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Neuropsychological Analysis of a Case of Reduplicative Paramnesia*

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ABSTRACT

Following a right cerebral hemispheric CVA, a patient showed extensive reduplication of place (his hospital) and occasional reduplication of person. His language was essentially intact, but he was deficient on tests of conceptualization and on nonverbal tasks, both visual and auditory. Short- and long-term memory for nonverbal but not verbal material were also impaired. However, he had sufficient "memory" for the reduplicated hospitals to comment that they were spatially identical. He simply seemed unable to treat these memories as representing the same phenomena. Neuropsychological analysis suggests that reduplicative paramnesia represents a combination of perceptual and memory deficits as well as impaired ability to integrate information.

Reduplicative paramnesia, a term first used by Pick (1903), is a condition in which the patient asserts the presence of two or more places with nearly identical attributes, while only one exists in reality. Weinstein and his associates (1952, 1955) have provided extensive descriptions of reduplicative phenomena in general and have argued that they are not linked to any particular neurological syndrome or deficit and that, however bizarre appearing, they are qualitatively similar to normal behavior.

More recent investigators, however, have attempted to explain possible cognitive and/or neuroanatomical bases for reduplication. Benson, Gardner, and Meadows (1976) reported three cases occurring as a result of head trauma. Each patient showed a period of posttraumatic amnesia followed by complete or nearly complete recovery of memory, while they continued to mislocate the hospital to a place significant to them in their earlier lives. The fact that the patients showed

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reduplication at a time when they were no longer amnesic suggested that the confabulatory condition could not be due to poor memory function *per se*. Benson et al. considered that a defect in integrating new information into a body of existing knowledge might account for the particular problem these patients had in incorporating their experience of hospitalization following a period of trauma and confusion. An hypothesized deficit in the synthesis of new into old information has subsequently been offered as the critical feature of reduplicative paramnesia (Staton, Brumback, & Wilson, 1982), yet such a deficit, Benson et al. suggest, would not be sufficient to maintain the condition unless the patient were, in some sense, "able" to ignore the overwhelming evidence that his or her localization of the hospital was incorrect. Finally, Benson et al. raise the question of why the information that is not being properly integrated is limited to the location of the hospital. Presumably, they argue, this restriction in the field of reduplication might be caused by an underlying visual-spatial deficit, while the patient's inability to use frequently provided information that his or her localization of the hospital was incorrect suggests a deficit in the ability (or perhaps, the motivation) to resolve conflicts. These conjectures are supported by the authors on a neuroanatomical rather than a neuropsychological basis. Results of neurologic examination indicated that all three patients had right-hemisphere damage, providing a basis for visual-spatial deficits, while two showed evidence and one, suggestions of frontal involvement, damage which might impair the ability to critically analyze the conflicting information with which they were confronted.

Although Benson et al. did not provide neuropsychological evidence to corroborate their conjectures, more extensive neuropsychological analyses of patients with a similar syndrome, reduplication of person, have subsequently been presented (Alexander, Stuss, & Benson, 1979; Staton et al., 1982). These single-case studies reported that their reduplicative patients had deficits in visual-spatial functions, visual nonverbal memory, conceptual identification, and in the planning and organization of behavior; deficits consistent with both right-hemisphere and frontal-lobe involvement. A recent report of a patient with striking reduplication of place, however, complicates the picture. This patient showed evidence of an isolated right parietotemporal infarction, along with frontal-lobe atrophy, but he showed a gradual remission of his reduplicative symptoms, even though the evidence of frontal-lobe atrophy remained unchanged (Vighetto, Aimard, Confavreux, & Devie, 1980). If the frontal-lobe damage was responsible for the patient's inability to use information concerning his incorrect localization of the hospital, it is not clear how his symptoms remitted in the presence of continuing evidence of frontal-lobe involvement.

