



Neurocase

The Neural Basis of Cognition

ISSN: 1355-4794 (Print) 1465-3656 (Online) Journal homepage: <http://www.tandfonline.com/loi/nncs20>

Callosal apraxia: a 34-year follow-up study

Adam D. Falchook, Robert T. Watson & Kenneth M. Heilman

To cite this article: Adam D. Falchook, Robert T. Watson & Kenneth M. Heilman (2016): Callosal apraxia: a 34-year follow-up study, *Neurocase*, DOI: [10.1080/13554794.2016.1148743](https://doi.org/10.1080/13554794.2016.1148743)

To link to this article: <http://dx.doi.org/10.1080/13554794.2016.1148743>



Published online: 29 Feb 2016.



Submit your article to this journal [↗](#)



Article views: 2



View related articles [↗](#)



View Crossmark data [↗](#)

Callosal apraxia: a 34-year follow-up study

Adam D. Falchook^{a,b}, Robert T. Watson^c and Kenneth M. Heilman^{a,b}

^aDepartment of Neurology and Center for Neuropsychological Studies, University of Florida College of Medicine, Gainesville, FL, USA; ^bMalcom Randall Veteran Affairs Medical Center, Gainesville, FL, USA; ^cBrain Rehabilitation Research Center and Geriatric Research Education and Clinical Center, Department of Clinical Sciences, Florida State University College of Medicine, Tallahassee, FL, USA

ABSTRACT

Loss of ability of the left upper limb (LUL) to correctly produce spatial and temporal components of skilled purposeful movements was reported 34 years ago in a woman with a callosal infarction. To learn about recovery, we recently reexamined this woman. This woman was tested for ideomotor apraxia by asking her to pantomime to command and to seeing pictures of tools. Whereas she performed normally with her right upper limb, her LUL remained severely apraxic, making many spatial (postural and movement) errors. Initially, she did not reveal loss of finger–hand dexterity (limb-kinetic apraxia), and when tested again with the coin rotation task, her left hand performance was normal. Without vision, she could name objects placed in her left hand but not name numbers written in this hand. Since this woman had a callosal lesion, failure to recover cannot be accounted for by left hemisphere inhibition of her right hemisphere. Although failure for her LUL to improve may have been related to not using her LUL for skilled actions, her right hemisphere was able to observe transitive actions, and this failure of her LUL to produce skilled purposeful movements suggests her right hemisphere may have not had the capacity to learn these movement representations. Without vision, her ability to recognize objects with her left hand, but not numbers written on her left palm, suggests graphesthesia may require that her left hand did not have access to movement representations important for programming these numbers when writing.

ARTICLE HISTORY

Received 8 November 2015
Accepted 25 January 2016

KEYWORDS

Corpus callosum; ideomotor apraxia; limb kinetic apraxia; agraphia; agraphesthesia

Introduction

Ideomotor apraxia (IMA), as defined by Liepmann (1920), is a loss of ability to correctly produce skilled purposeful movements as necessary for tool use, pantomimes, and communicative gestures. When attempting to perform purposeful movement, individuals with IMA make spatial and temporal errors. In regard to the spatial errors, some of the most common errors made by these individuals are postural errors, for example, using part of their body as the tool or implement (Goodglass & Kaplan, 1963). When not using their body part as an object, individuals with IMA will often not be able to correctly position their fingers and hand. In addition to postural errors, individuals with IMA often make egocentric errors, such as moving the incorrect joint or incorrectly coordinating multiple joint movements, and also make allocentric movement errors (Poizner, Mack, Verfaellie, Rothi, & Heilman, 1990; Rothi, Mack, Verfaellie, Brown, & Heilman, 1988).

