# Apraxias

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# Apraxias

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**Article Contents**



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**Apraxia is defined as the difficulty or inability to perform learned skilled actions. Identifying apraxia in patients has prognostic implications. The praxis network is predominantly in the left hemisphere, and comprises parietal lobe regions that connect to several circuits involving the frontal, temporal and occipital cortices and the basal ganglia. This review discusses the types of apraxia and the disorders associated with apraxia, highlighting studies based on corticobasal syndrome and stroke as disease models. The evolution of historical concepts of praxis leading up to the current 'pathway' models is discussed in the context of neuroanatomical and imaging studies. Bedside testing and interpretation of apraxia are elucidated with examples.**

## Introduction

The literal meaning of the Greek term 'apraxia' is 'without action'. Apraxia in behavioural neurology refers to the loss of the ability to carry out learned, skilled actions in the absence of motor, sensory, coordination or comprehension abnormalities (Rothi and Heilman, 2003). It is to be differentiated from akinesia, which is defined as a general failure to initiate movement in the absence of weakness (Heilman and Watson, 2008). Apraxia is a helpful localising sign on the mental status examination and often predicts disability in patients with stroke or dementia. It can affect both sides of the body, even when the underlying lesion is unilateral. Apraxia can occur in the absence of any language deficits, despite the proximity of cortical areas involved in praxis and language processing (Goldenberg, 2009). Since the earliest descriptions, the concept of apraxia has expanded to include 'unskilled' and 'novel', as opposed to 'skilled and learned' movements.

There are several different forms of apraxia, and these have been variously organised based on the body part affected and the specific type of dysfunction. This review will focus on the most commonly described types of apraxia, including ideomotor, ideational/conceptual, limb-kinetic and orofacial types. The designation of certain other disorders of action as apraxia is controversial, and this will be addressed below. Apraxia is a hallmark feature of corticobasal syndrome (CBS), a progressive condition characterised by frontal, parietal and basal ganglia dysfunction. This syndrome is associated with neurodegenerative diseases such as corticobasal degeneration (CBD), progressive supranuclear palsy (PSP), Alzheimer's disease (AD) and rarely, dementia with Lewy bodies (DLB). Clinically, CBS involves akinesia (lack of movement) and rigidity (stiffness) in combination with cortical signs such as apraxia (Kouri *et al*., 2011; Wadia & Lang, 2007). As such, CBS is a useful model to study the anatomic correlates of praxis, and this model is discussed below (Gross and Grossman, 2008) in addition to other praxis models. In addition, we touch upon findings regarding apraxia in progressive nonfluent aphasia (PNFA), a subtype of frontotemporal lobar degeneration (Rohrer *et al*., 2010).

## Epidemiology

There are no available studies assessing the collective prevalence of the various types of apraxia. Apraxia is prevalent in 25.3% of 'first stroke' patients, 51.3% of left hemispheric strokes and 6% of right hemispheric strokes (Zwinkels *et al*., 2004). It is also reported in several dementia syndromes such as AD and frontotemporal lobar degeneration (FTLD) and is often observed in head trauma involving the left parietal lobe, which makes it likely to be encountered in general neurological practice. Limb apraxia is highly prevalent in CBS (70–80%) and is a key defining clinical feature of CBS (Armstrong *et al*., 2013; Stamenova *et al*., 2009). CBD, its most common neuropathological correlate, is rare, with an estimated prevalence of 2–7 per 100 000 individuals (Togasaki and Tanner, 2000).

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# Clinical Subtypes

Liepmann (1920), in his first systematic descriptions of apraxia, suggested 3 different types: limb-kinetic apraxia, ideomotor apraxia, and ideational apraxia. Subsequent studies of anatomical substrates and various disorders where apraxia is a key feature have brought forth descriptions of other types of apraxia. Whether all of these are in fact disorders of 'skilled action' is debatable (Coslett, 2018). This controversial nature of apraxia is also reflected in the highly variable rules behind naming of apraxia types and apraxic phenomena. Some are named based upon the body parts involved (e.g. orofacial, ocular motor), some are named after their putative mechanisms (e.g. ideational, constructional), others are named after the tasks that are involved (e.g. writing, speech, eyelid opening), and in rare cases, naming involves the neuroanatomical substrate (e.g. callosal apraxia). Several authors have raised concerns about misclassification of apraxias (Zadikoff and Lang, 2005). In an attempt to be as comprehensive as possible, we will discuss both the traditional descriptions of apraxias and the nontraditional apraxias. See **Table 1** for a summary of apraxia subtypes. Practical clinical assessment of apraxias at the bedside is discussed in a separate section.

## Ideomotor apraxia

Ideomotor apraxia is the quintessential apraxia type, defined as an impaired performance of skilled motor acts despite intact sensory, motor and language functions (Rothi and Heilman, 2003). It is typically demonstrated when a patient is asked verbally to perform a gesture with a limb. Most researchers also include the inability to imitate another person's gesture, to perform the appropriate action in response to a visually presented object, or to carry out a movement using the actual object in this type of apraxia. Impaired mimicking of meaningless gestures, such as made-up hand postures, may suggest a deficit in converting visual information into action, rather than a deficit in retrieving encoded action sequences. This is also considered ideomotor apraxia by some researchers (Gross and Grossman, 2008; Sunderland, 2007), but contested by others (Coslett, 2018).