The problem in attempting to interpret reduplicative paramnesia as a cognitive deficit occurring as a result of single or combined focal brain lesions is that one must first precisely identify the nature of the deficit or deficits associated with the syndrome. Staton et al. (1982) have most explicitly asserted the hypothesis that the critical deficit underlying the reduplicative phenomenon represents a primary

amnesia, a defect in the ability to integrate new, properly registered information with past memory. They have minimized the contribution of visual-spatial impairments to the syndrome and considered the *memory* deficit (their italics) to represent a disconnection between the sites of old and new memories. Their explanation, however, does not appear to account for the problem raised by Benson et al. (1976), i.e., how to explain the restriction in the field of reduplication. Exactly how such a bizarre syndrome is produced in such an apparently small proportion of patients requires further understanding of the functional nature of the deficit. The present report concerns a case of a patient who exhibited reduplication of place who was given a detailed neuropsychological evaluation in order to determine what underlying deficits were present that might account for his highly interesting symptoms.

MEDICAL HISTORY

The patient was a 66-year old man who had lived his entire life within the region of his present hospitalization. He was a high-school graduate and had worked as an inspector and foreman on an assembly line until his retirement at age 64. He had led an active and healthy life, and his past medical history was negative with respect to any neurologic problems. Shortly prior to his presenting illness, he was hospitalized with transient syncope. Neurological examination as well as skull X-rays, nucleotide brain scan, and EEG were negative. He was asymptomatic at the time of discharge. Two weeks later he experienced an acute onset of slowed and slurred speech, urinary incontinence, and left-arm weakness. He was taken to the hospital, where he was found to be conscious and able to obey simple commands. He showed a mild left-sided weakness with hyperactive left knee jerk and a left Babinski sign. Minimal diagnostic studies were carried out. His spinal fluid was xanthochromic, but other laboratory findings were within normal limits. He was diagnosed as having suffered a right-hemisphere CVA and, after a month, was transferred to a rehabilitation hospital, where he remained for approximately 6 weeks until discharged to his home in the care of his wife.

In the rehabilitation hospital he was first described as alert but disoriented and confused, with dysarthria. He showed a nearly complete flaccid left hemiparesis with left facial weakness, impaired position sense on the left, and gross neglect of the left side of extrapersonal space. He required assistance with bathing and dressing and was considered to have difficulty in monitoring his errors. During the course of his hospitalization he showed a considerable degree of recovery. Within 4 weeks he was able to bathe and dress himself with some verbal guidance. He showed little improvement with respect to his left hemiparesis, however, and he continued to show evidence of left-sided neglect.

REDUPLICATIVE PARAMNESIA

During his stay in the rehabilitation hospital, initial psychological evaluation demonstrated extensive evidence of the patient's reduplication of place. From the time he was first seen for evaluation (about 2 months following the onset of his stroke) until his final testing session over 7 months later, he continued to provide vivid descriptions of hospitals, all with the same name, usually located in northeast Ohio and northwest Pennsylvania, areas familiar to the patient. He explained some as having particular functions, e.g., that in E- was for "therapy". Often his descriptions were detailed and "convincing", e.g., one was "in the vicinity of an old farm territory off of Fairchild Road in K-, near the railroad tracks on the left, a sizable place, built on the same plan as everything else". Several were located in exotic places, one a "... floating hospital", harbored at a lakeport city, another in "... a foreign country, in the Far East" (where his son had worked). These memories occasionally included reduplications of persons as well, as when he mentioned that the floating hospital was operated by a person whom he afterwards identified as his roommate in the hospital, and by another person, a friend who had died over 10 years ago. He showed no indication of other types of reduplication.