Limb kinetic apraxia (LKA), as defined by Liepmann (1920), is a loss of ability to produce precise, independent, and coordinated movements of the hand and fingers. Left hemisphere damage can induce bilateral IMA (Liepmann, 1920) as well as bilateral LKA (Hanna-Pladdy, Mendoza, Apostolos, & Heilman, 2002). The corpus callosum is the major structure supporting interhemispheric communication. Damage to the corpus callosum has been reported to induce several apraxic disorders including verbal

dissociation apraxia, where a patient cannot correctly perform to verbal commands using their left upper limb, but can imitate and use actual tools with their left upper limb (Geschwind & Kaplan, 1962). Callosal damage in a patient has also been reported to cause an LKA of the left hand (Acosta, Bennett, & Heilman, 2014). Liepmann and Mass (1907) reported a patient with a callosal lesion who had an IMA of his left upper limb. Unfortunately, this patient had a prior lesion of his pons with a right hemiparesis as well as a lesion of the medial left frontal lobe. Thus, Liepmann and Mass could not test this patient's right upper extremity and it is also possible that the left medial frontal lesion could have been inducing this patient's apraxia (Watson, Fleet, Rothi, & Heilman, 1986).

In 1983, Watson and Heilman reported a woman with an infarction that was limited to her corpus callosum who had an IMA of her left upper limb but not her right upper limb. Subsequently, other investigators such as Graff-Radford, Welsh, and Godersky (1987) were able to replicate these findings. Currently, it is not known how the brain networks that mediate purposeful skilled movements may reorganize during the process of recovery from callosal disconnection without damage to either cerebral hemisphere. We recently had the opportunity to reexamine the same woman with apraxia of the left hand from a callosal lesion that occurred 34 years ago, as was reported by Watson and Heilman (1983). This examination, reported here, allowed us to learn the type of recovery

from this neurological injury that occurred more than three decades ago.

Case report

Our patient is currently a 77-year-old right-handed woman. We will briefly summarize her medical history, previously reported by Watson and Heilman (1983). At the age of 43, she suddenly developed a severe headache, vomiting, mutism, and inability to stand. On admission to the hospital at that time, she had transcortical motor aphasia, apraxia of her left hand, and bilateral lower extremity weakness that was greater on the right than left. CT scan showed a hemorrhage above the corpus callosum, no aneurysm was seen on angiography, and bilateral vasospasm of the anterior cerebral arteries was present. During the first two weeks in the hospital, she demonstrated hypokinesia, persistent transcortical motor aphasia, and a complete inability to perform any gestures or use tools with the left hand although performance with her right hand was normal. She was also not able to write with her left hand. She demonstrated a tactile anomia of the left hand since she was able to identify objects placed in the left hand by pointing to them from an array of choices even though she was unable to name them. Her ability to perform deft movement with the left hand and fingers remained intact. A repeat CT scan showed infarction of the anterior two-thirds of her corpus callosum without any evidence of cortical damage. Magnetic resonance imaging of the callosal lesion subsequently confirmed these findings (Watson, Heilman, & Bowers, 1985).

By the fourth week after the intracranial hemorrhage, our patient's tactile anomia resolved. She continued to demonstrate an IMA of her left upper extremity. Although now her gestures were recognizable, she continued exhibiting spatial and temporal errors. There was some improvement with actual tool use with the left hand, though spatial and temporal errors persisted. At this time, the patient also demonstrated diagonal dyspraxia. For example, she once put her left and right arms into the sleeves of two different blouses at the same time. By the second month after the intracranial hemorrhage, her language fluency returned to normal. She demonstrated apraxic agraphia with her left hand but could type with her left hand. When copying a cube with her *right* hand, her copy had visuospatial errors. She continued to demonstrate IMA of her left hand, although proximal movements with the left arm were performed normally, and there was some improvement with actual tool use though spatial and temporal errors persisted.

Our patient and her family have remained in contact with her neurologist (RTW), and they had recently discussed a repeat evaluation to learn whether signs of interhemispheric disconnection have changed or remained. During the 34 years since the subarachnoid hemorrhage and callosal infarction, the patient and her husband reported she has had no new neurological symptoms or signs. She had worked as a teacher's assistant before the intracranial hemorrhage and returned to work as a cashier for 13 years after the intracranial hemorrhage. Her husband always managed their finances. She was not able to cook again after the intracranial hemorrhage.

She reports reduced power in the left arm and leg since the intracranial hemorrhage though she is able to walk 7–8 miles every day. The patient and her husband report she has had memory problems since the hemorrhage and she does not remember many of her life events that occurred before the hemorrhage. At a class reunion 8 years ago, she did not remember half the people or events. She and her husband report that her memory has gotten worse over the past few years. She has had lifelong difficulty spelling, though she always enjoyed reading and still reads books.