Patients with ideomotor apraxia show spatial and temporal (timing-related) errors affecting limb position in space, configuration, amplitude, timing and sequencing. They often use their limb as an object, rather than demonstrating how to use that object. Although patients may have difficulty miming the use of an object, they may be able to perform the same action in their daily lives without difficulty using the actual object. This phenomenon has been called the 'voluntary–automatic dissociation' (Leiguarda and Marsden, 2000).

Patients with ideomotor apraxia may exhibit differing degrees of impairment depending on testing conditions. As such, patients typically have greatest difficulty performing gestures when responding to verbal commands. They usually have less difficulty imitating a gesture or acting in response to an object that is shown to them; and they may be least impaired when asked to use the object itself (Randerath *et al*., 2011). However, these

**Table 1** Types of apraxia and their neuroanatomical correlates

| Type of apraxia | Description | Neuroanatomical correlates |
|---|---|---|
| Ideomotor | Impaired performance of skilled motor acts despite intact sensory, motor and language function. Usually assessed by verbal command to perform or imitate a gesture. Voluntary–automatic dissociation typically present | Lesions in bilateral frontal and parietal cortices, frontoparietal white matter connections and basal ganglia |
| Ideational | Difficulty carrying out a sequence of actions in performance of a complex, multistep task (e.g. mailing a letter) | Extensive left hemisphere damage |
| Conceptual | Loss of object or action knowledge: misuse of objects, difficulty matching objects and their actions, unawareness of the mechanical advantage provided by tools, inability to judge whether a gesture is well- or ill-formed | Posterior left hemisphere damage |
| Limb-kinetic | Inaccurate or clumsy distal arm or leg movements. Voluntary–automatic dissociation typically absent | Lesions involving connecting sensorimotor fibres of the hand (frontoparietal white matter) |
| Orofacial | Impairment of skilled volitional movements involving the face, mouth, tongue, larynx and pharynx (e.g. blowing out a candle). Considered a subtype of ideomotor apraxia | Inferior frontal, deep frontal white matter, insula and basal ganglia lesions |
| Dressing | Difficulty mapping a piece of clothing onto the spatial configuration of the body, thereby interfering with putting on clothing (a coat, T-shirt) | Parietal lesions |
| Constructional | Inability to copy visually presented information | Right parietal, frontal lesions |
| Writing | Difficulty using a writing tool to form letters | Superior frontal, parietal lesions |
| Gait | Impaired gait without associated weakness, as seen in vascular parkinsonism, normal pressure hydrocephalus | Frontal lesions, lesions affecting frontal–basal ganglia connections |
| Eyelid opening | Difficulty voluntarily opening the eyes in the absence of associated eyelid spasm | Medial frontal lobe, basal ganglia, upper brainstem lesions |
| Speech | Poor coordination of motor speech apparatus | Premotor, supplementary motor cortex lesions |

typical patterns have been contradicted by numerous reports (De Renzi *et al*., 1982; Fukutake, 2002; Merians *et al*., 1999).

Upon testing, apraxic patients' performance may differ depending on gesture type: *transitive* (involving an object; for example using a hammer) versus *intransitive* (a symbolic gesture not involving an object, for example waving goodbye). These meaningful gestures can be contrasted with meaningless gestures (Gross and Grossman, 2008). Given these dissociations, it is important to evaluate praxis using a broad range of tasks.

The role of visual cues is also important to consider, as some patients may have co-occurring visuospatial deficits. Visual information can potentially mitigate deficits of motor programming and execution due to loss of sensory feedback in apraxia (Gross and Grossman, 2008), and patients who do not benefit from visual input have been described (Graham *et al*., 1999). Likewise, cortical sensory loss (difficulty interpreting haptic information, such as agraphesthesia – the inability to interpret numbers written in one's hand without looking – or difficulty identifying objects by touch) may contribute to functional deficits in patients with apraxia.

## Ideational and conceptual apraxia

In ideational apraxia, patients have difficulty carrying out a sequence of actions in the performance of a complex, multistep task, such as mailing a letter. Ideational apraxia is often seen in patients with extensive left hemisphere damage, dementia and delirium (Rothi and Heilman, 2003). Problems with ordering actions may be due in part to executive and memory impairments, or to an overall deficit in cognitive resources (Giovannetti *et al*., 2002; Weintraub, 2000).

Some researchers have made a distinction between ideational and conceptual apraxia (Ochipa *et al*., 1992). In contrast to a disorder of action sequencing, patients with conceptual apraxia demonstrate loss of object or action knowledge. They may misuse objects, have difficulty matching objects and their actions, be unaware of the mechanical advantage provided by tools, or be unable to judge whether another's gesture is well- or ill-formed (Rothi and Heilman, 2003). Conceptual deficits often can be seen in patients with dementia who have a disorder of semantic memory, and have been associated with lesions of the left temporal lobe (Gross and Grossman, 2008). Importantly, both ideational and conceptual apraxias often lead to severe disability in the performance of activities of daily living (Rothi and Heilman, 2003).

