

CALLOSAL APRAXIA

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SUMMARY

A 43-year-old woman suffered a spontaneous corpus callosum disconnection, resulting in apraxia and apraxic agraphia confined to the left hand. She initially had a functionally total callosal disconnection. With time, the splenium of the corpus callosum became functional, and a computerized tomographic scan performed five months after the onset showed infarction of only the body of the corpus callosum. Concomitant with this improvement in callosal function, the apraxia changed from ideational (loss of the concept of skilled movements) to classic ideomotor apraxia. A temporal analysis of this case provided support for Liepmann's (Liepmann, 1900; Liepmann and Maas, 1907) hypothesis that there is a centre for visuokinaesthetic (space-time) engrams in the left hemisphere of right-handed patients that controls skilled motor acts in either hand. This patient's recovery also allowed us a better understanding of the mechanisms underlying various types of apraxia.

INTRODUCTION

Apraxia is a disorder in the execution of learned skilled movement that cannot be explained by lack of comprehension or by inattention, sensory loss, weakness, ataxia or basal ganglia disorder. Liepmann (1900) was the first to study apraxia systematically. He first described a government official with apraxia in both arms, the impairment being much greater on the right side than on the left. Liepmann used this case to show that impaired motor execution could exist with intact language comprehension.

In 1907 Liepmann and Maas described a second case, that of Ochs, a 70-year-old carpenter who had transcortical motor aphasia with apraxia and agraphia of the left arm and with right hemiplegia. His apraxic (left) hand did not improve with imitation or actual object usage, and the left hand agraphia did not improve with use of anagram letters. Post-mortem examination showed a left anterior cerebral artery infarction with damage extending from the first frontal convolution through the white matter to the paracentral lobule with destruction of the anterior two-thirds of the corpus callosum. There was also a lesion of the left pons, which explained the right hemiplegia. Liepmann and Maas postulated that engrams for skilled movements are lateralized and in right-handers these engrams are in the left hemisphere.

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These engrams were called *space-time engrams* by Liepmann in 1920 (see Brown, 1972) and *visuokinesthetic motor engrams* by Heilman (1979). Heilman (1979) proposed that these engrams were stored in the parietal lobe.

These engrams are needed to perform learned skilled acts. Depending upon the nature of the act, specific body parts must be placed in certain spatial positions. The spatial positions assumed by the relevant body part depend not only on the nature of the act but also on the position and size of an external object with which the body parts must interact if an external object is present. Unlike static postures, skilled acts require orderly sequential changes in the spatial positions of the body parts over time. These space-time engrams command the motor systems to adopt the appropriate spatial positions of the relevant body parts over time. Because these engrams for skilled movement are lateralized, Liepmann and Maas thought that a lesion of the corpus callosum would prevent the space-time engrams in the left hemisphere from reaching the right sensorimotor area necessary to carry out the skilled act with the left hand, thereby inducing apraxia.

Since Broca's (1865) description of eight aphasic right-handed patients who had left hemisphere lesions, it has been well established that in right-handers the left hemisphere mediates language. Liepmann and Maas (1907) recognized that a callosal lesion therefore disconnects the motor area in the right hemisphere from the language-dominant left hemisphere. If a right-handed patient with a callosal lesion had a language-motor disconnection, the patient should be unable either to perform skilled movements to command or to write with the left hand. If, however, as Liepmann and Maas suggested, right-handed patients also have space-time or visuokinaesthetic engrams in their left hemisphere, a callosal lesion would not only disconnect language from the right hemisphere but would also disconnect the left hemisphere space-time motor engrams from the right hemisphere motor areas. Such patients should be unable to carry out commands with their left hand and also fail to imitate and use actual objects correctly.