On exam, our patient was oriented to place and time, but she was unable to name the current president. She had a forwards digit span of 7, 3/3 word registration, and 1/3 word recall. To reevaluate ideomotor praxis, we asked her to pantomime the use of 16 common tools and implements in response to verbal command. Each hand was tested separately such that for half the items the left hand was tested before the right and for half the items the right hand was tested before the left. We also asked her to perform six intransitive (communicative) gestures with each hand. She performed 15/16 transitive gestures and 6/6 intransitive gestures normally with her right upper limb. In contrast, only 2/16 transitive gestures and 2/6 intransitive gestures were performed correctly with her left hand. Her inability to correctly perform gestures with the left hand with normal performance with her right hand is consistent with the predicted effects of a disconnection between the praxis networks in the left hemisphere and brain regions in the right hemisphere that control movements of the left hand.

As mentioned, Geschwind and Kaplan (1962) reported that a callosal disconnection between the left hemispheric networks important for language comprehension and brain regions in the right hemisphere that control movement of the left hand may also induce an inability to perform pantomimes and gestures with the left hand, such that their patient was unable to perform pantomimes to verbal command with the left hand, though he was able to imitate gestures with the left hand. To learn whether our patient's frequent left hand–arm apraxic errors in response to command were being produced by a language–motor disconnection, we wanted to avoid the potential effects of language disconnection during assessment of right hemisphere praxis networks. Therefore, we showed our patient pictures of tools and implements and asked her to pantomime the use of the items. These were the same 16 items our patient was previously asked to pantomime in response to verbal command. Pictures were selected from the Snodgrass and Vanderwart (1980) picture set. Performance of the gestures in response to pictures of the tools and implements with the left hand (2/16 correct) was no better than performance in response to verbal command. Performance of the gestures in response to pictures of the tools and implements with the right hand was normal (16/16 correct). Ability to imitate pantomimes and gestures was not tested during the recent evaluation. However, when Watson and Heilman (1983) initially evaluated this woman, she was unable to correctly perform pantomimes with the left hand–arm in response to imitation. Thus, then and now the apraxia of her left upper limb could not be attributed solely to a disconnection between her left

hemisphere language network and her right hemisphere, but she demonstrated evidence of a lasting disconnection between the left hemisphere praxis network and her right hemisphere.

Whereas the left hemisphere is dominant for ideomotor praxis, the ability to program the spatial and temporal components of learned purposeful skilled movements, in some people the left hemisphere may also be dominant for the ability to perform precise, independent, and coordinated finger movements. For example, in addition to left hemisphere damage being able to induce bilateral IMA, left hemisphere damage can also induce bilateral LKA (Hanna-Pladdy et al., 2002). Furthermore, Acosta et al. (2014) recently reported a man who developed IMA and LKA of the left hand after a lesion of the corpus callosum. However, when our patient was initially evaluated by Watson and Heilman (1983), shortly after she infarcted her corpus callosum, it was noted that her dexterity with the left hand was normal. When we reevaluated our patient, we asked her to perform the coin rotation test, a test for LKA (Hanna-Pladdy et al., 2002). During this task, an individual is requested to rotate a nickel between their thumb, index, and middle fingers as rapidly as possible and the number of 180° rotations in a 10 s period is counted. Our patient performed five trials with each hand, and performance was normal with no evidence of LKA with either hand (right hand: 11, 14, 11, 12, 14; left hand: 6, 12, 12, 12, 11).

When an object she was unable to see was placed in our patient's left hand, her right hemisphere was able to recognize the object, transmit this information to her left hemisphere, and she was then able to point, with the right hand, to the picture of the corresponding object (6/8 correct, with difficulty discriminating between a nail and a screw). She was also able to perform this task when the object was placed in her right hand and she responded by pointing with the left hand (8/8 correct). For this task, pictures of the eight objects were selected from the Snodgrass and Vanderwart (1980) picture set, and these line drawings were printed on a single 8.5 × 11 in. sheet of paper. When an object was placed in our patient's left hand, she could also point with her right hand to the corresponding tool that could act on the object (6/8 correct, with difficulty discriminating between a nail and a screw). Performance was the same when the objects were placed in our patient's right hand and she was asked to point to the corresponding tool with the left hand. Our patient was also able to name objects placed in her left hand (14/16 correct, called a nail "a screw" and called an envelope "paper"). Performance was the same when she named objects placed in the right hand.