## Limb-kinetic apraxia

Limb-kinetic apraxia has been used to describe inaccurate or clumsy distal arm or leg movements. It is typically noted in the limb contralateral to the affected hemisphere (Rothi and Heilman, 2003). For instance, Heilman *et al*. (2000) used selective hemisphere anaesthesia to demonstrate left hemisphere dominance for motor deftness in right-handed epilepsy patients with typical, left-sided language lateralisation. In addition, several studies have revealed that the left hemisphere influences the ipsilateral left hand more than the right hemisphere influences the right hand (Heilman and Watson, 2008). It is possible that the dominant left hemisphere influences the right hemisphere's motor programme via the corpus callosum, the largest white matter structure connecting the two cerebral hemispheres (Heilman and Watson, 2008). Thus, *bilateral* limb-kinetic apraxia may be observed with lesions limited to the left hemisphere.

This disorder has been associated with structural lesions of the frontal cortex and can be difficult to differentiate from associated limb weakness (Leiguarda and Marsden, 2000). Limb-kinetic apraxia has also been observed in patients with CBS and PSP (Leiguarda *et al*., 1997; Leiguarda *et al*., 2003; Quencer *et al*., 2007). In all cases, it can be difficult to parse limb-kinetic apraxia from the extrapyramidal features of these disorders (Graham *et al*., 1999), which include dystonia, parkinsonism and dyskinesias. Limb-kinetic apraxia tends to be independent of modality (e.g. verbal command versus imitation). It differs from classical ideomotor apraxia because limb-kinetic apraxia typically has no voluntary–automatic dissociation, or the superior performance of spontaneous actions compared with the same actions performed on command (Leiguarda and Marsden, 2000).

## Orofacial apraxia

Orofacial apraxia (also called oral or buccofacial apraxia) is characterised by an impairment of skilled volitional movements involving the face, mouth, tongue, larynx and pharynx. It is tested by asking patients to imitate both transitive (e.g. sucking on a straw, blowing out a candle) and intransitive (e.g. whistling) gestures (Stamenova *et al*., 2009). Orofacial apraxia has been associated with inferior frontal, deep frontal white matter, insula and basal ganglia lesions (Ozsancak *et al*., 2004). Automatic gestures involving the same muscles are often preserved, as is the case with ideomotor limb apraxia. Orofacial apraxia frequently coexists with limb apraxia, prompting many to consider orofacial apraxia as a subtype of ideomotor apraxia. However, orofacial and limb apraxia can be dissociated, suggesting that the neural systems underlying these disorders are at least partially separable (Ozsancak *et al*., 2004).

## Other apraxias: constructional, dressing, writing, gait, gaze, apraxia of eyelid opening, speech apraxia

The term 'apraxia' has been applied to a wide variety of clinical phenomena. Several types of motor dysfunction have been described in reference to the performance of specific actions, including constructional, dressing and writing apraxias. *Constructional apraxia*, or the inability to copy visually presented information such as a geometric design, can be seen in patients with right parietal lesions, where it is likely due to a higher-order visuospatial processing deficit, or in patients with frontal dysfunction, where there is impaired organisation and planning of visual representations (Damasio *et al*., 2000). *Dressing apraxia* appears to be a spatial disorder that interferes with mapping a piece of clothing onto the spatial configuration of the body, thereby interfering with putting on an article of clothing like a coat. As both constructional and dressing apraxias often result from parietal lesions, they may coexist in the same patients.

*Writing apraxia*, also known as *apraxic agraphia*, is characterised by difficulty using a writing instrument to form letters (Grossman *et al*., 2001). The patient has no difficulty demonstrating the use of a writing instrument. The instrument is held correctly, and letter formation proceeds in a remarkably slowed manner that is not automatised. For example, it may take an affected patient one minute to write a single five-letter word. This is quantified by demonstrating slower writing in direct proportion to the length of a word. Letters also may be formed in an odd manner that differs during the course of a writing session; for example, the letter 'a' in the word 'banana' may be formed in three different ways. Letters also may be spatially rotated or flipped. This type of apraxia is associated with disease in superior frontal and parietal regions of the dominant hemisphere.

In contrast to ideomotor apraxia, several forms of apraxia interfere with actions that are not explicitly learned. For instance, *gait apraxia* describes the gait of disorders affecting the frontal lobes or frontostriatal connections such as normal pressure hydrocephalus, vascular parkinsonism and several other conditions (Zadikoff and Lang, 2005). *Apraxia of eyelid opening* refers to difficulty voluntarily opening the eyes in the absence of associated blepharospasm (involuntary contractions of the eyelid); this can be observed in patients with CBS and PSP.