Since Liepmann and Maas's report, there has been little support for this hypothesis. Geschwind and Kaplan (1962) described a patient with a left hemisphere glioblastoma and a postoperative left anterior cerebral artery infarction that had caused destruction of the anterior four-fifths of the corpus callosum. That patient could not follow commands with his left hand but could imitate and use actual objects. He was agraphic in the left hand and could not type or use anagram letters with the left hand but performed flawlessly with his right hand. He followed commands with his right hand but not with his left. He failed when asked to draw with his right hand an object placed in his left hand. The aphasic agraphia was interpreted as disconnection of the right hemisphere from left hemisphere speech areas, and the apraxic difficulties were attributed to having stimulus (verbal or nonverbal) and response separated across the hemispheres. Geschwind (1975) concluded that in the verbal condition many cases of apraxia are caused by a verbal-pyramidal system disconnection, intrahemispherically or interhemispherically. For example, intrahemispherically, a left premotor lesion or a lesion disconnecting

Wernicke's area from the left premotor region would cause the patient to be unable to perform distal skilled movements with either hand in response to a command. Interhemispherically, a callosal lesion would disconnect the left hemisphere from right premotor cortex and cause apraxia isolated to the left hand. Clumsily performed attempts at skilled acts were thought to be under extrapyramidal control. This could explain the dysfunction in Geschwind and Kaplan's patient, who could imitate and use objects correctly, because nonverbal cues could presumably activate the right pyramidal system. Their conclusion could not explain the frequently observed impairment of praxis for imitation. However, these investigators also agreed with Liepmann and Maas (1907) and accepted the postulate that the left hemisphere in right-handed persons may dominate motor skills and that their callosal patient could be the exception. Further investigation (Heilman *et al.*, 1973, 1974) showed that dominance for language and dominance for skilled motor acts are separable. Therefore, a callosal disconnection may induce a verbal motor disconnection alone or disconnect language and space-time engrams from right hemisphere motor areas.

Gazzaniga *et al.* (1967) observed that relatively pure surgically induced callosal lesions caused mild disturbance of praxis, limited to fine differential movements of thumb and fingers. They concluded that each hemisphere exerts control over homolateral and contralateral limbs and that previous cases of severe apraxia following a callosal lesion were secondary to extracallosal damage. Limiting the surgery to the corpus callosum and hippocampal commissure and operating in two stages has reduced the 'acute disconnection syndrome'—apraxia of the left limbs, mutism, apathy, confusion and infantile behaviour (Wilson *et al.*, 1982). Furthermore, improvement in praxis was thought to be secondary to more acquisition of homolateral control with time (LeDoux *et al.*, 1978).

Recently we had the opportunity to examine a patient with an acute naturally occurring callosal lesion who, unlike the patient of Geschwind and Kaplan (1962), had severe apraxia with imitation and object usage, thereby providing support for Liepmann's hypothesis. This patient also had other signs and symptoms that may help to elucidate the neuropsychological mechanisms underlying other varieties of apraxia and the mechanisms underlying apraxic agraphia.

CASE REPORT

A 43-year-old right-handed woman without history of previous illness, on September 5, 1981, suddenly developed severe headache followed by vomiting, mutism and inability to stand. She was admitted to the University of Florida's Shands Teaching Hospital where examination showed neck stiffness and a blood pressure of 142/98. Initially she had no spontaneous speech but repeated complex phrases normally. She followed verbal commands by pointing with her right hand, and could follow written commands presented in her right visual field but not in her left visual field. She could perform a finger snap flawlessly and a coin flip to command with her right hand but not with her left hand. Her cranial nerves were normal. She orientated to visual stimuli in both visual fields with no evidence of hemianopia. She had bilateral weakness of her legs, the right being weaker than the left. Strength in her arms was normal.

A computerized tomogram (CT) showed a haemorrhage in the region of the corpus callosum, extending from the genu to the splenium. Coronal reconstruction showed this to be above the corpus callosum. The following day cerebral angiography did not reveal the cause of the haemorrhage but showed severe spasm of both anterior cerebral arteries.

Examination two days after admission showed the patient to be alert but severely hypokinetic with a long latency between any stimulus and response. She orientated to both sides equally. She was mute, except for use of a rare single word. After a long latency, she could repeat phrases such as 'no ifs, ands or buts'. She comprehended simple pointing commands such as 'point at your doctor, point to the television'. She comprehended simple written sentences and named objects without difficulty. On praxis testing, with her right hand she pantomimed flawlessly to commands like 'comb your hair'. Asked to pantomime with her left hand to command, she would extend her fingers, look at them and then flex and extend them slowly. During her performance she had a bewildered expression on her face. She did not improve with imitation or with the use of actual objects. She could write single words with her right hand but not with her left.