Finally, our patient was unable to write any words when holding a pen with the left hand, even though she was able to spell most of these words aloud. When asked to write individual letters and numbers with the left hand, 5 of the 9 numbers and 4 of the 12 letters that she printed were not recognizable. Our patient demonstrated excellent handwriting when she wrote words, letters, and numbers with the right hand. When our patient was asked to close her eyes and the examiner traced numbers in the palm of her right hand with a closed pen, she was able to identify eight of the nine numbers traced in this palm but only four of the nine numbers traced in the palm of the left hand.

Discussion

In adults with typical hemispheric specialization, the left hemisphere is dominant for language and praxis. A functional magnetic resonance imaging study of neurologically intact adults has demonstrated activation of a left lateralized praxis network during performance of gestures with either hand (Króliczak & Frey, 2009). It has been suggested that left hemispheric inhibition of the right hemisphere via the corpus callosum allows hemispheric specialization to be developed and maintained (Takeuchi, Oouchida, & Izumi, 2012). One of the possible reasons that individuals with left hemisphere strokes do not recover from disorders such as aphasia and apraxia is that those areas of the left hemisphere that remain intact may still inhibit the right hemisphere from developing the programs required to perform these activities. Conversely, in individuals with poststroke hemiparesis, a transcranial magnetic stimulation study has demonstrated increased interhemispheric inhibition from the intact to the injured hemisphere during attempted movement by the affected hand (Murase, Duque, Mazzocchio, & Cohen, 2004). It is not known whether a similar mechanism could be relevant to the ability to recover from poststroke apraxia. Increased interhemispheric inhibition from the intact (right) to the injured (left) hemisphere may prevent reorganization and recovery of the left hemisphere praxis network after left hemisphere injury. Thus, individuals with apraxia of their left upper limb from a callosal disconnection may have a better ability to recover from apraxia of their left upper limb than do individuals with left-sided apraxia from left hemisphere lesions. However, this patient, who was evaluated for the presence of IMA of her left hand after more than three decades, revealed no evidence of improvement.

The reason this woman revealed IMA but not LKA of her left hand is not known. There are at least two explanations for the presence of IMA and not LKA of the left hand after a callosal lesion. Left hemisphere ipsilateral motor pathways may have been able to mediate performance of deft finger movements and not pantomimes and gestures. However, research suggests that ipsilateral motor pathways primarily mediate more proximal limb movements rather than deft finger movements (Baker, 2011). It has been demonstrated that not all right-handed individuals with callosal disconnection develop IMA of their left upper limb (Lausberg, Cruz, Kita, Zaidel, & Pfito, 2003). There have even been reports of right-handed individuals who developed an IMA of the right hand from a right hemisphere lesion (Raymer et al., 1999), and therefore, it is also possible that in some individuals movement representations are stored in the right hemisphere or even in both cerebral hemispheres. Similarly, perhaps in some right-handed people the right hemisphere may contain the motor networks that can program independent but coordinated precise finger movements. Our patient reveals that these two networks are somewhat independent, such that her right hemisphere is able to mediate deft finger movements that are required for coin rotation. However, in order to perform pantomimes or gestures with the left hand, our patient's right hemisphere required input from multimodal spatial temporal movement representations, also called visuokinesthetic movement engrams (Heilman, Rothi, & Valenstein, 1982), that are

stored in her left hemisphere. Her callosal lesion disconnected these left hemispheric movement representations from the motor networks of the right hemisphere that control her left upper extremity. This presence of IMA without LKA suggests that the innervatory patterns necessary to guide deft finger movements do not require access to learned movement representations for transitive and intransitive gestures, which is consistent with Liepman's recognition of IMA and LKA as distinct forms of apraxia,