*Apraxia of speech* is an articulatory disorder that appears to involve poor coordination of the motor speech apparatus. The motor system underlying apraxia of speech appears to be independent of the system controlling gestures in limb apraxia (Barrett *et al*., 2002). Speech sounds are misformed, so that sounds that are not part of the speaker's native vocabulary are produced; these are called 'phonetic' errors. This differs from the frequently occurring substitutions and exchanges that occur in the speech of most healthy speakers; these are called 'phonologic' errors as they are governed by the linguistic system of phonology and involve production of recognisable speech sounds in an incorrect order. The rhythm and prosody of speech are also disrupted in apraxic speech. Apraxia of speech is often the most prominent symptom in several neurodegenerative disorders (Josephs *et al*., 2012; Wicklund *et al*., 2014). It can be seen in association with aphasia in FTLD, PSP and CBD, although apraxia of speech also can occur in isolation (Josephs *et al*., 2012; Josephs *et al*., 2014). Childhood apraxia of speech (also known as verbal dyspraxia or developmental apraxia) has also been described and is often idiopathic.

## Apraxia in stroke, corticobasal syndrome and progressive nonfluent aphasia

Apraxia may be associated with other neurologic deficits that cause weakness, executive dysfunction (e.g. difficulty with multistep processes), aphasia (a disturbance in the comprehension or formulation of language), inattention or hemispatial neglect. In stroke patients, several forms of apraxia have been associated with unilateral lesions to the left or right hemisphere. *In patients with left hemisphere damage, apraxia often coexists with aphasia; in patients with right hemisphere lesions, apraxia is often associated with a visuospatial disorder.* While co-occurring aphasia may in some cases account for patients' greater difficulty performing gestures to verbal command, aphasia cannot easily account for deficits with gesture imitation. A single lesion may account for both aphasia and apraxia by affecting the brain structures that contribute to both gesture and language production, or brain structures for gesture production that are near to structures important for language. This is discussed in further detail below in the sections on neuroanatomy and bedside testing of apraxia.

Apraxia frequently occurs as a feature of CBS, a rare clinical condition comprising only approximately 1% of patients clinically diagnosed with parkinsonism (Litvan *et al*., 1997). Clinical features in addition to apraxia include asymmetric rigidity and other unilateral or strikingly asymmetric involuntary movements, cortical sensory loss and alien limb phenomenon (the latter is specific, but not sensitive for CBS). The gradual accumulation of abnormal 4-repeat microtubule-associated protein tau in the frontal and parietal cortices and basal ganglia underlies CBD. Limb apraxia has been reported in up to 70–80% of CBS cases (Zadikoff and Lang, 2005) In an autopsy-confirmed series of cases with CBD, ideomotor apraxia was found in 40% of patients at onset and 72% at the time of death. In addition, orofacial apraxia may be seen in CBS (Zadikoff and Lang, 2005; Ozsancak *et al*., 2004), and constructional, writing and dressing apraxia also may be present.

PNFA or nonfluent variant of primary progressive aphasia (nfvPPA) is a subtype of frontotemporal degeneration that has been associated with *apraxia of speech*. In a neuroanatomical correlation study, Rohrer *et al.* (2010) noted that 69% of their PNFA patients had orofacial apraxia, and 44% had limb apraxia. Severity of orofacial, but not limb or speech apraxia, correlated with estimated disease duration. The severity of speech apraxia correlated with left posterior inferior frontal atrophy; orofacial apraxia with left middle frontal, premotor and supplementary motor cortical atrophy; and limb apraxia with left inferior parietal lobe atrophy.

Among dementia patients, limb apraxia was most associated with posterior cortical atrophy (PCA), AD, as well as logopenic progressive aphasia (LPA) and nonfluent variants of progressive aphasias (Ahmed *et al*., 2016). Of note, AD pathology accounts for the majority of PCA and LPA presentations. Apraxia of speech seemed to significantly differentiate LPA from the PNFA in that it is typically absent in the former. When compared to dementias like behavioural variant frontotemporal dementia (bvFTD) and semantic variant progressive aphasia, limb apraxia may be helpful in differentiating AD pathology.

Many of these disorders have been used as neuroanatomical lesional models to elucidate the mechanisms underlying praxis. This will be explored in the following sections.

# Neuroanatomy of Apraxia: Historical Models

Linguists and neurologists of the late nineteenth century identified the phenomenon of apraxia as part of aphasia, as a deficit in 'recognising the use' of tools, or as a deficit in 'memories of

kinesthetic perception' (Platz, 2006; Rothi and Heilman, 1996). Heymann Steinthal, the nineteenth century German philologist, is credited for using 'apraxia' in scientific literature for the first time in the context of deficits in executing skilled motor task (Rothi and Heilman, 1996). He considered apraxia as an 'amplification of aphasia' (Goldenberg, 2014). Hugo Karl Liepmann (1863–1925), through systematic analysis of neuropathological cases at the turn of the twentieth century, proposed that action representation is found in the left parietal lobe (Liepmann, 1920). To execute an action, he argued that the space-time plan is retrieved from the parietal lobe and conveyed to primary motor areas via the left premotor cortex. From this perspective, the inability to pantomime learned actions in patients with ideomotor apraxia is due to disruption of frontoparietal connections, whereas motor strength and limb movements are preserved due to an intact corticospinal tract. Left parietal lobe damage was thought to underlie ideational apraxia (disruption of the representation of action sequences) and left frontal damage was thought to cause the imprecision of actions found in limb-kinetic apraxia.