She was thought to have a callosal disconnection syndrome, a severe transcortical motor aphasia and generalized hypokinesia from supplementary motor area involvement, and proximal leg weakness from medial motor cortex ischaemia, all caused by anterior cerebral artery spasm.

Three days after the haemorrhage she was noted to be orientating more to her left than to her right. On bilateral simultaneous facial tactile stimulation, she could demonstrate that both sides of her face were touched by holding up two fingers. In spite of spontaneous orientating to her left, she failed to report all left limb tactile stimuli when stimulated either unilaterally or bilaterally. She continued exhibiting transcortical motor aphasia and was found to be hemialexic, reading 'boat' when presented with the word *houseboat* and 'house' when presented with the word *doghouse*. Because she spontaneously orientated more to her left than to her right, she did not have left side neglect; this, therefore, could not explain her hemialexia. When blindfolded she could not name objects placed in her left hand. She could, however, pick the object from a multiple choice array (four foils and the correct object). Unlike patients with astereognosis, she systematically explored the surface of large and of small objects, which she manipulated with normal dexterity with her left hand. She wrote single words with her right hand but not with her left. Her severe isolated left hand apraxia persisted. In spite of her inability to perform the simplest pantomimed act to command, to imitate or to use an actual object with her left hand, she would more often use the left hand rather than the right to scratch her head, wipe her brow, scratch her nose or rub her eye. These acts were also performed in a normal fashion without any evidence of clumsiness. She made fine, precise and well-coordinated finger movements. She could pick up small, thin coins from a table top by using her left thumb and forefinger in the normal pincher-like grasp that is often lost with pyramidal disease (Brinkman and Kuypers, 1973). There was never any evidence of extrapyramidal dysfunction or ataxia.

On the fifth day after the haemorrhage, she fluctuated from transcortical motor aphasia to almost normal linguistic ability. She could write with her right hand but not with her left hand and looked at the pen as if she did not know what to do with it.

By the ninth day she continued to initiate speech slowly. She was mildly dysarthric, but repetition was thought to be no better than was spontaneous speech. She still could not pantomime or imitate with the left hand. With actual objects there was some movement present in the left hand but nothing approximating to the correct movement. In palpating objects for identification with the left hand, she would simultaneously make movements with her right hand as if she were manipulating the object. She continued to show no buccofacial apraxia and when asked to 'blow a kiss' using her left hand, she raised the left hand but did not coordinate it with her mouth or with blowing. Nevertheless, this association with buccofacial praxis appeared to improve her left hand praxis slightly.

She had difficulty naming affective expression, pointing to a named facial affect like 'point to the happy face', and deciding whether two faces showed the same or a different emotion. When she was read sentences and asked to point to one of the four pictures depicting the semantic message of the sentence, she performed flawlessly. When read the same sentence in affective intonations (for example,

happy, sad, angry or indifferent), she was unable, either by naming or pointing, to identify the affect of the speaker. She seemed puzzled, and after a long latency stated, 'I am confused . . . this is confusing'. She also could not repeat affective intonation produced by the examiner. Once again, she appeared puzzled and stated, 'this is confusing'. In contrast, she flawlessly repeated lengthy and syntactically complex sentences without affective intonation. In her spontaneous speech she appropriately produced happy intonation.

By the twenty-fifth day she had consistently regained the ability to name small objects placed in her left hand. She had no drift of outstretched extremities or pseudoathetosis of her fingers. Clinical examination showed that position and vibration sense of the left fingers were normal. Strength and tone of her arms remained normal and without tremor or ataxia in performing distal movements. On praxis testing, when asked to pantomime to command, she would still look at her left hand in a bewildered fashion. Although the imitated act was poorly performed because of spatiotemporal movement errors, the intent of the act was recognized as appropriate. She improved further in the use of actual objects but continued to have severe spatiotemporal impairment (for instance, with a key in her hand she moved it slowly forward but then stared at it without making a turning movement with her wrist). With a hammer she made a few slow, large-amplitude swings to and fro but not in an up-and-down direction. She initiated language after a short latency and had excellent comprehension and fluent output. The strength in her lower limbs had improved, and she could stand with assistance. She complained spontaneously that her two hands 'fight each other'. For example, when she held an envelope, each hand independently and simultaneously tried to release and hold, so that she would tug at the envelope sometimes for as long as ten minutes before saying 'the hell with it' and throwing it away with her right hand. She stated 'I just can't make my left hand do what it is supposed to'.

