Address correspondence to Dr Glen R. Finney, University of Florida, PO Box 100236, 100 S Newell Drive, Rm L3-100, Gainesville, FL 32610, *finney@neurology.ufl.edu*.

Relationship Disclosure:
Dr Finney reports no disclosure.

Unlabeled Use of Products/Investigational Use Disclosure:
Dr Finney reports no disclosure.

Perceptual-Motor Dysfunction

Glen R. Finney, MD, FFSN

ABSTRACT

Purpose of Review: This article highlights the importance of integrated perceptual information (motor planning, sequencing, and representation) and discusses the integration of these cognitive domains by means of feedforward and feedback loops in the successful acquisition and execution of voluntary behaviors. The article also discusses the dysfunction in the perceptual-motor process that can occur with neurologic injury, resulting in apraxias, agnosia, hemineglect, and Balint syndrome.

Recent Findings: A combination of functional imaging and lesional studies continues to refine our understanding of the role of the posterior parietal region in the integration of perception with motor action. Different disorders provide contrasting views into the nature of perceptual-motor function and its disruption. Novel rehabilitation techniques may provide improved function in the future.

Summary: Studies continue to demonstrate the importance of unimodal and heteromodal association cortices, as well as the extrapyramidal system (especially the basal ganglia) in perceptual-motor functions across a wide range of activities and disease states. The nondominant hemisphere dictates where attention and intention are to be directed in space, and the dominant hemisphere provides information on how to accomplish skilled complex actions. While the role of perceptual-motor dysfunction in developmental disorders has been long considered, the role of perceptual-motor dysfunction in neurodegenerative diseases, from Parkinson disease to corticobasal syndrome to posterior cortical atrophy, is becoming more apparent. A clear need exists for more robust rehabilitation strategies in these neurodegenerative diseases.

Continuum (Minneap Minn) 2015;21(3):678–689.

INTRODUCTION

The term perceptual-motor dysfunction is a convenient way of addressing a variety of high-level cognitive dysfunctions of the unimodal and heteromodal association cortices and associated structures. The unimodal association cortex receives input from only one sensory modality upon which it performs higher order processing, whereas the heteromodal association cortex receives processed information from multiple sensory modalities from the unimodal association cortices. The *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*) adopted this terminology for one of the six cognitive domains—along with complex attention, executive function, learning and memory, language, and social cognition—that should be assessed in diagnosing either minor or major neurocognitive disorders.[1]

The unimodal association cortex is involved in the high-end information processing of primary sensory data, such as visual, auditory, or somatosensory information. The unimodal association cortex lies adjacent to the primary sensory cortex for that same sensory modality and only works to process input from that one sensory modality. These processed data are then passed along to other parts of the brain, such as the heteromodal association cortex, for further integration and processing.

In effect, the heteromodal association cortex integrates processed inputs originating from several sensory modalities (**Table 6-1**).

Perception used for motor programming requires accurate integration of sensory information from multiple modalities, specifically proprioceptive (kinesthetic), visual/visuospatial, and vestibular. Given the complexity of these perceptual-motor processing networks, any number of lesions along the pathway exiting the primary sensory cortex to the unimodal association cortex to the heteromodal association cortex to the motor planning areas of the brain can perturb the perceptual-motor systems and lead to failure of multimodal integrated perception. This review discusses apraxias, agnosias, and hemineglect as the most important examples of dysfunction at the level of the unimodal association cortex, either through lesions of the unimodal association cortex itself or disconnections from its inputs or outputs. The article then discusses Balint syndrome as an example of dysfunction in the heteromodal association cortex, provides Parkinson disease as an example of the critical role of the basal ganglia in perceptual-motor integration, and addresses developmental perceptual-motor syndromes. The article concludes with a discussion of rehabilitation strategies for patients with perceptual-motor dysfunction.

APRAXIA

Praxis is the ability to perform overlearned fine motor tasks; therefore, apraxia is the inability to perform these tasks in the absence of other neurologic deficits to account for the impairment in function.[2,3] There are several types of apraxia, but the classic model for apraxia is ideomotor apraxia. Ideomotor apraxia results in impairment in executing use of a tool (transitive ideomotor apraxia) or hand gesture (intransitive ideomotor

TABLE 6-1 Primary and Unimodal Sensory Cortices

Sensory Modality	Primary Cortex	Unimodal Association Cortex
Somatosensory	Postcentral gyrus of parietal lobe	Anterior parietal lobe
Auditory	Transverse temporal gyrus (Heschl gyrus)/superior temporal gyrus	Superior temporal lobe/ Wernicke area
Gustatory	Anterior insula, frontal operculum of inferior frontal gyrus	Dysgranular insular cortex, agranular insular cortex, perirhinal cortex
Olfactory	Pyriform cortex (anterior portion of the uncus of the parahippocampal gyrus of the temporal lobe), prepyriform area, entorhinal cortex, periamygdaloid cortex	Entorhinal cortex, agranular and dysgranular orbitofrontal cortex medially, and agranular insula
Visual	Striate cortex of the occipital lobe	Extrastriate cortex of the occipital lobe, inferior temporal cortex, posterior parietal cortex