Building upon Liepmann's model, Norman Geschwind conceived of apraxia as a phenomenon of disconnection between the posteriorly located receptive 'speech areas' (temporoparietal regions), which are the source of programs for motor action, and the association areas located anterior to the primary motor areas in the dominant frontal lobe (Geschwind, 1965) According to this model, a lesion in the superior longitudinal fasciculus connecting the left inferior frontal lobe to Wernicke's area would, therefore, compromise performance of tasks to verbal command, while sparing the comprehension of viewed gestures. The model also recast the idea of 'language-based' hemispheric dominance toward dominance of the hemisphere that is the 'major source' of motor action programs. Thus, the model explained why a left-handed patient with right hemispheric stroke can have apraxia of the unparalysed right hand, despite intact language function (Geschwind, 1975).

Rothi and Heilman provided evidence for dual-component models of praxis – anterior (execution–production) and posterior (conceptual–representational). According to this model, representations of objects are stored in the left inferior parietal lobe (including angular and supramarginal gyri), and transformed into an executive signal by the premotor cortex (including the supplementary motor area); this is utilised by the primary motor cortex to perform a gesture (Heilman *et al*., 1997). Damage to anterior areas generally causes production deficits, whereas posterior damage gives rise to both abnormal gesture production and gesture comprehension, including difficulty discriminating between normal and abnormal gestures. Lesion studies have provided substantial evidence for this dual-component model (Heilman *et al*., 1982).

While all the above models have merits, and the broad principles of the neuroanatomical correlates remain true today, none of them can adequately explain the anatomical underpinnings and physiological mechanisms of the *full range of apraxia* described above. Advanced structural and functional brain imaging techniques have recently enabled researchers to develop models of praxis that provide a more comprehensive account for the deficits seen in the various types of apraxia.

# Neuroanatomy of Apraxia: Current Thinking

## Dual-pathway model

Studies in the 1980s and 1990s revealed the existence of a bilaterally represented ventral and dorsal visual processing streams. In human models, the ventral 'what' stream projects from occipital cortex to the inferior temporal cortex, retrieving information on identifying an object. The dorsal 'how' stream projects from occipital cortex to the posterior parietal cortex and mediates visually guided actions directed at an object (Goodale and Milner, 1992). Binkofski, Buxbaum and others have developed a praxis model integrating these streams (Binkofski and Buxbaum, 2013). In this model, the inferior parietal lobe integrates ventral occipitotemporal and dorsal parietooccipital streams of higher-order spatial and shape information regarding object use. The dorsal stream was subsequently noted to have two substreams: dorso-dorsal and ventrodorsal. This model suggested that the dorso-dorsal system (the 'grasp' system) processes characteristics of a tool such as size, shape and orientation, whereas the ventrodorsal system (the 'use' system) is concerned with the storage of object-specific actions (**Figure 1**). This 'dual-pathway' model has gained great momentum in the past decade, with many voxel-based morphometric and lesion-symptom mapping studies in stroke patients opening new frontiers in the neuroanatomy of praxis. Kalénine *et al*., for example identified that impaired semantic gesture recognition was associated with damage to the posterior temporal lobe (posterior middle temporal gyrus) and that impaired spatial gesture recognition was associated with damage to the inferior parietal lobule. In this context, they note that the posterior middle temporal gyrus is probably a key node in the association of actions and meanings, whereas the inferior parietal lobule helps encode object-related postures and movements (Kalénine *et al*., 2010).

## Apraxia and aphasia

Lesional image analyses have also helped parse the complex relationship between apraxia and aphasia. By investigating 50 subacute stroke patients, Weiss and colleagues showed that in those with concurrent aphasia and apraxia, lesions were noted in the left inferior frontal gyrus, particularly in an anteroventral subarea of Brodmann area 44 (BA 44). It is notable that BA 44 and BA 45 form the traditional 'Broca's area' involved in speech production in the dominant hemisphere. Apraxia was tested in these patients with 3 tasks: pantomiming the use of a tool, imitation of a meaningful gesture and imitation of a meaningless gesture (all shown through pictures). These authors suggest that the anteroventral subarea of BA 44 is involved in extracting meaning from sensory information and semantic processing, and could explain the deficits in pantomiming and imitating *meaningful* gestures in these patients. It was also noted that deficits in imitating *meaningless* gestures correlated with strokes involving the postcentral gyrus in the parietal lobe (Weiss *et al*., 2016).