Forty-four days after the ictus, examination showed that she was alert and orientated, with normal comprehension and language output. She could write normally with her right hand. Writing with her left hand produced an illegible scrawl (fig. 1). When writing the alphabet with her left hand, she also produced only a few recognizable letters, but when copying the alphabet, although it was done with great effort, she showed definite improvement. In spite of this agraphia, she could type normal sentences using her left hand (fig. 1). With her left hand she slavishly copied a cube that had good visuospatial relationships; with her right hand it was done with motoric ease but was visuospatially incorrect. Praxis testing showed that in pantomime to command, she now showed the correct intent of actions although she remained severely apraxic. Proximal left arm movements were performed perfectly (for example, using her left arm as if it were a wing, pulling a chair from under a table). When she attempted to pantomime throwing a ball to command, the proximal movements were normal but hand release was severely abnormal. She was unable to pantomime correctly using a comb, key or hammer, or to flip a coin and improved only minimally with imitation. She improved much more using the actual objects; her movements, however, remained apraxic.

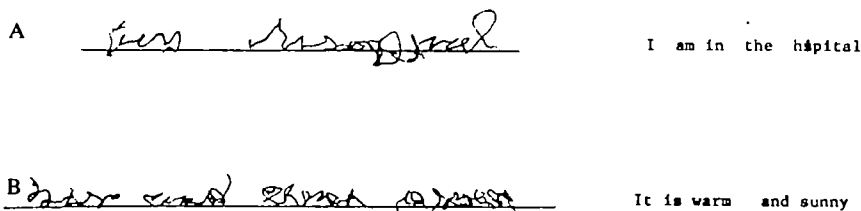


FIG. 1. A, on the left is an attempt by the patient to write a sentence with her left hand in response to 'Where are you?' On the right is her typewritten response using her left hand. The patient is not a skilled typist, and the error in *hospital* was typographical. B, on the left is an attempt to write a sentence with her left hand in response to 'What kind of weather are we having today?' On the right is her typewritten response with her left hand.

Her family stated that her generally happy premorbid mood was unchanged, but that when she saw something sad on television, she would cry, which she would not have done previously. The family kept records of some of the interesting behaviour that demonstrated the lack of cooperation between her left and her right hands. One day she decided to wear a blouse; she took one out of the closet with her left hand and a separate one with the right hand. She then put her left arm in one blouse and her right arm in the other. The two hands never helped each other; the left hand pulled the right blouse off, and the right arm put the blouse back on, whereupon the left pulled it off once again. Her daughter then intervened. On another occasion she opened a cabinet with her left hand and with her right hand reached into the cabinet only to have her left hand close the door on her right arm. On still another occasion she put her right arm half-way into a sweater and then her left hand started taking the sweater off. She repeated this twice. Her left hand then put the sweater on the floor, her right hand picked it up and started to put it on, whereupon the left arm again started to take it off.

