# Anosognosia in Parietal Lobe Syndrome

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Patients with right parietal lesions often deny their paralysis (anosognosia), but do they have "tacit" knowledge of their paralysis? I devised three novel tests to explore this. First, the patients were given a choice between a bimanual task (e.g., tying shoe laces) vs a unimanual one (e.g., threading a bolt). They chose the former on 17 of 18 trials and, surprisingly, showed no frustration or learning despite repeated failed attempts. I conclude that they have no tacit knowledge of paralysis (or, if such knowledge exists, it is not available for this particular task). Second, I used a "virtual reality box" to convey the optical illusion to the patient that she was moving her paralyzed left hand up and down to the rhythm of a metronome, and yet she showed no sign of surprise. Third, I irrigated patient BM's left ear canal with cold water, a procedure that is known to shift that patient's spatial frame of reference by stimulating the vestibular system. Surprisingly, this allowed her "repressed" memory of the paralysis to come to the surface; she said she had been paralyzed continuously for several days. I suggest that the vestibular stimulation produces these remarkable effects by mimicking REM sleep. These patients also employ a whole arsenal of grossly exaggerated Freudian "defense mechanisms" to account for their paralysis. To explain this, I propose that in normal individuals the left hemisphere ordinarily deals with small, local anomalies by trying to impose consistency but, when the anomaly exceeds threshold, an interaction with the right hemisphere forces a "paradigm shift." A failure of this process, in patients with right hemisphere damage, might partially account for anosognosia. Finally, I present a new conceptual framework that may help link several psychological and neurological phenomena such as Freudian defense mechanisms, vestibular stimulation, anosognosia, memory repression, visual illusions, anterograde amnesia, REM sleep, dreaming, and humor. © 1995 Academic Press, Inc.

The social scientists have a long way to go to catch up, but they may be up to the most important scientific business of all, if and when they finally get to the right questions. Our behavior toward each other is the strangest, most unpredictable, and almost entirely unaccountable of all the phenomena with which we are obliged to live.

Lewis Thomas

#### INTRODUCTION

In 1914, the French neurologist Babinski described an extraordinary neurological syndrome. He noticed that some of his patients, who were completely paralyzed on the left side of the body as a result of a right hemisphere stroke, tended to deny their paralysis and he coined the term "anosognosia" (denial of illness) to describe the condition. Anosognosia can vary in severity from a mere indifference to one's disability to a vehement denial of the paralysis, even when con-

fronted with incontrovertible proof. For example, if the patient is asked to perform a specific task with her paralyzed left hand, she may fail to do so but may continue to insist that she is not paralyzed. In its most extreme form, the patient may even deny that the arm belongs to her and ascribe it to either the examiner or to her spouse! (This disownership phenomenon was called "somato-paraphrenia" by Gerstmann (1942).)

One explanation for anosognosial would be in psychodynamic terms: the patient denies her illness in order to "protect her ego." This interpretation doesn't account for two important aspects of the syndrome. First, the syndrome is seen only when the right parietal lobe is damaged and only rarely when the left parietal lobe is involved (Critchley, 1966; Babinski, 1914). Second, the denial is often domain specific, e.g., the patient will admit she had a severe stroke but denies paralysis or, on occasion, will even admit that her leg is paralyzed but insist that her arm isn't. For discussions on these topics, the reader is referred to several lucid and insightful recent reviews (Galin, 1992; Edelman, 1989; Damasio, 1994; Heilman, 1991; Levine, 1990; McGlynn & Schacter, 1989; Halligan, Marshall, & Wade, 1993) as well as more classic papers in the older neurological literature (Critchley, 1962; Juba, 1949; Waldenstrom, 1939; Ehrenwald, 1930; Cutting, 1978; Weinstein & Kahn, 1950).