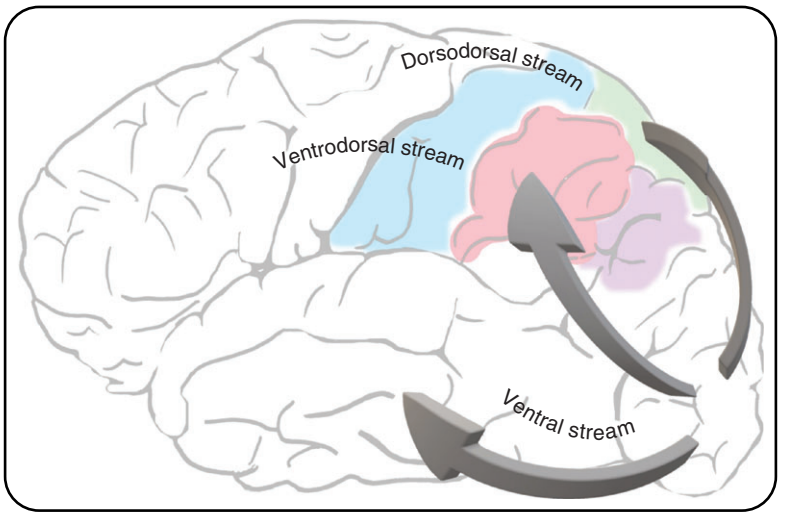


**Figure 1 Schematic representation of the anatomic location of the dorso-dorsal, ventro-dorsal and ventral streams (arrows).** The main parts of the parietal lobe are highlighted: postcentral gyrus (blue), the supramarginal gyrus (red), the superior parietal lobule (green) and the angular gyrus (purple). The arrows representing the streams emerge from the primary visual cortex. The ventral ('what') stream projects from the occipital cortex to the inferior temporal cortex, retrieving information on identifying an object. The dorso-dorsal ('grasp') stream processes characteristics of a tool such as size, shape and orientation. The ventro-dorsal stream ('use') stores object-specific actions.

## Apraxia in models of gestures and tool use

Apraxia, due to its interference with pantomiming, offers rich opportunities for the study of tool use. Sunderland and others evaluated real tool use and action planning in a small sample of four apraxic patients and ten age-matched controls (Sunderland *et al*., 2011). Under timed conditions, the subjects were asked to quickly reach for tools or abstract objects of similar dimensions. Apraxic patients frequently did not invert their hand to appropriately grasp inverted tools; this was dissociated from their relatively preserved ability to invert the hand and avoid a barrier to grasp an abstract object. The frequency of errors on the tool-grasping task correlated with severity of apraxia.

Goldenberg and Randerath (2015) reported that strokes involving the supramarginal and angular gyri of the left inferior parietal lobe were associated with defective pantomiming of tool use and imitation of meaningless hand postures. In an analysis of functional imaging and structural lesion studies in the context of pantomime deficits, Niessen *et al*. reaffirmed the emerging notion of a left hemispheric fronto-temporo-parietal network underlying pantomiming of tool use. They also confirmed the role of the left parietal cortex in both storing and activating motor schemas for tool use (Niessen *et al*., 2014). From acute left hemispheric stroke patients, Hoeren *et al*. inferred that imitation of meaningless hand and finger gestures is associated with nodes of the dorso-dorsal stream, providing visual-motor support for 'on-line' movement control. Pantomiming the use of tools may additionally require the ventrodorsal and ventral streams, which probably assist in accessing stored actions and their relationships with tools (Hoeren *et al*., 2014).

Meaningless gestures, due to being novel and unfamiliar, are thought to be processed without accessing preexisting motor action schemata and motor semantics. This process is, therefore, more demanding and error-prone than imitation of meaningful gestures, as was demonstrated in a large study of left and right hemispheric stroke patients (Achilles *et al*., 2016). Studies evaluating praxis in dementia patients have also noted that even mild AD patients have deficits in imitation of meaningless hand and finger postures, suggesting that early atrophy in the inferior parietal regions in these patients could be placing higher demands on visuospatial processing. In contrast, impairment in pantomiming the use of familiar tools may be correlated with deficits in semantic memory associated with temporal lobe degeneration in late-stage AD (Johnen *et al*., 2015).

In summary, the newer models of praxis propose bilaterally represented ventral and dorsal processing streams, with the dorsal stream further subdivided into dorso-dorsal and ventrodorsal substreams. The dorso-dorsal or the 'grasp' system processes characteristics of a tool such as size, shape and orientation, while the ventrodorsal or the 'use' system stores object-specific actions. The lesions of subareas within Broca's area could explain deficits in pantomiming and imitating meaningful gestures. Meaningless gestures, on the other hand, require processing in the absence of pre-existing motor action schema and may depend more on visuospatial processing. The left hemispheric lateralisation of apraxia has been consistently demonstrated in large studies of stroke patients, and the left fronto-temporo-parietal network involved in pantomiming tool use appears to be intimately involved in the development of apraxia.

# Testing Apraxia at Bedside

Ideomotor apraxia is the most straightforwardly visualised limb apraxia in a clinical setting and hence has been the focus of

both lengthy and abridged apraxia testing scales. Several apraxia scales have been developed over the years and are referenced here (Helm-Estabrooks, 1992; Leiguarda *et al*., 2014; Tessari *et al*., 2015). Individual testing of components of apraxia such as conceptual, conduction, visuoimitative and dissociation apraxia is beyond the scope of this review; below, we aim to provide 'practice pearls' for the general clinician and interested student.

