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Is Seeing Believing: Notes on Clinical Recovery

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A persistent phenomenon in the neurological clinic is that patients who experience mild to severe cerebral damage and who show at the onset serious behavioral deficits usually recover some, if not all, of the lost function. While this is an enormous benefit to the patient, for which all are thankful, it is downright disconcerting to the neuropsychologist, who is trying to identify steady-state changes in behavior that are traceable to specific neurological lesions.

There have been several attempts at trying to understand this recovery process. The easiest and clearly the least controversial is the recovery seen as a consequence of reduced cerebral edema, when the function lost was simply a by-product of a state of transient swelling. This, of course, is a common event and holds little mystery. What is more perplexing is the recovery of function seen following cases of real tissue damage, loss, or disconnection. Our first observations on the phenomenon were from the last category and out of that came insights into the mechanisms active in the first two. In general, it is our belief that recovery almost invariably is the product of an alternate behavioral strategy being brought into play, with a patient in a sense solving a behavioral task by taking a different "road to Rome."

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1. CROSS-CUING IN THE SPLIT BRAIN

1.1. Somesthesia

One of the earliest observations of ours that suggested the importance of changing behavioral strategies to cover for an actual and persistent neurological deficit came from the way split-brain patients learned how to identify objects from a limited set held in the left hand. Immediately after commissurotomy, the patients were unable to identify objects of any kind in the left hand. In time, however, they became adept at identifying the objects if they were told that it was one of two items. Thus, if a ball and a square were given to the left hand and the patient had to say which was which, good performance was soon seen in the commissure-sectioned cases. This meant that somehow somatosensory information from the left hand was getting to the left speech hemisphere.

On the surface, one interpretation was that stereognostic information from the left hand was somehow making its way to the left hemisphere either through subcallosal pathways or perhaps through ipsilateral routes. It turned out, however, that neither of these possibilities was the case. Instead, the ipsilateral tracks coursing up to the left hemisphere could carry quantitative information (Gazzaniga, 1970), and the patients learned to deduce by presence or absence of stimulation, by duration of feeling an edge, and the like what one of the two objects it might be. If these same objects, however, were placed in a larger set and there was no limit on what the objects might be, their performance quickly fell to chance. In other words, when the left hemisphere knew what the objects were, it could deduce from quantitative cues available to it from the ipsilateral pathways which of the two was being presented on a given trial. It did not know them because of a stereognostic sense of what the objects were.

Working with the somatosensory system, then, we find a clinical–surgical case in which there seems to be recovery of function for the recognition of objects. Yet, by careful behavioral analysis, it turns out that the knowledge of the object comes from quite a different behavioral and neurological base than is normally used by the patient.

1.2. Visual Functions

Perhaps a more dramatic example of apparent recovery came from our results (Gazzaniga and Hillyard, 1971) on visual testing of the right hemisphere. In visual testing of patient L.B., we noticed in a particular training session that he was able to name one of two numerals

flashed to the right hemisphere from the speech center on the left. At first we felt that perhaps the subcallosal pathways were opening up and able to transfer simple perceptual information. This proved not to be the case, however, and a careful behavioral analysis revealed that what appeared as recovery was instead the appearance of a sophisticated behavioral strategy.

In presenting the simple numerals, we suddenly flashed L.B. a new numeral, the number 2. After the trial, he winced, looked at me, and said, "That's not a zero or a one," and I replied that he was correct, and told him that from here on in, we were going to present a series of numerals. To our great surprise, after a few trials, L.B. was beginning to name not only the zero and the one, but any numeral up to eight.

Again, at first glance, this was a remarkable shift in behavior, and we concluded that either the right hemisphere was capable of some simple speech or the left hemisphere was now a recipient of subcallosal information. Both hypotheses, however, proved to be grossly incorrect. Careful analysis of the reaction time to each of the numerals showed that L.B. took more time to respond to one than to zero, two than one, four than three, five than six, and so on. What we discovered he was doing was using a very sophisticated cross-cuing strategy (Gazzaniga and Hillyard, 1971). The left hemisphere commenced a count, and with that process there was a slight head movement. When the number flashed coresponded to the counted number, the right hemisphere signaled the left hemisphere by stopping the head. The left hemisphere observed this and said to the experimenter "four" or "three," or whatever the number might be on a particular trial. This was evident because when the subject was not allowed an indeterminate time to respond, scores on the presented information dropped to chance.