KEY POINTS

- Perceptual-motor dysfunction is a term covering a wide range of disorders, including disorders of gnosis and visuoconstructional and perceptual-motor praxis, and is involved in the high-order integration of perception for the purpose of guiding actions, as well as the motor learning, representation, sequencing, and processing of those complex actions, including feedforward and feedback planning and correction of movements in near–real time.
- Every sensory modality (seeing, hearing, feeling, tasting, and smelling) has dedicated unimodal association cortices where high-level processing of that specific sensory type occurs, and where damage causes sensory-specific deficits without actual loss of sensation.
- Unimodal association cortices send information forward to heteromodal association cortices, which integrate multiple sensory percepts into high-order multimodal perception.
- Praxis is the ability to perform complex overlearned fine motor tasks such as using tools like scissors or a screwdriver or making a hand gesture such as saluting or waving goodbye. Lesions of the dominant hemisphere posterior parietal cortex are the most likely to result in apraxias.

apraxia) and may present with malformed hand posture, spatial movement errors, or use of a body part as the tool. For a more difficult pantomime of tool use, such as cutting with scissors, even people without apraxia may initially try to use their hand as a pair of scissors. However, people without apraxia should correct with prompting, whereas those with true ideomotor apraxia cannot correct their actions. Ideomotor apraxia can result from lesions to either the dominant parietal cortex, supplementary motor cortex, or in the case of a unilateral ideomotor apraxia of the nondominant side, this could also be because of damage to the corpus callosum, referred to as callosal apraxia (**Figure 6-1**[4]).

Ideational apraxia refers to impairment in executing a sequence of actions to perform overlearned complex tasks (eg, making a peanut butter and jelly sandwich) and localizes to the dominant posterior parietal cortex. Conceptual apraxia is an impairment in knowledge (semantic memory) of tool use and mechanical advantage (defined as the benefit gained by the use of a mechanism in transmitting force) and also localizes to the dominant posterior parietal cortex. These two types of apraxia are earlier stages in the initiation of tool use compared to the classic example of praxis, ideomotor praxis. Limb-kinetic apraxia is a type of apraxia representing loss of deftness rather than faulty representations of movement. Limb-kinetic apraxia manifests as impairment of distal limb movement and can localize to either the supplemental motor or motor cortex of the frontal lobes or basal ganglia.

Other types of apraxia include orofacial apraxia (also called buccofacial apraxia), which is an impairment of

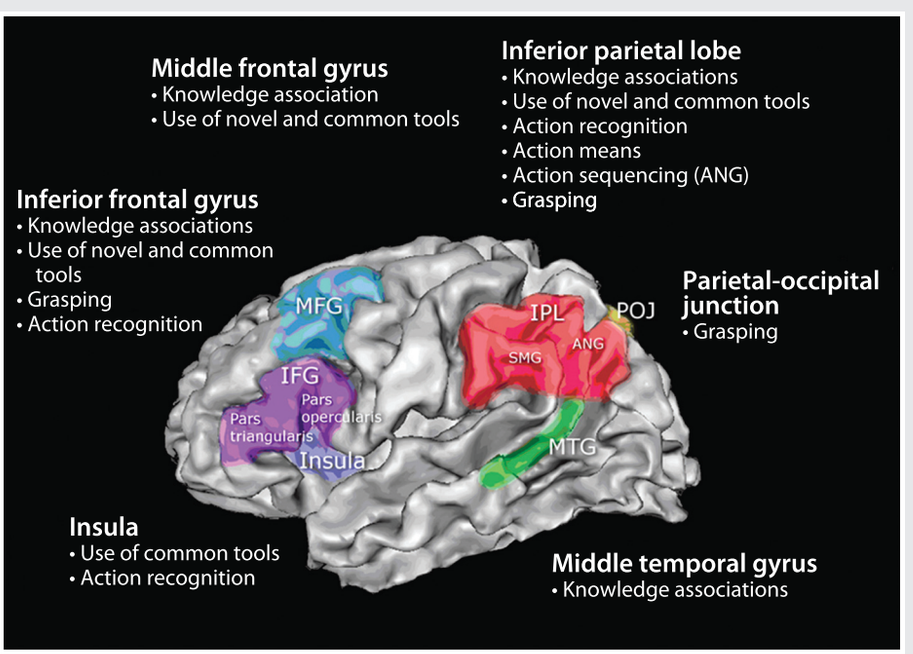

FIGURE 6-1 Cortical areas involved in praxis.

MFG = middle frontal gyrus; IPL = inferior parietal lobe; POJ = parietal-occipital junction; IFG = inferior frontal gyrus; SMG = supramarginal gyrus; ANG = angular gyrus; MTG = middle temporal gyrus.