Another more "cognitive" interpretation of the syndrome would be in terms of the hemineglect-heminattention that often accompany the denial, i.e., one could argue that the patient neglects her paralysis in much the same way that she neglects everything else on the left side. This hypothesis is probably at least partially correct but it doesn't account for why the denial usually persists even when the patient's attention is drawn to the paralysis. Nor does it explain why the patient does not *intellectually correct* her misconception even though she may be quite lucid and intelligent in other respects. Indeed, the reason anosognosia is so puzzling is that we have come to regard the "intellect" as primarily propositional in character and one ordinarily expects propositional logic to be internally consistent. To listen to a patient deny ownership of her arm and yet, in the same breath, admit that it is attached to her shoulder is one of the most perplexing phenomena that one can encounter as a neurologist.

What would happen if the patient is repeatedly asked to perform an action with her left hand? I recently tried this on a patient, Mrs. LR (clinical details for this and other patients are described in a later section). I found that if her failure to perform were pointed out to her, she would usually "rationalize" her failure with statements such as, "My shoulder hurts a lot today; I have arthritis, you know," or "I didn't really want to point that time." After several such trials, however, she eventually admitted she was paralyzed. Yet, curiously, when questioned again just 10 min later, she not only reverted to denial—insisting that her left hand was fully functional—but also claimed that she had successfully used that hand during the preceding testing session (Ramachandran, 1994a,b)! This was

<sup>&</sup>lt;sup>1</sup> Throughout this paper I restrict the use of the word anosognosia to denial of hemiplegia, not to the generic use of the word to indicate denial of other types of deficits.

true despite the fact her memory for other details of that session was completely intact. It was almost as though she had "forgotten" or selectively repressed the memory of her failed attempts as well as her verbal acknowledgment of her paralysis. What is especially surprising about this observation is that it implies that her "propositional knowledge" system has no access now to the explicit verbal confession that she had been engaging in just a few minutes earlier (Ramachandran, 1994a,b). I realized, therefore, that these patients may provide an opportunity not only for studying denial, but also for studying the mechanism underlying the storage and retrieval of new memories.

Consider another patient BM, a 76-year-old lady who had a recent stroke that left her completely paralyzed on the left side. Although quite lucid when discussing most other topics, she persistently denied her paralysis even when pressed and her answers were not hesitant or lacking in conviction. The following conversation was quite typical:

Mrs. M, when were you admitted to the hospital?

I was admitted on April 16 because my daughter felt there was something wrong with me.

What day is it today and what time?

It is sometime late in the afternoon on Tuesday. (This was an accurate response.)

Mrs. M, can you use your arms?

Yes.

Can you use both hands?

Yes, of course.

Can you use your right hand?

Yes.

Can you use your left hand?

Yes.

Are both hands equally strong?

Yes, they are equally strong.

Mrs. M, point to my student with your right hand.

(Patient points.)

Mrs. M, point to my student with your left hand.

(Patient remains silent.)

Mrs. M, why are you not pointing?

Because I didn't want to. . . .

The same sequence of questions was repeated the next day with identical answers except that toward the end of the session the patient looked at me and asked:

Doctor, whose hand is this (pointing to her own left hand)?

Whose hand do you think it is?

Well, it certainly isn't yours!

Then whose is it?

It isn't mine either.

Whose hand do you think it is?

It is my son's hand, doctor.

One interpretation of this would be that the patient begins with rather simple

"denial" of paralysis but when her paralysis becomes increasingly obvious to her with repeated questioning she is pushed into a corner and the only way she can "rationalize" the failure of her arm to perform is to progress into the even more full-blown delusion that the arm belongs to her son! Later in this article, I will consider the hypothesis that what one is seeing here is a replay of the same kinds of delusions and rationalization that all of us engage in some time or the other (Ramachandran, 1994a,b). What is puzzling about these cases, however, is the extreme lengths to which they will take the process even though their intelligence, clarity of thought, and mentation is relatively unaffected in every other domain *except* for matters concerning the left hand. The patient I just described, for example, refused a box of candy saying, "I am diabetic, doctor—I can't eat candy. You should know that!" Thus, her anosognosia included her limb but did not extend to her diabetes.