Again, what looked like a major change in behavior, with neurological implications for recovery, turned out to be a sophisticated behavioral strategy.

2. THE NEUROLOGICAL PATIENT

2.1. Disorder in Manipulo-Spatial Skills

It is my opinion that the same kind of process of what is essentially self-cuing goes on in more traditional cases of brain damage. For example, in a recent analysis of a patient with a large right parietal infarct, it became clear that the kind of recovery one sees in much of that syndrome was a function of switching behavioral strategies. In particular, in the first weeks after the infarct, the ability of the patient to con-

struct blocks, perform the Milner wire-figure test, and carry out other tasks usually thought to require proper right parietal lobe functioning was extremely poor, at best. Approximately 5 weeks after the infarct, however, the patient began to recover some ability to perform the block-design tasks, even though the performance was tedious and painfully slow, and in no way natural. The patient, who had a 139 verbal IQ, seemed to be putting the blocks together, not because of a return of the manipulo-spatial skills required (Le Doux, Wilson, and Gazzaniga, 1977), but by a verbal strategy and deduction, a skill that was clearly remaining to him. However, when the wire-figure test was used at the same point in time when he was performing better on the block design, his score immediately dropped to chance again. This task, which did not lend itself to easy verbal description, proved to be continuing to manifesting his deficit.

Our interpretation of these observations is that the actual deficit in manipulo-spatial activities produced by the right hemisphere lesion is persistent. The apparent return of abilities on the more verbalizable block test is a product of the verbal processor coming into play and assisting in solution of the task (Le Doux, Wilson, and Gazzaniga, 1978).

2.2. Disorders in Language

A frequent yet little-considered phenomenon in the neurologic clinic is that patients with dominant hemisphere disease frequently suffer from an anomia or dysnomia. This inability to find nouns is in marked contrast to the ability to use verbs. What is remarkable about this language disorder, as with many others, is that the deficit appears to recede. Recent experimentation of ours on a new split-brain patient offers us a clue on one of several possible underlying mechanisms that allows for this.

Verbal commands were presented to the right hemisphere of a split-brain patient (Gazzaniga, Le Doux, and Wilson, 1977) with a high language ability. The patient was able to carry them out. Thus, if the word "point" was flashed to the right hemisphere, the patient would point with the left hand. Under more routine conditions, words flashed to the right hemisphere that involve no discrete graphic motor response go undescribed because the speech center in the left hemisphere is disconnected from the right half-brain. In test situations like this, however, the patient, when asked what he saw, says "Oh, 'point.'" When the word "rub" was flashed, the patient rubbed the back of his head, and when asked what the command has been, he said, "Uh, 'itch.'"

What the patient is doing is this: The left hemisphere, watching the movement being produced and under the control of the right half-brain, then simply describes the action much as he would describe the action of another person. When the response is unequivocal, like pointing, his description is accurate. When it could easily be the result of a number of commands, such as words like "rub," he works at a chance level.

One can immediately see a possible parallel with the clinical problem of anomia. It is common experience to see that such a patient will be unable to name an object like a comb. Yet when the patient picks it up and starts to use it, and is asked "What are you doing?" he typically says "Combing—oh, it is a comb." When a paper clip is placed in front of the patient, he says he is unable to name it. When he is asked what he is doing, when he is using it, he says typically "Clipping papers—oh, a clip."

It would appear that the ability to name is a function of a stragegy. The item must be considered by its use, which then allows the object to become a verb, and verbs can still be accessed in these patients. The clever patient need not go through the actual action, but merely has to be asked how it is used.

3. SUMMARY

In example after example from the clinic, one can point to alternative behavioral strategies that seem to be active in covering for a neurological deficit. These clinical examples serve up fair warning that the improvement in function following neurological insult may not reflect recovery of function in neurological sense. They may reflect the ingenious ability of the organisms to maintain a behavioral status quo by using other mental and behavioral resources.

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