Reprinted with permission from Bieńkiewicz MM, et al, Front Psychol.[4] © 2014 Bieńkiewicz, Brandi, Goldenberg, Hughes and Hermsdörfer. *journal.frontiersin.org/article/10.3389/fpsyg.2014.00353/full*.

skilled movements of the mouth. Orofacial apraxia localizes mostly to either the inferior frontal lobe or basal ganglia and leaves the patient with difficulty moving the face in complex motor patterns. Apraxia of speech is an impairment of skilled formation of sounds and words and localizes to the dominant supplementary motor cortex and Broca area. While orofacial apraxia is a distinct entity and relatively unrelated to aphasia, apraxia of speech is akin to expressive aphasia in the sense that it localizes very closely to the location for expressive language formation and is essentially the next step in creation of word sounds. Orofacial apraxia can be assessed by simple pantomime on command of complex facial movements such as blowing out a match or licking a lollypop. Apraxia of speech, on the other hand, is assessed by observation of expressive speech.

The term constructional apraxia, where patients have difficulty with drawing, is often used for the visuoconstructional deficits referred to in the *DSM-5*, but unlike other apraxias, these tend to be heterogeneous in their cause as they require both visual-perceptive and motor-productive features. Lesions of either hemispheres, as well as neurodegenerative disorders such as Alzheimer disease, have been reported to result in constructional apraxias. Dominant hemisphere lesions tend to cause a loss of fine detail in drawing and copying, whereas nondominant hemisphere lesions result in loss of overall order and distortion, as well as an element of hemispatial neglect. Additionally, frontal cortex damage can lead to executive errors in planning out drawing and copying. **Case 6-1** is an example of ideomotor apraxia (**Table 6-2**).

AGNOSIAS

There can be several different types of agnosias and related unimodal dysfunctions within one sensory modality. It is also useful to note that agnosias can be either apperceptive or associative. Apperceptive agnosia results from a failure of perception itself, so that the patient cannot draw, match, or otherwise indicate a perception of the thing. An individual with associative agnosia, on the other hand, can match things, draw them, and otherwise indicate perception of the thing, but cannot access any knowledge from semantic memory about the thing, such as its name, what it is used for, or associated features. Visual agnosia is a well-studied form of this dysfunction. Unimodal visual association cortex lesions can lead to visual agnosia (inability to recognize objects by vision),[5] achromatopsia (loss of color awareness),[6] prosopagnosia (inability to recognize and distinguish faces),[7] and alexia (inability to recognize words, especially whole words).[8] **Case 6-2** provides an example of how surprising

KEY POINT

- Patients with agnosias have problems with knowing what something is when presented in a specific sensory modality such as vision, hearing, or somatosensory. Agnosias are caused by disconnection from or damage to the unimodal association cortex for that sensory modality.

Case 6-1

A 60-year-old right-handed woman presented with several months of gradually worsening problems with completing tasks at home and at work. Despite normal strength and preserved fine finger motion, on examination she was unable to pantomime tool use with either hand, often using her fingers as if they were the tools themselves. Brain imaging revealed a meningioma with mass effect and edema on the left parietal lobe.

Comment. The patient described in this case illustrates that a focal lesion of dominant heteromodal parietal cortex can lead to ideomotor apraxia of both hands. Note that this occurred in the absence of any more basic weakness or loss of corticospinal function.

TABLE 6-2 Types of Apraxias

Type of Apraxia	Manifestation	Assessment	Localization
Ideational apraxia	Impaired executing sequence of overlearned actions	Ask patient to report the steps in an activity (eg, how to make a peanut butter and jelly sandwich)	Dominant posterior parietal cortex
Conceptual apraxia	Impaired knowledge/ semantic memory of tool use/mechanical advantage	Ask patient to choose from a series of objects that can best perform a job even if not made for it (eg, a butter knife to put in a screw)	Dominant posterior parietal cortex
Transitive ideomotor apraxia	Inability to perform overlearned fine motor tasks, tool use	Have patient pantomime tool use on command (eg, show how to hold and use a pair of scissors)	Dominant parietal cortex/ supplemental motor cortex/corpus callosum
Intransitive ideomotor apraxia	Inability to perform overlearned fine motor tasks, hand gestures	Have patient make hand gestures (eg, salute)	Dominant parietal cortex/ supplemental motor cortex/corpus callosum
Orofacial apraxia	Inability to make skilled mouth movements	Ask patient to pantomime mouth use (eg, blow out a match)	Inferior frontal lobe/basal ganglia
Apraxia of speech	Impaired speech sound formation	Ask patient to speak (eg, say a few sentences)	Broca area/dominant supplemental motor cortex
Limb-kinetic apraxia	Impaired distal limb movement	Ask patient to make simple fine movements (eg, rotate a coin with thumb and forefinger)	Supplemental motor cortex/motor cortex
Constructional apraxia	Impaired drawing and copying	Have patient draw and copy items (eg, copy intersecting pentagons)	Nondominant parietal lobe/dominant parietal lobe/frontal lobe

and unusual the sudden acquisition of one of these disorders can be to the patient.