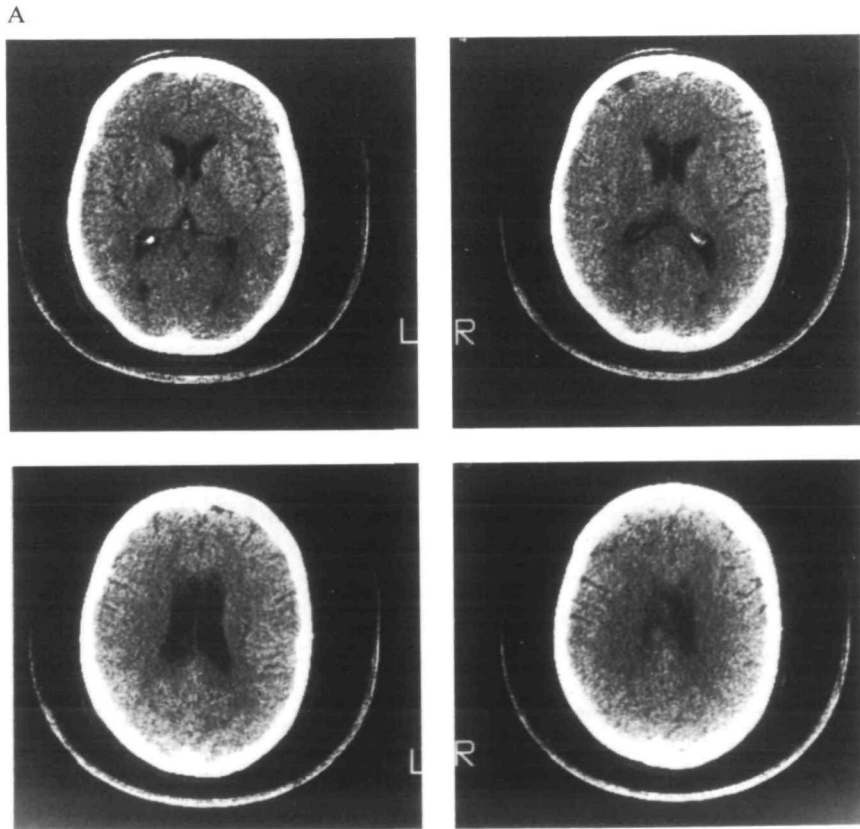
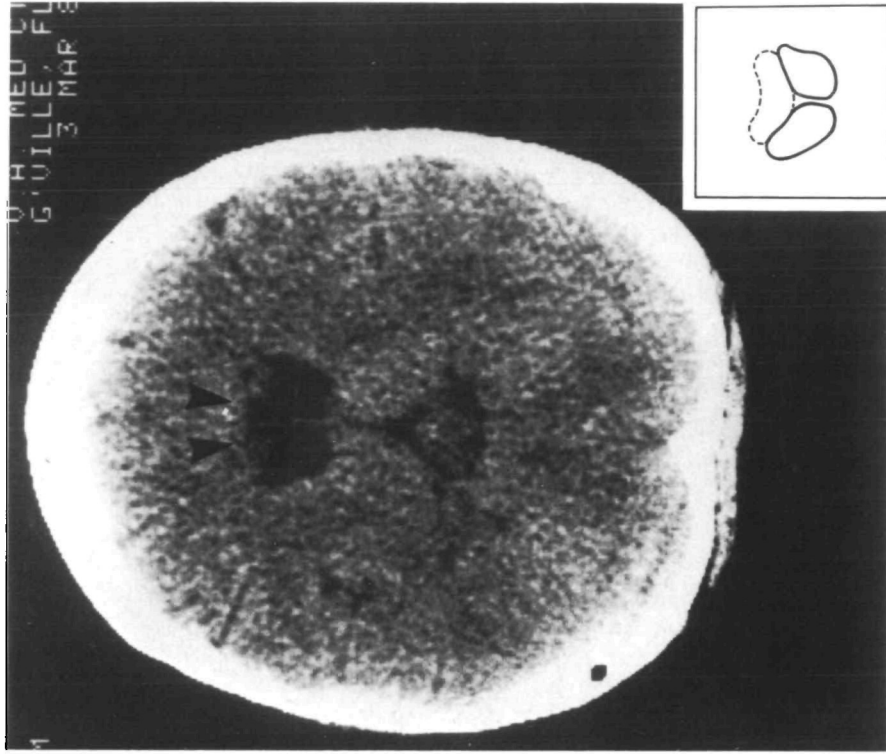
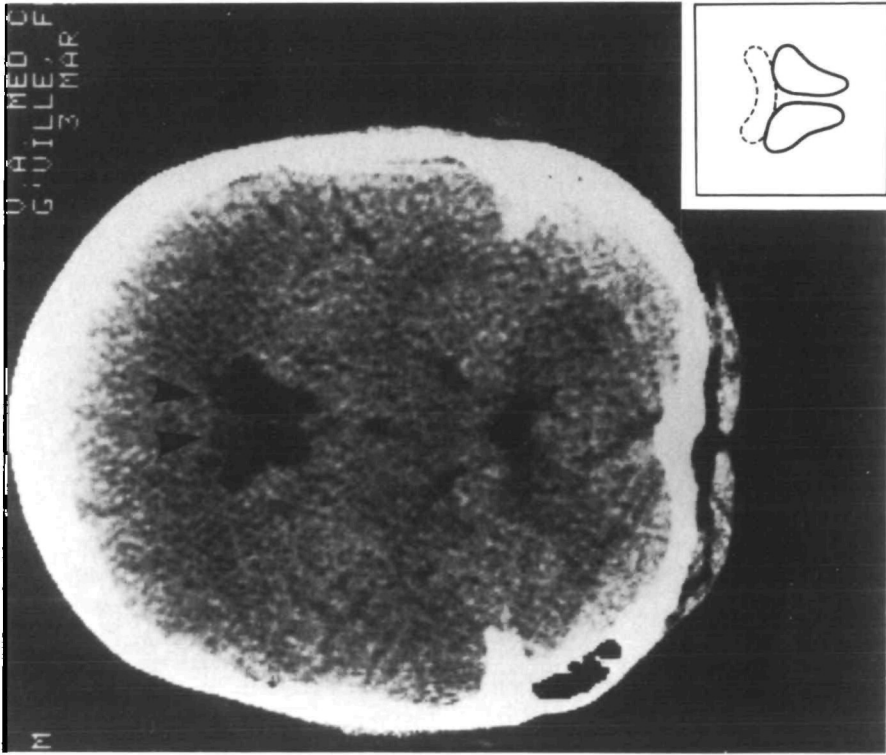
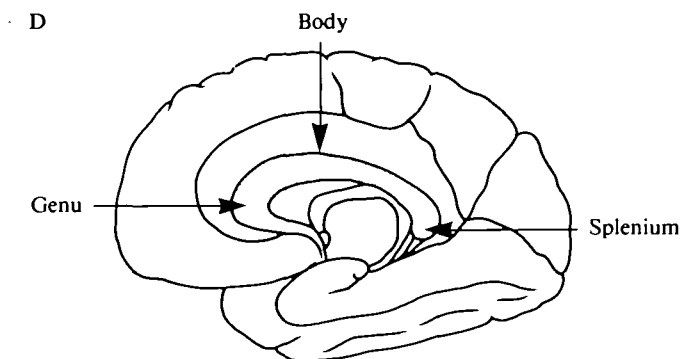


