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IDEATIONAL APRAXIA—A RE-DEFINITION

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LIEPMANN described ideomotor apraxia (IMA) as a movement disorder where there was a disconnexion between the idea of the movement and its motor execution (Liepmann, 1908). The patient with IMA has greatest difficulty with movement to commands. Imitation may help a little but frequently not at all, whereas with manipulation of the object there is usually a significant improvement (Geschwind, 1965). Ideational apraxia (IA) has been defined by several authors as a defect in the actual handling of objects (De Renzi *et al.*, 1968; Morlaas, 1928). Several investigators have raised questions as to whether a separate classification is justified (Geschwind, 1965; Zangwill, 1960; Brown, 1972) or whether IA might simply represent such a severe stage of IMA as to involve even the easiest gestures, i.e. use of actual objects (De Renzi *et al.*, 1968; Zangwill, 1960). A recent study demonstrated that although patients with IMA showed improvement with actual handling of objects, these patients nevertheless show a defect of their non-paretic hand in more sensitive tests for actual object handling, i.e. rapid tapping with their left hand (Heilman, 1973). Is there then an ideational apraxia?

To better understand the nature of apractic problems the following 3 patients were intensively evaluated and appear to exhibit features definable as IA.

CASE REPORTS

Case 1.—A 34-year-old right-handed woman had sudden loss of consciousness and when she awoke she noted a transient language disturbance. EEG and brain scan performed at another hospital showed questionable abnormalities of the left parietal region. She was treated with diphenylhydantoin and phenobarbital; because of urticaria, however, the former had to be discontinued and the patient had several other episodes of loss of consciousness followed by language disturbances. She was referred to the University of Florida Teaching Hospital and on examination it was noted that the patient was aphasic. She used frequent circumlocutions in her spontaneous speech and upon attempts to name on confrontation the patient showed a severe anomia which was not modality specific or unilateral. She was, however, able to describe what objects were used for and could always select the correct object in a multiple choice type question. The patient's comprehension was intact as was series speech. She had difficulty with repetition of highly grammatic sentences, i.e. no if, ands or buts. In addition, the patient had

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dyslexia, dysgraphia, dyscalculia, and constructional apraxia but did not demonstrate finger agnosia or right-left confusion.

On apraxia testing, when asked to pantomime certain motions with either her right or left arm (i.e. "Show me how you would use a key") the patient looked at her outstretched hand, then asked the examiner to repeat the question. When the question was repeated the patient would look down at her hand and say, "I can't do it." When asked if she understood the question, the patient would verbally demonstrate comprehension, i.e. "Keys are used for opening locks." When the correct movement was shown to this patient in a multiple choice fashion, she was always able to select the correct movement. She was able to imitate in a flawless manner even with minimal cuing and she performed extremely well with the actual object. She had no difficulty with multiple object sequencing (i.e. taking a cigarette out of a pack, putting it in her mouth, lighting it, and then smoking).

Motor and sensory examinations were normal except for mild right-sided hyperreflexia with bilateral flexor plantars. A Technetium 99 brain scan showed an abnormal uptake in the left parietal lobe and a left carotid arteriogram revealed a mass lesion with abnormal vascularity in the left parietal lobe which on craniotomy was found to be metastatic carcinoma. The patient was treated with corticosteroids and radiation therapy in a hospital in her home city. Despite treatment the patient rapidly deteriorated and died. A post-mortem examination was not obtained.

Case 2.—A 64-year-old right-handed diabetic hypertensive female had sudden onset of aphasia. Examination on admission was unremarkable except for some signs of mild cardiac failure. On neurological examination the patient had an anomic aphasia, a mild recent memory defect, finger agnosia, dyscalculia, right-left confusion, constructional dyspraxia, with dysgraphia without dyslexia. In addition, the patient demonstrated an apraxia which was quite similar to Case 1. When asked to perform movements to commands with her non-paretic limb, she would make either irrelevant movements or look perplexed and would ask the examiner to show her how to do it. She would, however, be able to demonstrate verbally that she understood the command and her imitation of movement and use of actual object was excellent. The remainder of neurological examination revealed a very mild hemiparesis with hyperreflexia. An EEG showed left posterior hemispheric slowing and a brain scan was normal. The patient's condition remained stable and she was discharged.