Auditory association cortex damage can result in auditory agnosia (inability to identify things by sound), pure word deafness (inability to perceive the spoken word), and receptive amusia (inability to recognize music).[9] Similarly, somatosensory association cortex damage causes somatosensory agnosias including astereognosia (inability to identify things by how they feel in the hand) and agraphesthesia (inability to identify shapes and symbols when traced on the skin). Common causes of damage in these association areas include strokes, tumors, and neurodegenerative diseases. Corticobasal syndrome is a neurodegenerative disease well known to affect the unimodal and heteromodal parietal cortices. Corticobasal syndrome (sometimes referred to as corticobasal ganglionic degeneration or Rebeiz syndrome),[10] consists of a rigid-akinetic hemiparkinsonism and myoclonus, as well as unilateral ideomotor apraxia, agraphesthesia, astereognosis,

Case 6-2

A 68-year-old right-handed man with a history of hypercholesterolemia, hypertension, and atrial fibrillation presented with the inability to recognize his wife. He reported that she sounded like his wife and acted like his wife, but that he couldn't tell her apart from other women on sight. On further testing, he was unable to recognize faces of famous people or faces of people he knew well. Brain MRI showed an old wedge-shaped infarction in the left frontal lobe, another old wedge-shaped infarction in the left temporal-occipital area, and one acute infarction on diffusion-weighted imaging in the right temporal-occipital area. Anticoagulation was started for his atrial fibrillation with cardioembolic stroke.

Comment. This case illustrates an example of acutely acquired prosopagnosia from one or more cerebral infarctions of the inferior occipitotemporal cortex (fusiform gyrus). The patient has lost the ability to identify faces, which can be quite frightening and debilitating for the patient. While cases occurring due to a single lesion of the nondominant fusiform gyrus have been reported, prosopagnosia is even more likely to occur with bilateral lesions, and the lesions need not occur at the same time, with the syndrome appearing abruptly, as in this case, after the acute infarction of the remaining fusiform gyrus.

and even alien limb syndrome. Case 6-3 highlights this syndrome.

HEMINEGLECT

While the nondominant parietal cortex mediates holistic multimodal attention and awareness to both hemispaces as well as intention of action in space (directing action toward one side of space),[11,12] the dominant parietal cortex manages attention only to the contralateral (usually right) hemispace.[13] The nondominant parietal cortex is so critical to holistic (both hemifields) visuospatial attention that even unimpaired young people will bisect a long horizontal

Case 6-3

A 55-year-old right-handed woman presented with a 1-year history of problems completing tasks such as tying her shoes or making a sandwich. On examination, her primary sensory testing (light touch, vibratory, and pain and temperature sensations) were all intact, but she was unable to make out letters written in the palm of her left hand and could not identify objects placed in her left hand. Her left upper extremity was also quite rigid, and she could not pantomime tool usage on command. Brain imaging showed atrophy of one hemisphere more than the other and no evidence of vascular damage. The patient was diagnosed with corticobasal syndrome.

Comment. Corticobasal syndrome is most often due to a tauopathy, although there are some cases with syndromic features that turn out to have underlying Alzheimer disease pathology. The classic syndrome includes disconnection between the dominant and nondominant hemispheres that results in the nondominant hand having ideomotor apraxia, although cases that have bilateral ideomotor apraxia are common. This case highlights a commonly occurring initial presentation for this syndrome as well as the fact that ideomotor apraxias can be encountered in forms that impair functionality in activities of daily living.

KEY POINTS

- The nondominant hemisphere parietal cortex is ''dominant'' for visuospatial attention and awareness, and lesions of this region lead classically to loss of awareness of the left side of space (left hemispatial neglect).
- Balint syndrome is caused by a number of different etiologies impacting the posterior parietal regions, particularly bilaterally. Core features of the syndrome include only being able to perceive one feature at a time (simultanagnosia), inability to control reaching out via vision (optic ataxia), and breakdown of deliberate movement of gaze while preserving basic extraocular movement (ocular apraxia, also referred to as optic apraxia and psychic paralysis of gaze).

line a few millimeters to the left of the center, highlighting the fact that the nondominant hemisphere is so dominant for spatial tasks that it skews our perception of space left of center. Therefore, damage to the visuospatial attention system can result in hemispatial neglect, most classically involving the left side of space, where patients ''lose'' that whole half of space. Since both hemispheres attend to the right hemispace, right hemineglect is very unusual. As the damage that causes hemineglect occurs to the heteromodal association cortex of the parietal lobe, the hemispatial neglect often impacts multiple sensory modalities, including visual, auditory, and somatosensory. Absence of knowledge of the deficit (anosognosia) is a common part of hemispatial neglect, making rehabilitation difficult.[14] While hemispatial neglect from an acute stroke often seems to fade quickly, lingering subtle deficits can occur in these cases that can be more disabling than similar-sized dominant hemisphere lesions.[15] **Case 6-4** highlights the lack of insight and impairment this syndrome can cause.