FIG. 2. Computerized tomographic scans five months after the ictus. A, horizontal sections showing infarction of the body of the corpus callosum. B, 35 deg coronal section showing infarction at the junction of the genu and body of the corpus callosum. The insert shows the relationship between the infarction and the ventricles diagrammatically. C, 35 deg coronal section showing infarction of the body of the corpus callosum. This insert also shows the relationship between the infarction and ventricles. D (*overleaf*), artist's reconstruction of callosal infarction.





Five months post-onset, she continued to have severe ideomotor apraxia (*see* Table) confined to the left arm. When she attempted to perform a pantomime to command, the intent of her actions was clearly recognizable although severely apraxic. She improved with imitation and further improved with actual object usage although remaining apraxic. The results of her neurological examination were otherwise normal. Horizontal and 35 deg coronal CT scans were sent to Dr William Scott (New and Scott, 1975). Without providing him with clinical details, we asked him to determine all abnormalities and their exact anatomical extent. His 'blind' interpretation was of an extensive infarction of the body of the corpus callosum (fig. 2A-D). The most anterior extent of the infarction was at the junction of the genu and body. The posterior one-fourth to one-fifth of the body and all of the splenium were intact. There was no cortical involvement, including supplementary and cingulate cortex.

TABLE. DISTINGUISHING FEATURES OF VARIOUS TYPES OF APRAXIA

<i>Type of apraxia</i>	<i>Pantomime to command</i>	<i>Imitation</i>	<i>Object usage</i>
Ideational	Does not demonstrate correct intent. Loss of movement concept	Same as to command	Same as to command
Verbal-motor disconnection	Same as ideational	Correct	Correct
Ideomotor	Correct intent but spatiotemporal errors	Improves over command but spatiotemporal errors may persist	Improves over imitation but spatiotemporal errors may persist
Verbal-motor disconnection plus ideomotor	Same as ideational	Shows correct intent. Improves over command but spatiotemporal errors may persist	Improves over imitation but spatiotemporal errors may persist

DISCUSSION

This is a case of spontaneous disconnection of the body of the corpus callosum caused by haemorrhage-induced spasm of the anterior cerebral arteries resulting in infarction. During the patient's hospital course she showed many of the signs and symptoms that have been previously described for surgical disruptions of the corpus callosum. The major focus of this report, however, is the left side apraxic disorder.