Our patient demonstrated a profound IMA during performance of both transitive gestures and intransitive gestures with the left hand. This finding is consistent with left hemispheric lateralization of visuokinesthetic movement engrams for both types of gestures. Our patient's inability to perform transitive gestures with the left hand in response to pictures of the tools or implements provides evidence that even when visual semantic representations, rather than language, activated visuokinesthetic movement engrams in the left hemisphere, the left hemisphere praxis network remained isolated from the right hemisphere. This nonverbal method of evaluation of transitive gestures cannot be used to evaluate intransitive gestures, and we cannot exclude the possibility of a disconnection between the left hemisphere language network and brain regions in the right hemisphere that may be necessary to perform intransitive gestures with the left hand. We did not test our patient's ability to imitate gestures during the recent evaluation, although ability to imitate gestures was reported to be impaired during the initial evaluation (Watson & Heilman, 1983). During imitation of a gesture, visual analysis of upper limb movement may activate visuokinesthetic movement engrams and allow the observer to then imitate the gesture. However, the ability to imitate meaningless gestures that have no learned visuokinesthetic movement engrams suggests that movement imitation may not require access to visuokinesthetic movement engrams (Ochipa, Rothi, & Heilman, 1994).

In addition to the brain networks that program the spatial and temporal components of purposeful skilled learned actions, as well as the motor cortex programming of deft movements, which in most right-handed people are lateralized to the left hemisphere, there are also left lateralized brain networks that represent conceptual mechanical knowledge (Heilman, Maher, Greenwald, & Rothi, 1997). This conceptual knowledge includes the knowledge of the mechanical advantage that tools may afford, as well as the types of actions that may be performed with specific tools, and the types of objects that may be acted upon by tools. Tactile input is transmitted by ascending sensory pathways to the contralateral hemisphere. If conceptual knowledge regarding tools and tool-object relations were lateralized to the left hemisphere and inaccessible to the right hemisphere, we would not have expected her to be able to point to pictures of tools with one hand when its associated recipient object was placed in the opposite hand, but she performed this task well. The posterior portions of her corpus callosum were not destroyed by the infarction and it is possible that this information could have been transferred using the posterior portions of her corpus callosum. However, it is also possible that pathways such as the anterior commissure that connects the anterior temporal

cortices may have been able to facilitate transfer of semantic information regarding tools, though this pathway was not able to transmit information regarding how to use the tools necessary to perform pantomimes and gestures with the left hand. Furthermore, the hemisphere that received sensory input from the tool placed in the contralateral hand may have been able to control ipsilateral proximal upper extremity muscles that mediate the ability to point to different locations in space.

Tactile anomia for items placed in the left hand is a classical sign of interhemispheric disconnection since the left hand provides sensory input to the right hemisphere and the disconnected left hemisphere contains the lateralized language network important for naming. Thus, either our patient's right hemisphere was able to program the articulatory motor programs necessary to name objects or there were available pathways, perhaps by means of the posterior corpus callosum, for sensory and/or conceptual representations of the objects placed in the left hand to access the left hemisphere's language-speech network. During a dichotic listening task, there is suppression of auditory input from the left ear in individuals with lesions of the splenium of the corpus callosum (Pollmann, Maertens, von Cramon, Lepsien, & Hugdahl, 2002). This suggests that interhemispheric transfer of auditory language information occurs posterior to the regions of the corpus callosum necessary for interhemispheric transfer between the left hemisphere praxis network and the right hemisphere.

Our patient appeared to have an apraxic agraphia of her left hand. Although both apraxia and agraphia of the left hand are characteristic signs of interhemispheric disconnection, the action representations for writing and tool use are potentially dissociable. For example, an individual may have IMA without apraxic agraphia (an inability to write letters) or apraxic agraphia without IMA (Coslett, Gonzales Rothi, Valenstein, & Heilman, 1986; Roeltgen & Heilman, 1983). This likely reflects a difference in the nature of the multimodal representations for tool use and writing. For example, while the actions needed for tool use are related to the kinesthetic structural properties of the tool and the spatial interaction between the tool and recipient object, the actions needed for writing are strongly influenced by visual representations of the letters, words, and the spatial and semantic relationships between words. However, in right-handed individuals, for both tool use and writing, multimodal action representations are acquired by sensory feedback from the right hand to the left hemisphere. This lateralized sensory input may contribute to the development of left hemisphere multimodal representations for ideomotor praxis and writing. However, it is not known to what extent left hemisphere specialization for deftness and motor learning initially facilitate the use of tools and writing implements by the right hand.