BALINT SYNDROME

Balint syndrome is a classic syndrome of acquired perceptual-motor dysfunction affecting the heteromodal cortex, which was first described by the Hungarian physician Rezső Bálint in 1909.[16] This syndrome is composed of simultanagnosia, optic apraxia, and optic (or ocular) ataxia. This condition was originally referred to as psychic paralysis of gaze. Optic ataxia and optic apraxia can occur separately from the complete Balint syndrome, but all of these dysfunctions are thought to involve damage to the dorsal occipitoparietal "where" system. Balint syndrome can be caused by multiple etiologies and has a number of variations.[17] Simultanagnosia is a fascinating phenomenon where the patient can only perceive one item or

Case 6-4

A 75-year-old right-handed woman presented to the emergency department after being pulled over by police for erratic driving and veering over into the opposite lane, after which she was unable to walk a straight line. On examination the patient had a gaze preference to the right, did not respond to commands from examiners when they stood on the left but did respond to examiners who stood on the right. She bisected a line almost all the way to the right end of the line and could not cancel letters on the left of a sheet of paper, on which multiple letters had been written throughout. Brain MRI revealed a right parietal lobar hemorrhage and hemosiderin deposits elsewhere, suggesting cerebral amyloid angiopathy as the cause of her brain hemorrhage.

Comment. The anosognosia seen in this heteromodal cortical deficit, hemispatial neglect, demonstrates both how counterintuitive this phenomenon can be as well as how disabling it can be to the unaware patient. This type of case also shatters several of our common-sense concepts of the human mind as being unitary and functioning primarily through logic. A patient with a dense hemispatial neglect cannot show awareness of the left side of space, or even knowledge of the left side of space, with no awareness that something is wrong. This incorrect belief cannot be dispelled by explanation to the patient or leading the patient through the logic that there must be a left side. It can be devastating for a patient to only possess awareness of the right side of space and have no appreciation that anything else is missing.

feature at a time, having lost the ability to perceive the holistic gestalt of what the senses present.[18] If two items are presented, only one will be appreciated. If a battle scene is shown, the patient can only see a tank.[19]

Optic ataxia is a failure of vision to be able to guide complex voluntary movement, specifically in reaching.[20] When optic ataxia occurs separately from the complete Balint syndrome it is thought to localize damage to the posterior parietal lobe of either side and, if unilateral, causes the deficit only on the contralesional side.[21] Cavina-Pratesi and Milner[22] provide an elegant case for the occurrence of reach-specific unilateral optic ataxia (an ataxic problem specific to visually guided movement of the hands toward an object, as in grasping) for any limb directed to the contralesional side, although other research seems to implicate at least some limb-specific parts of the posterior parietal cortex for this task.[23] This can be tested for by having the patient reach for objects both on the same side and opposite side of each hand using only vision to guide them. Ocular apraxia (psychic paralysis of gaze) refers to an inability to control the movements of the eyes either voluntarily or on command, although the eyes can move spontaneously in all quadrants.[24] Asking the patient to direct gaze to the various quadrants of space should elicit this finding. This difficulty in movement is not to one specific side, unlike either forced gaze deviation (from a frontal eye field lesion) or hemispatial neglect. Additionally, patients with Balint syndrome can perform single-letter reading but not word reading and, thus, experience a type of alexia, but for nonlanguage reasons, as this can be thought of as yet another manifestation of simultanagnosia. The complete Balint syndrome is typically the result of bilateral lesions in the posterior parietal regions. Very rarely, a unilateral nondominant posterior parietal lobe can cause this syndrome.[25] **Case 6-5** emphasizes the importance of bilateral parietal lobe damage to the emergence of most cases of Balint syndrome, even when the lesioning is separated in time. A number of conditions can cause Balint syndrome, including cerebrovascular disease, although neurodegenerative conditions such as either the posterior cortical atrophy variant of Alzheimer disease or corticobasal syndrome can also manifest the features of the Balint syndrome.[26] Other etiologies can include trauma, autoimmune disease, neoplasia, and infection. Posterior reversible encephalopathy syndrome can also manifest as Balint syndrome. Evaluation of Balint syndrome typically begins with neuroimaging, with MRI being the most reliable for the range of etiologies typical for this phenomenon. Treatment is focused on management of the underlying etiology, patient and family education, and occupational therapy.[27]

PERCEPTUAL-MOTOR IMPAIRMENT AND THE BASAL GANGLIA

Parkinson Disease

While the motor dysfunction seen in Parkinson disease is well known among physicians, awareness still lags regarding the higher, perceptual-motor disabilities that occur in this condition. The basal ganglia have been directly implicated in implicit motor learning, which is believed to decrease with age, but is even more profoundly disordered in Parkinson disease.[28] There are conflicting reports as to whether this is independent of the mesial-temporal lobe memory system.[29] Parkinson disease causes deficits in the key perceptual-motor components of sequencing and predictive voluntary movement.[30] More recently, patients with Parkinson disease have been found to have deficits in hand-eye coordination and predictive feedforward control of

KEY POINT

- Patients with Parkinson disease demonstrate problems with implicit motor learning, hand-eye coordination, and prediction of movements. While the basal ganglia are believed to mediate these functions and be the site of damage in Parkinson disease, these problems do not improve with dopaminergic treatment, unlike other symptoms of Parkinson disease.