During recovery she has evolved through three types of apraxia (*see* Table): initially she had ideational apraxia, then verbal-motor disconnection with ideomotor apraxia and, finally, severe and lasting ideomotor apraxia. The apraxia was exclusively limited to her left extremities. She could always recognize the appropriate act from multiple similar acts performed by the examiner.

Initially, when asked to pantomime skilled acts with her left hand, she looked at the hand and alternately pronated and supinated it or flexed and extended her fingers. She could not even attempt the requested pantomime. This behaviour resembled that previously described by Heilman (1973). Heilman (1979) postulated that the disorder in his patients was being induced by a verbal-motor disconnection; that is, although they verbally comprehend the command to pantomime, this command will not excite the visuokinaesthetic (space-time) engrams. However, unlike performance of patients who can imitate and use actual objects flawlessly (Heilman, 1973), this patient's performance during imitation or with the actual object was precisely the same as it was to command; initially her performance appeared to be like that described by Liepmann and Maas (1907). Some aphasics with profound comprehension disorders cannot pantomime a skilled act to verbal commands but can perform skilled acts when given the actual objects. These observations suggest that verbal comprehension and knowledge of object use are dissociable. The failure of this patient to use actual objects with her left hand therefore cannot be entirely explained by an inability of her right hemisphere to understand a command (either directly or *via* the corpus callosum). Similarly, although severely apraxic patients with left hemisphere lesions may have difficulty with the spatial and temporal dimensions of their movements when using actual objects, these patients demonstrate that they know the intended use of the objects. Our patient's inability to demonstrate the intended use of an object, or even to attempt using actual objects, cannot therefore be explained by a verbal-motor disconnection or by a disconnection of visuokinaesthetic motor engrams from the right hemisphere motor areas. When she first held objects in her left hand, her performance was as if she had never seen or worked with these objects and did not know their intended use. We therefore suspect that initially there was a conceptual disorder. The disconnected right hemisphere did not know what the object was used for or even that it was to be used. Unfortunately, the term *ideational apraxia* has been used for several disorders (Heilman, 1979). Ideational apraxia perhaps best describes the left-hand performance by this patient. Because the right hand performed normally, the concepts of what objects are used for were not destroyed but were disconnected from the right hemisphere.

At about three weeks after the haemorrhage, the nature of the apraxia in this patient changed. When asked to pantomime, she continued either to supinate and pronate her hand or to flex and extend her fingers. Given objects, she attempted to use them. Although she made clumsy movements that were incorrectly orientated and sequenced in space, it was possible to recognize the correct intent of the movement. This improvement coincided with a return of her ability to name objects

in her left visual field and in her left hand. The hemialexia had also resolved. These functions are thought to be mediated by the posterior portions of the corpus callosum. The CT scans showed that the posterior portion of the corpus callosum was spared, and although initially it may not have been anatomically disconnected, it may have been functionally disconnected. This dysfunction improved, allowing posterior callosal interhemispheric communication. Furthermore, it may be that the concepts of what objects are used for reached the right hemisphere *via* the posterior route. There are, however, alternative possibilities. Because use of objects did not change when this patient used her vision during the act, we conclude that visual control of ipsilateral pathways could not account for her improvement with object use. The right hemisphere nevertheless may have gained the ability to recognize the object and conceive its use, or the patient may have been using ipsilateral motor pathways. With regard to the latter postulate, proximal movements can be mediated by ipsilateral pathways more than distal movements (Brinkman and Kuypers, 1973), and our patient was best able to perform proximal movements. Against this ipsilateral postulate is the observation that despite normal dexterity, distal movements were poorly performed, although the intent was clearly appropriate. At this stage her total inability to carry out recognizable gestures and to pantomime with the left hand in response to a command but to demonstrate the correct intent with actual objects suggested that the callosal lesion induced a verbal-motor disconnection. The observations that apraxic motor behaviour (spatiotemporal errors) were also found with imitation and actual use of objects with the left hand, also suggested a disconnection of the visuokinaesthetic motor engrams (space-time engrams) from the right hemisphere.