Reliable testing of apraxia depends on the patient's ability to understand commands and move the limbs without weakness. Hence, it is important to first assess the patient's attention, language comprehension and motor strength. Pantomiming the use of a tool is the most difficult for the apraxic patient. Imitating the examiner using a tool is less difficult, and demonstrating the use of a tool when it is provided to the patient is the least difficult (Randerath *et al*., 2011). Hence, we recommend asking the patient to pantomime the use of a tool first; if this fails, we advise asking the patient to imitate the examiner performing gestures; if this fails as well, then the patient can be given objects to demonstrate appropriate gestures involving the object. Items like a pen, a key and a pair of scissors that are often found in a hospital or office setting can be used. A list of examples of commands and actions is provided in **Table 2**.

Apraxia, particularly when present with other deficits, can help localise lesions, and hence it is useful to test praxis of individual limbs, the buccofacial region and axial structures separately. *Transitive* and *intransitive* gestures are tested separately. As discussed earlier, apraxia often coexists with aphasia, which may impair a patient's ability to understand commands. Severe limb apraxia may be associated with impairment in gestures, and severe orofacial apraxia may be associated with impaired verbal communication. Agnosia and spatial neglect are also often associated with apraxia, especially in strokes, and this may significantly impair accurate assessment of apraxia as well.

The patient's ability to understand gestures is tested by demonstrating the gestures to the patient and asking her to indicate what the action represents. Testing *gestured pictures* involves showing the patient the picture of an everyday item and asking them to demonstrate its use through a gesture. Errors in the sequence in which the components of a complex task are performed are the hallmark of ideational apraxia. Asking the patient to choose the relevant items from a set of objects such as toothbrush, toothpaste, a comb and a spoon and demonstrate how she would brush her teeth (for example) may be cumbersome in some clinical settings. Another task would be to ask the patient to demonstrate mailing herself a letter (provide paper, envelope and a pen to fold the paper into the envelope, write the address and seal the envelope).

*Apraxia of speech* in the acute setting of stroke is commonly misdiagnosed as aphasia. Detailed testing at the bedside can be difficult, but if the patient's writing and reading/auditory comprehension are normal, and speech is notable for phoneme prolongation and inter-syllabic segmentation, then apraxia of speech rather than aphasia should be considered (Polanowska and Pietrzyk-Krawczyk, 2016). As discussed under section titled 'Clinical Subtypes', apraxia of speech can distinguish certain FTD variants from AD, in that limb apraxia can be an early feature of the latter. An apraxia of speech rating scale has been recently developed (Strand *et al*., 2014).

Meaningful and meaningless gestures are tested separately. Imitation of meaningless gestures appears not to be influenced by severity of aphasia in left hemispheric stroke patients. Testing meaningless gestures may be a particularly sensitive way to detect imitation deficits independently of which hemisphere is affected, as it depends less on prior motor engrams and more on visuospatial processing (Achilles *et al*., 2016).

## What to look for

Hesitancy and self-correction, or a lack of smooth movement coordination or 'gracefulness' can be seen in milder presentations of apraxia (Helm-Estabrooks, 1992). Common errors in ideomotor praxis include incorrect direction of movements, use of the wrong limb, wrong posture of the limb or digits, and use of a body part as the object. The latter is easily recognisable; for example, when pantomiming the use of a comb, the patient might run his fingers along his hair, or when pantomiming the use of a toothbrush, the patient might use her index finger as the brush. Pretending that the index and middle fingers are the blades of a pair of scissors when asked to cut a piece of paper is another common error. In some cases, the component movements of a complex act may be preserved, but objects can be misused (e.g. combing the face instead of the hair) (Helm-Estabrooks, 1992). Looking for errors in the sequencing of actions in a multistep task can detect ideational apraxia.

# Treatment

Therapy for apraxia is still experimental, and much of the evidence for targeted rehabilitation comes from studies on stroke patients. Compared to conventional rehabilitation for aphasia, a behavioural training program of gestural exercises has been shown to improve limb apraxia specifically and functional independence generally (Smania *et al*., 2006). There is some evidence that using communicative gestures alongside rehabilitation for aphasia in stroke patients may improve not only the practised gestures but also unpractised gestures (Daumüller and Goldenberg, 2010; Rose *et al*., 2013). Anodal stimulation using transcranial direct current (tDCS) over the left parietal cortex improved ideomotor upper limb apraxia in small samples of CBS patients (Bianchi *et al*., 2015) and left hemispheric stroke patients (Bolognini *et al*., 2015). Cholinesterase inhibitors shown to improve cognition in dementia have not been specifically studied for subcomponents of impairments such as apraxia (Liepelt *et al*., 2007). There is no pharmacologic therapy with evidence for improving apraxia currently available.

# Future Directions

As we have noted here, despite recent progress in the understanding of network dysfunction underlying apraxia, there remains disagreement among researchers regarding what specific impairments constitute apraxia. The lack of consensus on nomenclature and the defining criteria for apraxia are also evident. Larger studies with better-defined apraxia cohorts are still lacking.