KEY POINT

- Many developmental disorders include elements of perceptual-motor dysfunction. One common syndrome associated with this is developmental coordination disorder, which has a significant co-occurrence and overlap with other common and important developmental disorders including attention deficit hyperactivity disorder and autism spectrum disorder. Rehabilitation therapies do help performance.

Case 6-5

A 57-year-old right-handed man with a history of hypertension and hypercholesterolemia presented with a 1-day history of problems with vision, explaining that ''things were looking weird.'' The patient described people looking distorted on one side of their body and stated that when he looked in a mirror, he saw an image of himself that looked like a photo negative. He also stated that he had difficulty reading. His neurologic examination was significant for a left homonymous hemianopia and difficulties moving his eyes properly on command, although the patient was observed to have full range of spontaneous eye movement. He was unable to identify more than one object at a time even when presented in his good visual field. He also was only able to make out single items in a large scene. He often had difficulties reaching out to targets, but with his eyes closed he was able to touch his nose bilaterally. He was able to read single letters but not complete words. Brain MRI revealed an old, previously unknown infarction in the left parietal lobe and an acute hemorrhage deep in the right parietal and occipital region.

Comment. While this case had several additional features localizable to the secondary visual cortex (part of the unimodal visual cortex), this patient also presented with the classic features of Balint syndrome. Balint syndrome, as shown in the case, can occur in conjuncture with other symptoms depending on the extent of damage caused by the precipitating etiology. However, Balint syndrome itself is enough to cause disability in a number of simple to complex activities despite preservation of elemental unimodal perception and action. Heteromodal modeling and guidance of action is necessary to be as functional as possible in the real world. These sorts of deficits are often missed or confused with other more common types of neurologic problems, which can lead to confusion in both the practitioner and the patient as to what is wrong and how best to improve function in patients afflicted with this syndrome.

movement, again suggesting an important role for the basal ganglia in these activities; however, these patients did not experience improvement in this domain when on dopaminergic medications, although their other symptoms of Parkinson disease improved.[31] **Case 6-6** illustrates this dichotomy in treatment effect.

PERCEPTUAL-MOTOR DYSFUNCTION AND DEVELOPMENT

Clumsiness has often been noted as a common term for a range of perceptual-motor developmental dysfunctions, particularly in hand-eye coordination. While its causes are yet uncertain and its localizations still being delineated, it is suspected to include parietal lobes, the extrapyramidal system (especially basal ganglia), and premotor frontal cortex. Patients with developmental coordination disorder, which has been typically defined as simply being clumsier than the average person, have been shown to perform normally for the simplest motor tasks but lag in speed and accuracy for more complex tasks.[32] Interestingly, these patients also have problems with motor imagery, but again only when moving beyond simple tasks.[33] Developmental coordination disorder has a high rate of overlap with attention deficit hyperactivity disorder and autism spectrum disorders.[34] **Case 6-7** demonstrates this complicated overlap. Rehabilitation programs including core stability exercises and task-oriented motor training

Case 6-6

A 65-year-old right-handed man presented with shaking in his right hand at rest and with action and a feeling as if his limbs were dragging. These symptoms had started gradually and had been getting progressively worse over the past year. His history was significant only for hypertension and hypercholesterolemia, treated with lisinopril and atorvastatin. He had no exposure to illicit drugs and no exposure to pesticides. Neurologic examination was remarkable for increased tone at the neck, cogwheel rigidity in the right arm, and a pill-rolling resting tremor of the right hand. He had mild slowness of movements, with fine finger motion slower on the right than the left. Gait was slightly stooped with small steps and squared turns. The patient improved significantly after initiation of dopaminergic therapy. While the patient was pleased with the improvement, he still did not return to his previous level of function in more complex motor activities like golf and tennis.

Comment. This case highlights the need for complementary treatment in addition to dopaminergic therapy in patients with Parkinson disease, for example, by the use of cognitive therapy to address the cognitive aspects of Parkinson disease, not just the dopaminergic deficits.

have been shown to improve performance in these patients.

REHABILITATION FOR PERCEPTUAL-MOTOR DYSFUNCTION

Rehabilitation for patients with perceptual-motor dysfunction can be challenging. There may be a role for more nontraditional modes of rehabilitation in the improvement of these disorders. For example, there is evidence that covering the right hemifield of vision in patients with left hemispatial neglect can lead to improvement in function.[35,36] Also, a study found that healthy older individuals who participated in tai chi had better hand-eye reaction times than those who did not in a finger-pointing task.[37] Similarly, training on computerized game systems (mostly the Wii system) improved hand-eye coordination for the nondominant

Case 6-7

A 19-year-old left-handed woman presented with a life-long history of mild motor developmental delay, severe clumsiness, and impulsivity. As a child she was considered bright, but she had difficulty with writing, both in speed and clarity. She was the result of a normal pregnancy and delivery and had no significant past medical history. There was a family history of clumsiness from both of her parents, but nothing considered disabling. Neurologic examination was within normal limits except for hyporeflexia. Neuropsychological testing showed attentional deficits on multiple measures. Neuroimaging showed no significant abnormalities. The combination of intensive rehabilitative therapy, including sensory-motor integration and core-strengthening exercises, plus initiation of therapy with methylphenidate, led to greatly improved performance.