This woman was able to perform well when naming objects that were placed in her left hand when her eyes were closed; however, she had difficulty naming numbers written in the palm of her left hand. This dissociation of performance suggests, at least in part, that these two recognition-naming tasks may be mediated by different networks. To recognize letters or numbers written in their hand, with their eyes closed, healthy people may be able to recognize these letters or

numbers by two means. Based on the tactile input, people may develop a spatial representation and then access graphic visual representations. Alternatively, people can use movement working memory and match incoming movement patterns to stored letter or number movement representations.

To learn the method normally used to perform this graphesthesia task, Drago, Foster, Edward, Wargovich, and Heilman (2010) studied healthy participants who were blindfolded, by writing letters on their palm. These letters were written either in the normal manner or in a reverse direction. If letters written on the hand are recognized by their spatial features (as when visually reading), then the direction in which these letters are written should not influence letter recognition. In contrast, if the letters written on the hand are recognized by movement patterns, then recognition should be impaired when they are written in the reverse direction. Drago et al. (2010) found that when letters were written normally there were no differences in recognition errors between the tactile and visual modality; however, when letters were written in reverse, the normal participants made more errors in the tactile than visual condition. These results suggest that normally people identify letters written on their hand by covertly copying (mirroring) the examiner's movements and then accessing letter movement representations.

Individuals with corticobasal degeneration often reveal an asymmetrical IMA and apraxic agraphia with the same hand. The means by which letters written on the hand in the absence of vision are recognized might help explain why individuals with corticobasal degeneration often have graphesthesia associated with apraxia of the impaired hand (Heilman, Coenen, & Kluger, 2008). This may also help to explain that while without vision this patient could recognize objects placed in her left hand, she could not recognize numbers because recognition of these numbers would require that her left hand have access to the movement representations important for programming these number writing movements.

It is not known why some individuals develop IMA after a left hemisphere lesion while others do not, and factors that affect recovery from apraxia are also incompletely understood. Our patient developed profound and lasting IMA and apraxic agraphia of her left hand after a callosal infarction. The apraxia and agraphia cannot be attributed to an elemental motor deficit of the left hand or even to an inability to program sequential differentiated movements with the left hand and fingers since coin rotation ability was normal bilaterally. Furthermore, our patient did not appear to have a disconnection between the right hemisphere and the brain networks that mediate language and conceptual knowledge. However, the brain networks that store the knowledge of how to perform learned skilled transitive movements and the movements important for writing appeared to have remained completely inaccessible to our patient's right hemisphere.

Multimodal visual and kinesthetic memories for how to hold and use tools and implements are represented in left hemisphere brain networks. When the two cerebral hemispheres are connected, it is sufficient for these action representations to be lateralized to one hemisphere; however, it

remains unknown whether the left hemisphere specialization for praxis and writing develops because of many years of lateralized somatosensory and kinesthetic input to this hemisphere or whether other factors including left hemisphere specialization for language, dexterity, and motor learning facilitate initial adaptation of the right hand for the use of tools and implements. Over 30 years after a callosal infarction induced an interhemispheric disconnection, our patient's right hemisphere remained unable to acquire the ability to perform learned skilled purposeful transitive movements or gestures with her left hand. Based on the results of this woman's test performance, this inability of the right hemisphere to acquire, access, and/or maintain action representations cannot be primarily attributed to a lack of access to language or conceptual knowledge by her right hemisphere.

One possible explanation for this failure of the right hemisphere to acquire these skills is that our patient only used tools and implements with her right hand, and therefore, her right hemisphere never acquired the sensory input from the left hand that may be necessary to establish these action representations. However, our patient's vision was entirely intact, and therefore, her visual input had full access to the right hemisphere. Since we can acquire skills by just observation, there appears to be interhemispheric differences in the type and strength of synaptic connectivity between different brain regions, and these differences may determine the extent that the left and right hemispheres are capable of learning and maintaining different types of information.