Perhaps the most striking feature of his reduplication was the strength of these false memories. On several occasions he spontaneously commented on the inconsistencies they created. On one occasion he commented that in two separate hospitals they were laying fresh tar on the roof, which he considered "... too much of a coincidence, that two facilities could have fresh tar". He also could not understand how "they are all laid out the same" or how he got from one hospital to another. In spite of the patient's acknowledgement of such discrepancies, which "... leads me to agree with you" (i.e., that there was only one hospital), he persisted in accepting his multiple memories and even seemed to be adding to the details up to the time he was last evaluated. One was left with the impression that he might at any moment produce yet another hospital. He was clearly perplexed by the discrepancies between his memories and the reports of others. He expressed concern that no one agreed with him, frequently requested a map to demonstrate that there was more than one hospital, and was relieved when he was encouraged to speak openly of his memories. This reaction to the inconsistencies between his memories and his perceptions was similar to the responses of the patient reported by Alexander et al. (1979) and quite different from those seen in patients with gross confabulations of memory and little or no insight into their problem (Stuss, Alexander, Lieberman, & Levine, 1978). It was as if his judgment were intact yet simply not applied to the problem. In other respects he appeared thoughtful, organized, and systematic in his conversation and reasoning.

During the same period of time in which he persisted in maintaining and producing reduplications of the hospital, he showed numerous instances of acquiring new information about both the hospital and other events that transpired

at home following his discharge. He was able to recall accurately events that occurred during his hospitalization and relate these events to one another, to information that he possessed prior to his illness, and to information he subsequently acquired. Thus his reduplications of the hospital and its associated activities remained a highly restricted domain.

NEUROPSYCHOLOGICAL TESTING

The patient was first seen for psychological testing approximately 3 weeks following his transfer to the rehabilitation hospital. He was subsequently tested at home following discharge during the 3rd, 4th, and 9th months of his illness.

Verbal Abilities and Language

He was above average verbal ability, with a WAIS V-IQ of 112. His auditory comprehension was excellent. Although his reading of paragraphs was initially handicapped by his left visual neglect, by the 4th month of his illness, he was reading complex material without difficulty, though he was still showing signs of a left upper quadrantic field cut on confrontation testing and evidence of left visual neglect on other tests. He was quite fluent in spontaneous speech, but his ability to produce words beginning with a given letter of the alphabet was somewhat low (40th per centile) for a person of his level of verbal ability. There was no indication of problems in word finding or confrontation naming. Arithmetic skills were normal, and he was not apraxic; however, he occasionally tended to use a body part as an object when asked to demonstrate the use of an object in its absence. In only one respect did he show clear evidence of verbal problems. He had considerable difficulty integrating complex verbal material in a coherent and systematic fashion. When asked to describe complex pictures or to relate well-known fairy tales, he tended to give rather sparse, poorly integrated narrations, although in response to questions, he was always able to provide more complete information. This deficit suggested the syndrome of dynamic aphasia (Luria, 1980).

Conceptualization and Categorization

On all tests involving abstract reasoning he showed signs of moderate to severe deficits. He initially had great difficulty in matching items by such simple principles as color, form, and number on the Wisconsin Card Sorting Test. By the 9th month poststroke he was able to match by the principles of color and form but was still moderately impaired on this task. On the Columbia Mental Maturity Scale he had great difficulty in selecting from one of four or five pictured items the one that was not conceptually related to the others. He was able to solve only those items requiring the completion of a simple visual pattern on the Raven Progressive Matrices and did quite poorly in solving problems that required analogical reasoning. Test results strongly suggested the presence of frontal-lobe involvement.

Nonverbal Abilities

His worst performances were consistently observed on nonverbal tests. His lowest scores on the WAIS were on the Block Design and Object Assembly subtests, and his overall PIQ was 84. Perhaps his worst performance was seen on the Porteus Maze Test, where he achieved a Test Age of only 4-1/2. He had gross visual-spatial deficits. Simple visual designs were copied in a distorted and primitive fashion, although his approach to the copying task was logical and well-organized. His copy of the complex figure of Rey omitted the left side of the design. His left-sided neglect, however, did not impair his performance in bisecting horizontal lines, a task on which he was quite accurate. When motor elements were removed from a spatial construction task, as on the Hooper Visual Organization Test, he continued to perform at a deficient level. Color perception was intact. Tests of right-left discrimination were carried out accurately, and he was able to orient to the compass points when asked which way he would be facing if he was facing one direction and then made a series of right or left turns.