Comment. This case shows the common co-occurrence of perceptual-motor problems with attentional problems in developmental disorders and the utility of multimodal, multitargeted treatment to optimize function.

KEY POINT

- Rehabilitation can help improve perceptual-motor function, although it may take new approaches and out-of-the-box thinking to move forward in treatment of this type of deficit.

hand for laparoscopic surgeons[38] and, more to the point, improved balance problems for children with developmental coordination disorder.[39]

CONCLUSION

Disorders of perceptual recognition and knowledge are localized to the unimodal association cortex adjacent to the primary sensory cortex for that sensory modality. Dominant posterior parietal lesions often result in apraxia, with ideomotor apraxia presenting as the classic syndrome. Disorders of perceptual integration for use in actions are mostly located in the heteromodal parietal cortices and associated networks. Balint syndrome serves as one example of perceptual integrative failure and the failure of vision to mediate action, specifically reach and gaze. Left hemispatial neglect is a loss of perception or attention to the left side of space due to nondominant parietal cortex dysfunction. Patients with Parkinson disease provide an example of patients with deficits in motor sequencing and prediction that likely involves the basal ganglia but not necessarily the dopaminergic system. Developmental disorders such as developmental coordination disorder are examples of more diffuse system deficits in perceptual-motor skills. The use of rehabilitation modalities of therapy including nontraditional techniques and technologies may provide opportunities for improving the lives of patients experiencing perceptual-motor dysfunction.

REFERENCES

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. Washington, DC: American Psychiatric Association, 2013.
2. Petreska B, Adriani M, Blanke O, Billard AG. Apraxia: a review. Prog Brain Res 2007;164:61–83.
3. Heilman KM. Apraxia. Continuum (Minneap Minn) 2010;16(4 Behavioral Neurology):86–98. doi:10.1212/01.CON.0000368262.53662.08.
4. Bieńkiewicz MM, Brandi ML, Goldenberg G, et al. The tool in the brain: apraxia in ADL. Front Psychol 2014;5:353. doi:10.3389/fpsyg.2014.00353.
5. Riddoch MJ, Humphreys GW. Visual agnosia. Neurol Clin 2003;21(2):501–520.
6. Swanson WH, Cohen JM. Color vision. Ophthalmol Clin North Am 2003;16(2):179–203.
7. Rossion B. Understanding face perception by means of prosopagnosia and neuroimaging. Front Biosci (Elite Ed) 2014;6:258–307.
8. Starrfelt R, Olafsdottir RR, Arendt IM. Rehabilitation of pure alexia: a review. Neuropsychol Rehabil 2013;23(5):755–779. doi:10.1080/09602011.2013.809661.
9. Vignolo LA. Music agnosia and auditory agnosia. Dissociation in stroke patients. Ann N Y Acad Sci 2003;999:50–57.
10. Rebeiz JJ, Kolodny EH, Richardson EP Jr. Corticodentatonigral degeneration with neuronal achromasia: a progressive disorder of late adult life. Trans Am Neurol Assoc 1967;92:23–26.
11. Na DL, Adair JC, Williamson DJ, et al. Dissociation of sensory-attentional from motor-intentional neglect. J Neurol Neurosurg Psychiatry 1998;64(3):331–338.
12. Sampanis DS, Riddoch J. Motor neglect and future directions for research. Front Hum Neurosci 2013;7:110. doi:10.3389/fnhum.2013.00110.
13. Heilman KM, Watson RT, Valenstein E. Neglect and related disorders. In: Clinical neuropsychology. New York: Oxford University Press, 1985.
14. Vallar G, Ronchi R. Anosognosia for motor and sensory deficits after unilateral brain damage: a review. Restor Neurol Neurosci 2006;24(4–6):247–257.
15. Hier DB, Mondlock J, Caplan LR. Recovery of behavioral abnormalities after right hemispheric stroke. Neurology 1983;33(3):345–350.
16. Balint R. Seelenlähmung des ''Schauens,'' optische Ataxie, räumliche Störung der Aufmerksamkeit. Monatschr Psychiat Neurol 1909;25:51–81.
17. Chechlacz M, Humphreys GW. The enigma of Bálint's syndrome: neural substrates and cognitive deficits. Front Hum Neurosci 2014;8:123. doi:10.3389/fnhum.2014.00123.
18. Mevorach C, Shalev L, Green RJ, et al. Hierarchical processing in Balint's syndrome: a failure of flexible top-down attention. Front Hum Neurosci 2014;8:113. doi:10.3389/fnhum.2014.00113.
19. Shakespeare TJ, Yong KX, Frost C, et al. Scene perception in posterior cortical atrophy: categorization, description and fixation patterns. Front Hum Neurosci 2013;7:621. doi:10.3389/fnhum.2013.00621.