Since the initial evaluation was completed over 30 years ago, we did not have the same test materials or access to recordings from the initial evaluation. A recording of the more recent evaluation was reviewed by an investigator who had also reported the initial evaluation (KMH). During the recent evaluation, our patient continued to demonstrate a profound asymmetric apraxia of the left upper limb consistent with that reported by Watson and Heilman (1983). We cannot say with certainty that there was no subtle improvement over time. However, during the recent evaluation it is clear that left hemisphere motor programming regions for performance of gestures and writing with the right hand remain intact and do not appear to have access to the right hemisphere as necessary to correctly perform gestures and write with her left hand as well as recognize numbers written on her left hand.

A limitation to this study is that we do not have recent imaging to confirm there have been no new extra-callosal lesions since the previously reported imaging (Watson, Heilman, & Bowers, 1985), although clinical evaluation does not suggest the presence of elemental motor deficits of either hand or damage to the left hemisphere praxis system. Our patient has had memory difficulty since the callosal infarction over 30 years ago, most likely related to injury to midline structures that are part of Papez circuit. It is not clear whether our patient's recent increased memory symptoms are related to longstanding memory difficulty or to development of age-related cognitive impairment, although aging and our patient's memory symptoms do not appear to be primary causes of persistent callosal apraxia. Apraxia is a major source of disability that can be caused by a variety of diseases and is a major determinant of prognosis for reestablishment of

functional independence after cerebral injury. Therefore, the effects of interhemispheric disconnection on the development of and recovery from apraxia should remain an important area for continued research and rehabilitation.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

Dr Heilman's research is funded by the United States Department of Veterans Affairs, the National Institutes of Health, and the State of Florida Department of Elder Affairs. The authors would like to acknowledge their patient for allowing them to learn from her case and share this report.