Approximately seven weeks after the haemorrhage, she was able to pantomime with her left hand in response to a command. Although we could recognize the intent of the pantomime, it was apraxic. Her performance with actual object use and imitation was better than that with pantomime, but remained apraxic. The performance of her left hand now could not be accounted for by a verbal-motor disconnection but appeared to be induced by a disconnection of her visuokinaesthetic motor engrams (space-time engrams) from the motor apparatus that controls the left hand. The patient's performance now appeared to be like that of ideomotor apraxia.

We are unsure how the right hemisphere regained access to language. Although we suspect that it used the posterior portions of the callosum, we cannot rule out that her right hemisphere was now directly comprehending commands.

Our patient also had true apraxic agraphia of the left hand. With her right hand, she wrote normally, but her left hand produced an illegible scrawl. Unlike the patients of Liepmann and Maas (1907) and Geschwind and Kaplan (1962), our patient could type linguistically flawless sentences with her left hand. She could type spontaneously and to dictation. Although the linguistic portion of this task may have been mediated by the right hemisphere, she used functional words, was not asyntactic, used abstract words and therefore was most probably using the left

hemisphere. Because she could not write with her left hand but could type supports the postulate that the apraxia was not induced by a verbal-motor disconnection. The patients described by Liepmann and Maas (1907) and by Geschwind and Kaplan (1962) had destruction of the genu of the corpus callosum, which was spared in our patient. Because their patients had left-handed apraxic and aphasic agraphia (could not write with anagram letters) and our patient had only apraxic agraphia (could write with a typewriter), perhaps the genu of the corpus callosum is responsible for transmitting verbal-motor programmes to the right hemisphere and the body of the corpus callosum is responsible for transmitting space-time (visuokinaesthetic) engrams to the right hemisphere.

Two recent cases demonstrated aphasic agraphia of the left hand, without apraxia, caused by lesions of the splenium of the corpus callosum (Sugishita *et al.*, 1980; Gersh and Damasio, 1981). Perhaps linguistic information from Wernicke's area destined for the right hemisphere crosses at the splenium. This linguistic information may be different from the verbal-motor information crossing at the genu, and these different lesions might induce different forms of disconnection aphasic agraphia.

Finally, this patient demonstrates the 'alien hand' sign. Her left hand would act in an uncooperative fashion. This has been thought to be secondary to callosal dysfunction but recently was attributed to damage of the medial frontal cortex, including the supplementary motor area and cingulate gyrus, contralateral to the alien hand (Goldberg *et al.*, 1981). Although the present patient had clinical evidence of medial frontal dysfunction early in her illness, manifested by transcortical aphasia and bilateral leg weakness, she subsequently maintained the alien hand after these problems cleared. Furthermore, at no time did she show grasp reflexes or perseveration. The CT scans did not show abnormalities in the medial cortex. Therefore, our patient's alien hand seems more related to callosal dysfunction than to medial frontal cortex infarction.

Why does our patient's disorder differ from previously described cases of callosal disconnection? Many of the patients who had surgical callosal lesions had prior seizures and brain injury. Either of these dysfunctions may have induced brain reorganization. However, Geschwind and Kaplan's (1962) patient did not have these factors; therefore, the absence of a left-hand ideomotor apraxia cannot be entirely explained by brain reorganization. In right-handers right hemispheric lesions almost never produce apraxia; however, left hemisphere lesions in areas known to induce both aphasia and apraxia more often induce aphasia. In one study only 20 of 35 aphasic patients were also apraxic (Heilman, 1975). The discrepancy between the incidence of aphasia and apraxia suggested that although in right-handers visuokinaesthetic (space-time) motor engrams for skilled movements are localized in the left hemisphere, the right hemisphere in many of these persons can substitute for the left. Because patients with large left hemisphere lesions in the region known to induce apraxia sometimes have aphasia without apraxia, it should not be surprising that callosal damage can induce not only verbal-motor

disconnection apraxia but also ideomotor apraxia. Alternatively, the difference between our patient and the one described by Geschwind and Kaplan (1962) may not relate to brain organization but rather to the size of the lesion and its location.

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