Nonverbal deficits were not limited to the visual modality. He showed left-sided problems on tasks of stereognosis, grip strength, and speed of finger oscillation. He performed at chance level on the Seashore Rhythm Test, and he had moderate difficulties in judging the emotional content of a message from tone of voice. Thus, the patient's extensive nonverbal deficits strongly suggested widespread right-hemisphere brain damage.

Attention and Memory

The patient showed mild difficulties on tests of concentration and short-term memory. His span of attention for both verbal and nonverbal material was slightly deficient. He was able to repeat only three digits in reverse order, and serial addition was carried out with some errors. On a Brown-Peterson task, in which he was required to recall simple stimuli after performing an interfering task for 15 s, he achieved normal results with verbal material but did less well with nonverbal, visual material.

On tests of long-term memory he showed no evidence of a global amnesic syndrome. His memory for prose passages, both immediately following their presentation and after a 45-minute delay was somewhat sparse, but when questioned he readily produced more information, a performance which was suggestive of his dynamic aphasia, discussed above. On a paired associate learning task, he learned and retained verbal material at a normal level. He also performed at a normal level on a test of episodic memory using the Krovitz technique. He was normally oriented with respect to person, time, and place, except, of course, for his reduplication of place.

In contrast to his intact verbal memory, the patient's nonverbal long-term memory was clearly deficient. His delayed recall of complex visual designs was quite impaired, though somewhat improved by the 9th month of his illness. He was never able after many trials to learn the correct path through a stylus maze

while blindfolded. His memory of well-learned nonverbal information, acquired prior to his illness, was relatively intact. He was able to reproduce a floor plan of the living room of his house with only a few minor errors in the placement of furniture. When asked to indicate the location of principal cities on a U.S. map without features designating state boundaries, he correctly placed only those cities that were clearly located on the circumference, with gross misplacements of such cities as Detroit, Minneapolis, Washington, Pittsburg, and Cleveland (!). These misplacements, however, were probably a function of his visual-spatial problems rather than the result of his impaired nonverbal memory. Interestingly, he had no difficulty in recognizing pictures of familiar faces, including faces of hospital staff with whom he had become acquainted only following his illness.

Emotional Adjustment

There was no evidence that the patient was suffering from any emotional difficulties prior to his admission to the hospital. During his hospitalization he was consistently described as alert and cooperative, with a capacity for working in tasks for a considerable length of time without complaint or loss of interest. He was quite aware of his deficits, including his left-sided weakness and neglect, such that he made active attempts at compensation and was able, for example, to read quite readily by the time of his hospital discharge. His results on personality testing fell generally within normal limits, as illustrated by his MMPI code, 9/3 58 4-17206/ F-K/L:. The MMPI results indicated that the patient was a relatively active, though not strongly assertive individual who was somewhat inclined to deny psychological concerns. Thus, while he by no means showed a picture of a conversion hysteric adjustment, he was not particularly insightful or psychologically minded. Furthermore, he seemed somewhat dependent on others for emotional support. Subsequently at home he became more overtly depressed, spent long periods of time sitting with his head lowered, complained of physical problems, and expressed the feeling that he was useless and that people had given up on him. Thus, the patient appeared much like those patients of Weinstein and Kahn (1955) who did not explicitly deny their illness but had considerable need for security. These patients did not deny or rationalize their symptoms but were dependent, depressed, and frank in their declaration of their needs and feelings. In fact, the patient seemed to become most overtly unhappy during the period in which he was showing the greatest degree of intellectual recovery, although his reduplicative problems persisted unchanged.