**Table 2** Examples of commands and actions for testing apraxia

| Body part and type of apraxia | Command/action |
|---|---|
| Oral buccofacial apraxia | 'pretend to … '<br>• sniff a flower<br>• suck through a straw<br>• bite an apple<br>• blow out a candle<br>• lick ice cream on a cone |
| Axial (proximal) transitive | 'show me … '<br>• how you will swing a bat or a golf club<br>• the posture of a boxer<br>• how you dribble a basketball<br>• how you kick a soccer ball<br>• using a bow and arrow at a target practice |
| Distal (limb) transitive | 'imagine you have … '<br>• a comb in your hand and show how you would comb your hair<br>• a hammer and pretend hammering this imaginary nail<br>• a toothbrush and show how you brush your teeth<br>• a pair of scissors in your hand and show how you would cut a sheet of paper |
| Intransitive meaningful gestures | '*Show me how you will … '*<br>• *wave goodbye*<br>• *salute an officer*<br>• *wipe sweat off your forehead*<br>• *stop the traffic in the middle of the road*<br>• *blow a kiss*<br>• *fold hands in a prayer*<br>• make an 'OK' sign[a]<br>• make a victory sign[a] |
| Intransitive meaningless gestures | These are made-up meaningless hand and body gestures. Examples include:<br>  |
| Gestured pictures: | Show the patient pictures of everyday items and say, 'here is the picture of a spatula; here is how I would use the spatula'<br>Ask the patient to demonstrate the use of the item in the picture using a gesture<br>Examples of items:<br>Pen, Spatula, Whistle, Key, Toothbrush, cone ice cream, paintbrush, bow and arrow |
| Ideational apraxia | Patient must be provided with a set of items to do a task. Observe for appropriate use of items, and sequence of actions.<br>'Show us how you brush your teeth'<br>Provide: a toothbrush, toothpaste, a spoon and a comb<br>'Show us how you would mail a letter'<br>Provide: a piece of paper, and envelope, and a pen to write the address |

[a]Some of these gestures can be different, depending on the cultural background of the patient.

Correlating *in vivo* imaging data with neuropathological evaluation of the same patients – the current gold standard for diagnosing neurodegenerative diseases such as CBD – would undoubtedly lend greater insight into the precise neuroanatomical correlates underlying various apraxia types. Until then, network models of apraxia will remain imperfect in their ability to explain disparate clinical observations.

## Glossary

***Alien limb phenomenon*** A clinical finding in which there is involuntary movement of the hand or foot, which is not recognised by the limb's owner as his/her own; the patient's limb moves as if on its own volition.

***Aphasia*** A disturbance of language comprehension and/or expression caused by dysfunction in specific brain regions.

***Apraxia*** The difficulty or inability to perform learned skilled actions.

***Basal ganglia*** A group of deep brain nuclei in vertebrate brains that are connected to a number of other brain areas and are responsible for a number of functions, including voluntary motor control and procedural learning.

***Corticobasal degeneration*** A rare, progressive neurodegenerative disease involving the accumulation of abnormal microtubule-associated protein tau in the frontal and parietal cortices and basal ganglia. Its most common clinical manifestation is corticobasal syndrome.

***Corticobasal syndrome*** A rare clinical condition with features that include asymmetric rigidity and other unilateral involuntary movements, cortical sensory loss, alien limb phenomenon and apraxia. It is most commonly caused by corticobasal degeneration.

***Frontotemporal lobar degeneration (FTLD)*** A group of clinically, pathologically and genetically heterogeneous disorders associated with atrophy in the frontal lobe and temporal lobe of the brain, with sparing of the parietal and occipital lobes. Progressive nonfluent aphasia, a type of FTLD, is associated with apraxia of speech.

***Functional magnetic resonance imaging (MRI)*** An MRI procedure that measures brain activity by detecting associated changes in blood flow. When an area of the brain is in use, blood flow to that region also increases, and this can be detected and visually represented on an MR image.

***Normal pressure hydrocephalus*** A condition that occurs when there is an increase in intracranial pressure due to an abnormal accumulation of cerebrospinal fluid in the ventricles of the brain. It is associated with apraxia of gait.

***Parkinsonism*** A clinical syndrome consisting of one or more of the following: rigidity, tremor at rest, slowness or paucity of movements, and postural instability.

***Positron emission tomography (PET)*** A nuclear medical imaging technique that produces a three-dimensional image or picture of functional processes in the body. The system detects pairs of gamma rays emitted indirectly by a positron-emitting radionuclide tracer, which is introduced into the body on a biologically active molecule. If the biologically active molecule used for PET is FDG (a glucose analogue), the imaged concentrations of tracer indicate tissue metabolic activity through the regional glucose uptake.

***Praxis*** The process by which a theory, lesson or skill is enacted, practised, embodied or realised.

***Voxel-based morphometry (VBM)*** A neuroimaging analysis technique that allows investigation of focal differences in brain anatomy, VBM registers every brain to a template, which gets rid of most of the large differences in brain anatomy among subjects. The brain images are then smoothed so that each voxel (a volume element representing a value on a regular grid in three-dimensional space) represents the average of itself and its neighbours. Finally, the image volume is compared across brains at every voxel.

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