY FROM APRAAXA AND TREATMENT**

Apraxia may occur over a period of 6 months. However, some degree of apraxia may persist (Sunderland, 2000) and may never disappear, as it can interfere with activities of daily living (Bjorneby and Revnag, 1985). Basso et al. (1987) demonstrated that patients with apraxia from anterior lesions recover better than those with posterior lesions. In addition, the presence of a right hemisphere lesion did not appear to retard recovery, suggesting that recovery was mediated by uninjured portions of the left hemisphere. The portions of the brain that are responsible for recovery are unknown. There are several possible scenarios. Damaged areas may recover. Undamaged portions of the hemisphere dominant for praxis may take over function for the damaged areas. Possibly, the nondominant hemisphere may also have a residual ability to compensate for the damaged hemisphere.

Strokes patients are often anosognosic for their apraxic disability (Rothi et al., 1990) or attribute their disabilities to right hemisphere/ineptness and inexperience using their left arm. Therefore, these patients often do not request therapy. In addition, no information is presently available on methods for, or efficacy of, treatment of apraxia. However, information from animal recovery models (Basso and Horn, 1983) suggests that therapy should be aimed initially at restitution of function: the underlying disorder should be treated so that maximum function can be achieved within the limits set by the recovery process. Then other therapeutic measures can be attempted to foster substitution strategies. Compensatory strategies can help these apraxic patients develop the skills needed to perform activities of daily living (van Heugten et al., 1988) apraxic patients should be taught alternative strategies for performing tasks that pose difficulty for them.

**REFERENCES**


CLINICAL NEUROPSYCHOLOGY


APRAAXIA