DISCUSSION

The results of the neuropsychological evaluation of this man with reduplicative paramnesia are quite consistent with those of previous patients showing evidence of reduplication. In general his verbal abilities, including measures of verbal

intelligence, language ability, and memory, were quite intact. He was severely impaired on a number of visual-spatial tasks, particularly those requiring constructional ability, and he showed deficits on nonverbal tasks outside the visual modality. His nonverbal memory was impaired, and he had great difficulty with tasks requiring conceptual ability and abstract reasoning. While neurologic evaluation of the patient was limited, the overall results of both neurological and neuropsychological examination were consistent with extensive right cerebral dysfunction. Unfortunately, neuroradiological procedures were not performed, and the extent of his lesion is uncertain. The point of the present paper, however, is not to examine the underlying neuroanatomic basis of the reduplicative phenomenon but to address the question of the nature of cognitive disturbance that produces the syndrome. Benson et al. (1976) have posed the basic difficulties facing our attempts to explain reduplicative paramnesia: (1) Why was the memory defect restricted to the location of the hospital?, and (2) Why was the patient able to ignore the considerable evidence that his memories were false, in spite of the fact that he recognized their inconsistency with respect to both his own perceptions and those of others?

It has been argued (Benson et al., 1976) that visual-perceptual disturbance is a necessary though not sufficient condition for the occurrence of reduplication. That is, the patient who no longer has intact visual perceptions is in some sense at risk for the development of reduplicative memories. Indeed, as Weinstein, Kahn, and Sugarman (1952) have reported, the reduplication was by no means totally restricted to the location of the hospital. The patient reduplicated persons associated with the hospital and added the confabulated memory of an old friend, now dead, as a person in charge of one hospital. Furthermore, not only the visual aspects of the hospital were reduplicated but the smells (the odor of fresh tar) and sounds of the hospital were reduplicated as well. In this respect it is important to note that the patient showed numerous nonverbal perceptual defects beyond those relating to visual perception. The multisensory nature of his reduplications combined with the multimodal nature of his impairments is consistent with assigning an important role to the perceptual disturbance as a necessary basis for reduplication. It is also important to note that other patients with reduplication have also been reported to have had nonverbal impairments aside from the visual modality (Alexander et al., 1979; Staton et al., 1982).

The role of the patient's nonverbal memory problems is less clear. Staton et al. (1982) have emphasized the memory problem as basic to the reduplicative syndrome. Several investigators have argued that the memory problem consists of an inability to integrate new acquired information into the patient's previously existing store of knowledge (Benson et al., 1976; Staton et al., 1982). The present patient, though clearly having nonverbal memory problems, was able to acquire a considerable amount of new nonverbal information. He recalled not only the names but the appearances of hospital staff. His topographic memory was sufficient to enable him to comment on the identical layout of the floor plans of the

numerous hospitals that he remembered. Furthermore, he seemed able to take much of this new information and incorporate it into his existing knowledge. He was able to recall quite normally the present authors' identities, the circumstances in which he met them, and relate them and their actions to his previous and present life. In short, he by no means showed a total inability to integrate new verbal or nonverbal information into his past memories. Furthermore, his nonverbal memory, though still impaired by the time of his latest testing, was considerably improved compared to his results during the first few months of his illness. Yet up to the last time he was seen, he persisted in his reduplications.

Benson et al. (1976) have suggested that the post-ictal confusion associated with the acute onset of symptoms may create some special relevance for those pieces of information acquired at that time. The present patient was transferred from his initial hospital to the rehabilitation setting (the focus of his reduplications) at about the time he began to resume a normal level of alertness, though he remained "confused" for a few more weeks. Was his recovery then sufficient to enable him to acquire new information, faulty though his perceptions might be, yet not sufficient to ward off false or uncertain memories about the location of the hospital? The problem with this explanation is that, as he continued to improve, to the point where his nonverbal memory and even his conceptual and abstract abilities, though still impaired, were considerably closer to the normal level than before, he continued not only to maintain his reduplications but to produce new, or at least previously unmentioned, false memories. Thus one must account for his continuing to develop reduplications during a period of considerable recovery at home, yet the reduplications remained restricted to the location of the hospital.