Joseph (1993) has argued that the right parietal patient's statement that her left hand does not belong to her "makes sense" from her point of view since "she," i.e., the left hemisphere, no longer has access to either the sensory input or memories of the right hemisphere. (This seems implausible since it is very likely that most memories are bilaterally represented at some level.) In any event it can't be the whole story because it doesn't explain why she doesn't simply plead ignorance instead of engaging in confabulations or why she fails to intellectually correct her false belief even in the face of flatly contradictory evidence. For example, in the case of split-brain patients, even though the left hemisphere lacks access to information in the right hemisphere and is sometimes surprised by the actions of the "alien" left hand, the patient certainly doesn't rationalize this by saying, "This isn't my arm," or "This is my brother's arm. It looks big and hairy!" (And I would venture the prediction that if his left arm were to become accidentally paralyzed from a peripheral nerve lesion, he would admit rather than deny the paralysis.) Clearly, something more is involved; something other than just cloudy judgment or dementia, for right parietal patients are often perfectly lucid in other domains.

Mrs. M's excuse for not moving her hand ("Because I didn't want to") is, of course, a classic example of a Freudian rationalization. And, as noted earlier, Ms. LR, who also persistently denied her paralysis, had a whole arsenal of such "defense mechanisms" at her disposal that she used on different occasions. In addition to engaging in rationalizations, she frequently resorted to the use of euphemisms to describe her failure (anything to avoid the dreaded word "paralysis"). For example: "I've never been very ambidextrous, doctor," or "I guess it isn't very fast today, is it?" or, "My arm isn't facilitated." The patient may also use ingenious distraction maneuvers to take the emphasis away from the main thrust of the question. When asked whether she could use her left hand, another patient, Mrs. OS (see below), responded, "Did you know that my father was left-handed? So is my sister. . . . They were all left-handed—all of them," etc. And on other occasions, she produced confabulatory responses such as, "Yes, I can use my left hand. In fact, I used it this morning to wash my face," even though her memory for other types of events remained undistorted.

There has been a tendency in the past to regard anosognosia and somato-

paraphrenic delusions as a bizarre manifestation of cerebral disease. Contrary to this view, I suggest that what one is really seeing in these patients is an amplified version of Freudian defense mechanisms (Freud, 1946, 1895/1961) caught in flagrante delicto; mechanisms of precisely the same sort that we all use in our daily lives. However, since the defenses are grotesquely exaggerated, studying them might give us, for the first time, an experimental handle on defense mechanisms, i.e., we might actually be able to study the rules governing their development by manipulating the stimulus contingencies in individual patients (Ramachandran, 1994a,b). For example, it may help us answer an important theoretical question that was never addressed by Freud, namely, what determines which particular defense mechanism is used in a given situation (e.g., rationalization, projection, denial, reaction-formation, intellectualization, etc.)? To what extent is the choice determined by the particular environmental stressor and to what extent by the patient's premorbid personality?

There are, in fact, two important theoretical issues that we need to deal with. First, why do the Freudian defense mechanisms exist in the first place—even in normal individuals? Do they have a specific biological and/or social role? Second, why are these mechanisms grossly exaggerated in right parietal patients?

Before I answer these theoretical questions, however, I will describe three sets of experiments that we performed in patients with anosognosia. Although anosognosia has been recognized since the turn of the century, and there have been numerous valuable clinical case studies, there have been remarkably few *experiments* devoted to elucidating the nature and extent of the denial. One of the goals of my experiments will be to conduct formal experiments to determine whether the patient has "tacit" knowledge at some level that she is indeed paralyzed even though she denies it verbally. Additional goals will be to explore memory functions in these patients, e.g., if the paralysis is made evident to her, how long does she remember it? Is there a selective amnesia for paralysis? If so, does the amnesia result from "repression" or from failure to acquire the memory in the first place?