References

- Acosta, L. M., Bennett, J. A., & Heilman, K. M. (2014). Callosal disconnection and limb-kinetic apraxia. *Neurocase*, 20, 599–605. doi:10.1080/13554794.2013.826683
- Baker, S. N. (2011). The primate reticulospinal tract, hand function and functional recovery. *The Journal of Physiology*, 589, 5603–5612. doi:10.1113/jphysiol.2011.215160
- Coslett, H. B., Gonzales Rothi, L. J., Valenstein, E., & Heilman, K. M. (1986). Dissociations of writing and praxis: two cases in point. *Brain and Language*, 28, 357–369. doi:10.1016/0093-934X(86)90111-2
- Drago, V., Foster, P. S., Edward, D., Wargovich, B., & Heilman, K. M. (2010). Graphesthesia: a test of graphemic movement representations or tactile imagery? *Journal of the International Neuropsychological Society*, 16, 190–193. doi:10.1017/S1355617709990762
- Geschwind, N., & Kaplan, E. (1962). A human cerebral deconnection syndrome: a preliminary report. *Neurology*, 12, 675–675. doi:10.1212/WNL.12.10.675
- Goodglass, H., & Kaplan, E. (1963). Disturbance of gesture and pantomime in aphasia. *Brain*, 86, 703–720. doi:10.1093/brain/86.4.703
- Graff-Radford, N. R., Welsh, K., & Godersky, J. (1987). Callosal apraxia. *Neurology*, 37, 100–105. doi:10.1212/WNL.37.1.100
- Hanna-Pladdy, B., Mendoza, J. E., Apostolos, G. T., & Heilman, K. M. (2002). Lateralised motor control: hemispheric damage and the loss of dexterity. *Journal of Neurology Neurosurgery and Psychiatry*, 73, 574–577. doi:10.1136/jnnp.73.5.574
- Heilman, K. M., Coenen, A., & Kluger, B. (2008). Progressive asymmetric apraxic agraphia. *Cognitive and Behavioral Neurology*, 21, 14–17. doi:10.1097/WNN.0b013e318165b133
- Heilman, K. M., Maher, L. M., Greenwald, M. L., & Rothi, L. J. (1997). Conceptual apraxia from lateralized lesions. *Neurology*, 49, 457–464. doi:10.1212/WNL.49.2.457
- Heilman, K. M., Rothi, L. J., & Valenstein, E. (1982). Two forms of ideomotor apraxia. *Neurology*, 32, 342–342. doi:10.1212/WNL.32.4.342
- Królczyk, G., & Frey, S. H. (2009). A common network in the left cerebral hemisphere represents planning of tool use pantomimes and familiar intransitive gestures at the hand-independent level. *Cerebral Cortex*, 19, 2396–2410. doi:10.1093/cercor/bhn261
- Lausberg, H., Cruz, R. F., Kita, S., Zaidel, E., & Ptito, A. (2003). Pantomime to visual resensation of objects: left hand dyspraxia in individuals with complete callosotomy. *Brain*, 126, 343–360. doi:10.1093/brain/awg042
- Liepmann, H. (1920). Apraxia. *Ergebnisse der gesamten Medizin*, 1, 516–543.
- Liepmann, H., & Maas, O. (1907). Fall von linksseitiger Agraphie und Apraxie bei rechtsseitiger Lahmung [The case of left-sided agraphia and apraxia with right sided paralysis]. *Zeitschrift für Psychologie und Neurologie*, 10, 214–227.
- Murase, N., Duque, J., Mazzocchio, R., & Cohen, L. G. (2004). Influence of interhemispheric interactions on motor function in chronic stroke. *Annals of Neurology*, 55, 400–409. doi:10.1002/(ISSN)1531-8249
- Ochipa, C., Rothi, L. J., & Heilman, K. M. (1994). Conduction apraxia. *Journal of Neurology, Neurosurgery & Psychiatry*, 57, 1241–1244. doi:10.1136/jnnp.57.10.1241
- Poizner, H., Mack, L., Verfaellie, M., Rothi, L. J. G., & Heilman, K. M. (1990). Three dimensional computer graphic analysis of apraxia. *Brain*, 113, 85–101. doi:10.1093/brain/113.1.85
- Pollmann, S., Maertens, M., von Cramon, D. Y., Lepsien, J., & Hugdahl, K. (2002). Dichotic listening in patients with splenial and nonsplenial callosal lesions. *Neuropsychology*, 16, 56–64. doi:10.1037/0894-4105.16.1.56
- Raymer, A. M., Merians, A. S., Adair, J. C., Schwartz, R. L., Williamson, D. J., Rothi, L. J., ... Heilman, K. M. (1999). Crossed apraxia: implications for handedness. *Cortex*, 35, 183–199. doi:10.1016/S0010-9452(08)70793-7
- Roeltgen, D. P., & Heilman, K. M. (1983). Apractic agraphia in a patient with normal praxis. *Brain and Language*, 18, 35–46. doi:10.1016/0093-934X(83)90004-4
- Rothi, L. J. G., Mack, L., Verfaellie, M., Brown, P., & Heilman, K. M. (1988). Ideomotor apraxia: error pattern analysis. *Aphasiology*, 2, 381–387. doi:10.1080/02687038808248942
- Snodgrass, J. G., & Vanderwart, M. (1980). A standardized set of 260 pictures: norms for name agreement, image agreement, familiarity, and visual complexity. *Journal of Experimental Psychology: Human Learning and Memory*, 6, 174–215. doi:10.1037/0278-7393.6.2.174
- Takeuchi, N., Oouchida, Y., & Izumi, S. (2012). Motor control and neural plasticity through interhemispheric interactions. *Neural Plasticity*, 2012, 1–13. doi:10.1155/2012/823285
- Watson, R. T., Fleet, W. S., Rothi, L. J. G., & Heilman, K. M. (1986). Apraxia and the supplementary motor area. *Archives of Neurology*, 43, 787–792. doi:10.1001/archneur.1986.00520080035016
- Watson, R. T., & Heilman, K. M. (1983). Callosal apraxia. *Brain*, 106, 391–403. doi:10.1093/brain/106.2.391
- Watson, R. T., Heilman, K. M., & Bowers, D. (1985). Magnetic resonance imaging (MRI, NMR) scan in a case of callosal apraxia and pseudoneglect. *Brain*, 108, 535–536. doi:10.1093/brain/108.2.535