20. Andersen RA, Andersen KN, Hwang EJ, Hauschild M. Optic ataxia: from Balint's syndrome to the parietal reach region. Neuron 2014;81(5):967–983. doi:10.1016/j.neuron.2014.02.025.

21. Meek BP, Shelton P, Marotta JJ. Posterior cortical atrophy: visuomotor deficits in reaching and grasping. Front Hum Neurosci 2013;7:294. doi:10.3389/fnhum.2013.00294.

22. Cavina-Pratesi C, Connolly JD, Milner AD. Optic ataxia as a model to investigate the role of the posterior parietal cortex in visually guided action: evidence from studies of patient M.H. Front Hum Neurosci 2013;7:336. doi:10.3389/fnhum.2013.00336.

23. Yttri EA, Wang C, Liu Y, Snyder LH. The parietal reach region is limb specific and not involved in eye-hand coordination. J Neurophysiol 2014;111(3):520–532. doi:10.1152/jn.00058.2013.

24. Ptak R, Müri RM. The parietal cortex and saccade planning: lessons from human lesion studies. Front Hum Neurosci 2013;7:254. doi:10.3389/fnhum.2013.00254.

25. Kas A, de Souza LC, Samri D, et al. Neural correlates of cognitive impairment in posterior cortical atrophy. Brain 2011;134 (pt 5):1464–1478. doi:10.1093/brain/awr055.

26. Rajagopal R, Bateman R, Van Stavern GP. Visual involvement in corticobasal syndrome. J Neuroophthalmol 2012;32(4):338–340. doi:10.1097/WNO.0b013e3182305162.

27. Zgaljardic DJ, Yancy S, Levinson J, et al. Balint's syndrome and post-acute brain injury rehabilitation: a case report. Brain Inj 2011;25(9): 909–917. doi:10.3109/02699052.2011.585506.

28. Gobel EW, Blomeke K, Zadikoff C, et al. Implicit perceptual-motor skill learning in mild cognitive impairment and Parkinson's disease. Neuropsychology 2013;27(3): 314–321. doi:10.1037/a0032305.

29. Schendan HE, Tinaz S, Maher SM, Stern CE. Frontostriatal and mediotemporal lobe contributions to implicit higher-order spatial sequence learning declines in aging and Parkinson's disease. Behav Neurosci 2013;127(2):204–221. doi:10.1037/a0032012.

30. Stern Y, Mayeux R, Rosen J, Ilson J. Perceptual motor dysfunction in Parkinson's disease: a deficit in sequential and predictive voluntary movement. J Neurol Neurosurg Psychiatry 1983;46(2):145–151.

31. Lukos JR, Snider J, Hernandez ME, et al. Parkinson's disease patients show impaired corrective grasp control and eye-hand coupling when reaching to grasp virtual objects. Neuroscience 2013;254:205–221. doi:10.1016/j.neuroscience.2013.09.026.

32. Cantin N, Ryan J, Polatajko HJ. Impact of task difficulty and motor ability on visual-motor task performance of children with and without developmental coordination disorder. Hum Mov Sci 2014;34:217–232. doi:10.1016/j.humov.2014.02.006.

33. Noten M, Wilson P, Ruddock S, Steenbergen B. Mild impairments of motor imagery skills in children with DCD. Res Dev Disabil 2014;35(5):1152–1159. doi:10.1016/j.ridd.2014.01.026.

34. Gillberg C, Kadesjö B. Why bother about clumsiness? The implications of having developmental coordination disorder (DCD). Neural Plast 2003;10(1–2):59–68.

35. Smania N, Fonte C, Picelli A, et al. Effect of eye patching in rehabilitation of hemispatial neglect. Front Hum Neurosci 2013;7:527. doi:10.3389/fnhum.2013.00527.

36. Barrett AM, Buxbaum LJ, Coslett HB, et al. Cognitive rehabilitation interventions for neglect and related disorders: moving from bench to bedside in stroke patients. J Cogn Neurosci 2006;18(7):1223–1236.

37. Tsang WW, Kwok JC, Hui-Chan CW. Effects of aging and tai chi on a finger-pointing task with a choice paradigm. Evid Based Complement Alternat Med 2013;2013:653437. doi:10.1155/2013/653437.

38. Middleton KK, Hamilton T, Tsai PC, et al. Improved nondominant hand performance on a laparoscopic virtual reality simulator after playing the Nintendo Wii. Surg Endosc 2013;27(11):4224–4231. doi:10.1007/s00464-013-3027-z.

39. Jelsma D, Geuze RH, Mombarg R, Smits-Engelsman BC. The impact of Wii Fit intervention on dynamic balance control in children with probable developmental coordination disorder and balance problems. Hum Mov Sci 2014;33:404–418. doi:10.1016/j.humov.2013.12.007.