The answers to these questions will, in turn, set the stage for developing a new Darwinian theory of defense mechanisms in general and of anosognosia in particular. The rest of this article will, accordingly, consist of two parts. In Part I, I describe the experiments themselves and their immediate implications. In Part II, in keeping with the spirit of this journal, I will use these experiments as a starting point for putting forth a number of highly speculative ideas on such diverse topics as REM sleep, dreams, and laughter. I would emphasize, however, that the two parts are logically independent of each other. Since each part is self-contained, the reader whose main interest is in anosognosia can profitably skip Part II altogether and just focus on the experiments.

## PART I: EXPERIMENTS ON ANOSOGNOSIA

#### **Patients**

The four patients who participated in our study were elderly women who had recently sustained a right hemisphere stroke causing a left hemiplegia. No formal

neuropsychological tests (such as WAIS-R or CVLT) were administered but a routine neurological workup, including a mental status examination, was conducted on each patient. At the time when my experiments were conducted, they did not have any obvious signs of dementia, aphasia, or amnesia and were able to clearly understand our instructions. (LH and BM were sometimes somnolent and/or distractable but I tried to confine our experiments to lucid periods, when they were alert and willing to participate.) Whenever possible a CT scan and/or an MRI was otained.

#### Case 1

Patient LR, a 78-year-old, right-handed, Caucasian woman with 16 years of schooling and a degree in journalism, was admitted on March 13, 1994, after the sudden onset of loss of strength in her left limbs. The patient was alert, cooperative, and conversed fluently with the experimenters and hospital staff. There were no gross deficits in her memory and orientation (e.g., when I saw her on 2 consecutive days, she clearly recognized me the second time and even remembered the tests that were administered). Touch sensation was partially spared on the left side and clear signs of severe left hemiplegia were present. The patient had left hemispatial neglect, as seen in line cancellation and bisection tasks and also had right head and gaze deviation. She denied any motor or visual impairments, yet admitted she had come to the hospital for treatment of a stroke.

The CT scan performed at admission revealed a right frontoparietal CVA.

### Case 2

BM, a 76-year-old, right-handed woman with 3 years of schooling, was admitted April 8, 1994. The patient initially appeared slightly somnolent and easily distractible but was able to successfully communicate through a Spanish/English language interpreter. There were no obvious sign of aphasia or dementia but she did experience difficulty with serial subtraction. Touch and pain sensations were absent on the left side and she showed clear signs of severe left hemiplegia. Extreme left hemispatial neglect was evident from her line cancellations and bisections, and her head, gaze, and trunk were deviated to the right. The patient also denied having any motor or visual deficits and when questioned about the ownership of her left hand, she falsely ascribed it to either the experimenter or to her son, Tony.

An MRI obtained 1 month after admission revealed a large infarct involving much territory of the right middle cerebral artery especially in the right parietooccipital region. There was also an area of hemorrhage in the head of the caudate nucleus.

## Case 3

OS, a 65-year-old, right-handed, Caucasian woman, was admitted to the hospital on May 19, 1994. Three days after the stroke she was easily distracted and somnolent, but after a week she became more lucid, mentally alert, and personable. Her memory functions seemed intact but she exhibited some left-right con-

fusion. The patient demonstrated almost complete left hemiplegia with some limited preservation of sensations. Left unilateral neglect and denial of motor or visual impairments were present as well as somatoparaphrenic delusions. When questioned about her left arm, she reported that it belonged to either her son or to her husband.

A CT scan performed 5 days after admission revealed a right temperoparietal infarct.

#### Case 4

Patient FD was a 77-year-old right-handed female patient who developed a complete left hemiplegia following right hemisphere stroke. At the time when I saw her (8 days after her stroke) she had complete paralysis of left upper and lower limbs, no visual hemineglect (line bisection, line cancellation), and no visual extinction. Touch sensations were partially spared in her left hand, but she showed mild tactile extinction. Visual fields were apparently normal.