Finally, how is one to account for the patient's inability to reject the false memories in the face of so many indications that they were wrong? The patient's condition was by no means analogous to the condition of anosognosia. He was able to point out the low probability of the coincidental conditions in the various hospitals. When the patient was being interviewed, he never insisted that he was anywhere but in the hospital which everyone acknowledged to truly exist. He seemed to have the knowledge sufficient to enable him to reject his false memories, yet he steadfastly held to them, in a manner quite similar to the patient of Alexander et al. (1979).

In many respects the best description of the nature of the problems experienced by the present patient is that he had difficulty in integrating information, *per se*. At times he was able to acquire new information and integrate it with both other new information and old knowledge. In his reduplications he failed to integrate new information, sometimes with other newly acquired information, sometimes with old information. Finally, on tasks that did not involve either memory (complex picture description) or new information (relation of fairy tales) he had difficulty in integrating information. In other words, his reduplicative paramnesia may represent one aspect of the problem that also produced his dynamic aphasia.

The foregoing explanation of the neuropsychological basis of reduplicative

paramnesia thus posits three basic problems which account for the syndrome:

- (1) A perceptual problem, rendering certain types of received information of dubious quality and thus susceptible to reduplication.
- (2) A memory problem, rendering both the quality of certain memoranda and the resultant confidence which one has in one's memory suspect.
- (3) An integration problem, not restricted to memory, which lead poorly perceived stimuli held in poorly operating memory systems particularly likely to be improperly associated with another.

A fourth condition which is necessary for the expression of reduplicative paramnesia is that the problems listed above be only partial deficits. That is, the patient must be able to perceive, remember, and integrate information well enough to identify his surroundings and compare his perceptions to past memories. It is the partial nature of the deficits which renders his perceptions and memories suspect and promotes confabulation, and which accounts, in part, for the rarity of the phenomenon, i.e., most patients with lesions extensive enough to produce the three posited cognitive deficits are unlikely to be sufficiently intact to produce consistent reduplicative memories. In our experience, fragmentary reduplicative phenomena may be observed in dementia patients, but they are generally obscured by the extensive cognitive disorganization.

This explanation leaves open the question of the patient's emotional adjustment. While he was by no means significantly emotionally disturbed or classically hysterical, his adjustment in many respects was quite similar to that of a number of patients with reduplicative paramnesia or anosognosia (Weinstein et al., 1952). This similarity cannot be disregarded. It seems quite possible that certain types of adjustment may interact with the cognitive problems that have been suggested as being instrumental in the production of reduplication to increase the level of confidence with which the patient regards his false memories. Of course, the whole issue of the basis of the patient's adjustment is relevant to this consideration. Patients with frontal-lobe involvement have often been reported to show adjustments similar to that of the present patient (Blumer & Benson, 1975), so that it is conceivable that, if the patient did have a frontal-lobe lesion, it could account for both his integrative deficits and his unwillingness to reject his false memories, his, perhaps, rigidity.

The present explanation of the cognitive bases of the phenomenon of reduplication does not entirely coincide with the simpler explanations that posited a disconnection between new and old memories (Alexander et al., 1979; Benson et al., 1976; Staton et al., 1982). It suggests the importance of a more generalized deficit in the integration of all kinds of information, not simply new with old. It remains a question as to whether or not these hypothesized integrative deficits are a common part of most frontal-lobe syndromes (cf., for example, Luria, 1980) or a less frequent concomitant of frontal-lobe involvement whose relative infrequency in part explains the low frequency of reduplication. It seems more likely to us that the first hypothesis is correct and that the low frequency of reduplication is due to the necessity of the co-occurrence of perceptual and memory impairment with the

integrative deficit as well as to the stipulation that the deficits be partial rather than complete. A further question, of course, relates to the issue of whether or not such hypothesized integrative deficits were present in those other patients with reduplicative paramnesia. In view of the likelihood that such deficits are a common concomitant of frontal-lobe involvement, the present authors feel it likely that such is the case, but the issue at present is not resolvable. Certainly future cases of reduplication should be evaluated for the presence of problems with integration beyond simply failing to integrate new information with old. In any case, the present patient's deficit does not appear to be explainable on the basis of a simple deficit in integrating new and old learning.

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