Case 3.—A 56-year-old right-handed female with a history of rheumatic fever at age 18. Because of progressive heart disease she had a commissurotomy six years previously followed by a mitral valve prosthesis three years before admission. She had chronic atrial fibrillation and two years ago following a fall while on anticoagulants, she had a small left subdural hæmatoma which was evacuated. Post-operatively, she had a mild transient anomia. On the night of admission the patient had sudden onset of a right hemiparesis and aphasia. Although she initially had a mild comprehension difficulty, she gradually improved except for a persistent anomic aphasia, an agraphia, and a constructional apraxia. She also demonstrated a severe apraxia which was similar to Cases 1 and 2. Although she had intact comprehension of language, she was unable to perform useful movements to command with either limb. Her imitation of movements and her ability to use objects was excellent. The remainder of her neurological examination revealed a right hemiparesis, a right hemianopsia, right-sided hyperreflexia with a right-sided extensor plantar response. Her EEG showed left-sided slowing and Te^{99} scan was within normal limits. She was given anticoagulants and when seen one year after her ictus had no residual symptoms.

Discussion

Unlike the errors made by patients with IMA (Goodglass and Kaplan, 1963) (i.e. body part as object, clumsy movements, gestural enhancement, vocal overflow and pantomimed context), the patients described herein demonstrated movements which were often irrelevant and appeared as if they did not understand the command.

These patients did show both verbally and by picking the correct movement from a multiple choice that they did understand the command. The disorder was present with intransitive as well as transitive movements and involved the face as well as the extremities. Unlike patients with IMA who frequently fail to improve on imitation, imitation here was flawless. Furthermore, in contrast with descriptions of other patients presumed to demonstrate IA because they were unable to handle actual objects (De Renzi *et al.*, 1968), in the patients described here actual object handling was flawless.

Two of these patients had infarction, one from thrombosis and the other from embolism. The third patient had a neoplastic lesion in the left parietal lobe. Although the anatomical locus of the lesion was not pathologically identified in the two patients with infarction, these patients had many of the signs frequently associated with dominant parietal lobe disease.

While the anatomical locus for the production of this apraxia appears to be the dominant parietal lobe, the mechanism responsible for this apraxia remains unclear. Another form of IA was described by Pick (1905) and Marcuse (1904) in which patients could not perform a series of complex separate movements to commands. Marcuse (1904) suggested that the defect in this apraxia was a memory defect with loss of "goal concept." This mechanism (i.e. loss of goal concept) does not appear to be important in our cases. Although our cases had mild memory disturbances and anomia, they had difficulty not with series of movements but with isolated movements, and they could always verbally demonstrate the goal concept. In addition, it is felt that although the aphasic disturbance is similar to the apraxic disturbance, neither can be completely explained as a simple memory defect because when these patients were given multiple choice type questions, they always produced the correct answer.

It appears then that these patients can verbally understand commands for movements and that they can adequately maintain these commands in their immediate memory. In addition, their excellent imitation and use of actual objects suggests that the engrams for motor sequences are intact. What may be at fault in this form of IA is the process or processes which occur between language comprehension and motor encoding. After the command is understood there could be a mechanism which helps select the motor engram which best fulfils the command. Wernicke (1895) stipulated that before performing willed movement the goal is imaginatively anticipated and that the anticipation of the movement produces the desired kinetic effect by evoking the proper kinæsthetic engram. One may consider that the defect in these cases of IA is a defect in the verbal mediated motor sequence selector which is responsible for choosing the correct motor sequences or the connexions from language decoding areas to this selector or from this selector to the motor engrams. The loss of memory for motor sequences or the inability of these memories to reach the motor cortex would then cause IMA.

Another perhaps less mechanistic and hierarchical explanation may be similar to Goldstein's explanation of anomic aphasia, with which these cases of IA were

associated. Goldstein (1948) recognized that naming to confrontation was a symbolic act and that patients require an abstract attitude to perform this act. In a similar manner, the loss of the ability to pantomime and gesture are also symbolic acts and may be caused by loss of the abstract attitude. Unfortunately, however, this explanation does not help us understand the defect in neural processing which occurs in this disorder.

Summary

Ideational apraxia (IA) has been defined by certain authorities as a defect in the actual handling of objects. Others have felt that a defect in actual handling may represent a severe form of ideomotor apraxia (IMA) and a recent investigation supports the latter hypothesis. IA has been considered by some as a defect in the performance of a series movement; this is considered by others, however, to be a defect in memory.

Three anomic aphasia patients had what is probably a true IA because unlike the patients with IMA who perform to command poorly but with appropriate motions, these three patients responded to command by looking down at their hands, appearing perplexed and either saying they could not do it or else making a completely irrelevant movement. They could, however, demonstrate verbally that they understood and also would choose the correct movement when presented in a multiple choice fashion. Unlike the patients with IMA who improve slightly on imitation, these patients performed flawlessly. The evidence is that all these patients had a lesion in the dominant parietal lobe. They were able to use actual objects well and perform a series of movements with these objects.

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