Patient FD was very alert and there were no obvious signs of dementia or aphasia; in fact, her intelligence seemed above average. She could do a serial subtraction by twos from 100 without difficulty and was clearly oriented in time and place. When questioned about her family, she provided detailed and accurate descriptions of her son and daughter. She also described the circumstances that led to her hospitalization and was aware she had had a stroke.

Despite the fact that she was mentally lucid, Mrs. D was densely anosognosic and denied her paralysis every time she was questioned. When asked whether she could point she insisted that she could (see under Experiment 2 for details). A CT scan performed on the day of admission showed an infarct involving the territory of the right middle cerebral artery and the right cerebellar artery.

# Experiment 1: Is There Tacit Knowledge of Paralysis in Anosognosia?

The patients described above repeatedly denied their paralysis, even on those occasions when they were asked to point with their left hand and failed to do so. Is it conceivable, however, that even though they deny their paralysis verbally, they are "aware" at some deeper level that they are in fact paralyzed? And if such tacit knowledge does indeed exist for what types of output is it available?

In my first experiment I confronted the patient with a choice between a unimanual task (e.g., stacking a set of blocks) vs a bimanual task (e.g., tying a shoelace) in a game-like atmosphere. She had to choose only one of these and successfully complete it to obtain a reward (see Table 1 for a complete list of unimanual/bimanual task pairs and corresponding prizes). Before each trial, she was first given careful demonstrations of both tasks and told that if she was able to complete one of them, she would be given the corresponding prize. She was also told that if she was unable to accomplish the task successfully, she would be given nothing. The combinations of bimanual and unimanual tasks were randomized, along with their prize pairs, but the larger or more valuable prizes were always coupled with the bimanual tasks and the smaller or less valuable prizes with the unimanual tasks.

	TABLE 1		
Complete List of Bimanual and	Unimanual Tasks	Used in the	Experiment

Tasks	Prizes
Bimanual	
Tie the laces of a baby shoe	\$5.00
Sew yarn around a small card	A ceramic angel
Tie a bow around the large box	A large box of candy
Use scissors to cut a paper circle	,
Unimanual	
Screw the nut onto the bolt (mounted on wood to remain perpendicular)	\$2.00
Stack five blocks	A bar of scented soap
Pick up objects with a clamp and put them into a bag	A small box of candy
Pick up a toy octopus with a fishing hook and put it into a cup	
Screw a lightbulb onto its holder	

Note. The tasks (left-hand column) were paired randomly with different rewards (right-hand column) on different trials.

Since all of the tasks were matched for simplicity, one would expect that non-paralyzed individuals would probably choose the bimanual task, in hopes of receiving the greater reward. (This was validated using two "control" subjects; see below.) Likewise, if the hemiplegic patients were completely unaware of their paralysis then, consistent with their denial, one would expect them also to choose the bimanual task. If, however, they had "tacit" knowledge of their paralysis, they might spontaneously choose the unimanual task.

#### Results

A total of 19 trials of unimanual versus bimanual choices were administered—8 to Mrs. LR, 6 to Mrs. BM, and 5 to Mrs. OS (Mrs. FD did not participate in this experiment). The experiments were done approximately 2 weeks (LR and BM) or 1 week after the stroke. The tasks (Table 1) were chosen randomly on different trials and there was usually a 5-min delay between trials. To Mrs. LR, 4 trials were given on the first day, 2 on the second day, and 2 on the last day and to Mrs. OS all trials were administered during a single testing session. To Mrs. BM the trials were administered on 3 separate days—2 trials on each day. The tasks, and the corresponding rewards, are described in Table 1. Two representative trials are described below in some detail.

Patient: LR. TASK CHOICE 1: TYING BOW AROUND BOX VS NUT AND BOLT. The patient was instructed to choose one of two tasks; tying a bow around a large box of candy to receive it as a prize or fastening a nut onto a bolt to win a small box of candy. (The bolt was fixed to a heavy base on the table so that it stood upright and could be easily threaded with one hand.) After hesitating for a few seconds, she decided in favor of the bimanual tying task. The explanation she

gave when asked about her choice was that the bigger box "would be easier to work with."

On the second trial with the same tasks and prizes, she immediately chose the unimanual nut and bolt task, but I soon realized that this was because the bowtying task was located too far in her left visual field, thereby causing her to neglect it. As a result, this trial was not included in the analysis. On all subsequent tasks I took the precaution of presenting the tasks entirely in the right visual field with the exact locations of the objects randomized on different trials.

On the third trial, also with the same task and prize pairs, the patient chose the bimanual option without hesitation. It is interesting to note that her explanation was again, "it seemed easier to work with," yet not even 5 min had elapsed since she had tried the very same task and failed.

TASK CHOICE 2: CUTTING CIRCLE VS NUT AND BOLT. In this trial the choice was between using scissors to cut out a paper circle (bimanual) for the large box of candy and fastening a nut onto a bolt (unimanual) for the small box. The patient reached for the scissors (bimanual) and attempted unsuccessfully to cut a paper circle (almost 2 min pass before the first question).

Exp: Why was cutting a circle difficult for you?

Sub: I don't know.

Exp: Try using your other hand.

Sub: Oh, I've never been ambidextrous.

Exp: How many hands does it take to tie a bow?

Sub: Two.

Exp: Can you tie a bow?

Sub: Yes.

Again, it appears that LR has either disregarded or forgotten her previously unsuccessful attempt with the bow-tying task.

Summary of results from individual patients. LR: On 7 of 7 trials, patient LR chose the bimanual task, slowly at first, though it took no more than 2 trials before she was selecting without hesitation. She continued making ineffective attempts to complete the task until interrupted by the experimenter.

BM: On 5 of 6 trials, patient BM chose the bimanual task, sometimes right away and sometimes after a pause. On one occasion (Task choice 2 in Table 1) she immediately chose the unimanual task, reporting that it somehow seemed easier for her.

MS: On 5 of 5 trials, patient MS chose the bimanual task without hesitation. Total for all subjects = 17 of 18 trials bimanual.

Control condition. I also repeated these experiments on two age-matched "control" subjects who had been admitted following an acute right hemisphere stroke producing a left hemiplegia. (The stroke was caused by an infarct in the territory of the right middle cerebral artery.) Both patients had clear signs of left hemineglect, including extinction and right gaze preference but neither of them had any trace of anosognosia. They were thus as close as possible to the experimental group as one could get.

I gave them the unimanual VS bimanual tasks to choose from, as for the experimental group, using identical procedures and instructions. On all 12 trials (8 trials

on one subject and 4 on the other) the patients spontaneously chose the unimanual task without any hesitation. At the end of the experiments when I asked them why they had chosen these tasks, they seemed surprised by my question and answered that they had done so because they couldn't use their left hand.

#### Discussion

We conclude that far from being a mere facade-like condition that leaves room for traces of insight to leak through, anosognosia runs deep. The patients either have no "tacit knowledge" of their paralysis or, even if they do, they cannot access this knowledge when choosing between a unimanual vs bimanual task. It remains to be seen, however, whether other types of tasks might allow the patient to access the knowledge that she is paralyzed. For example, what would happen if the patient were to try to lift a tray full of cocktail glasses? Would her right hand go toward the right side of the tray (as a normal person's might) or would it go straight toward the center of the tray? And what would happen if one were to videotape one of her failed attempts at the bimanual task and play it back to her immediately? Would this enable her to adopt a more abstract attitude toward herself and elicit a confession of paralysis (Ramachandran, 1994a,b) or would it precipitate what Goldstein (1940) has called a "catastrophic reaction?"

It is also important to emphasize, especially, that the patients neither showed obvious signs of *frustration* or distress while trying the bimanual task, nor showed any *learning* despite repeated failures, i.e., they seemed to have a selective amnesia for these frustrated attempts even though they recalled other details from the same session quite accurately. For example, during one testing session patient LR remarked that I had been wearing a "tie with pictures of brain scans on it" during the previous session; a fact that many of my students failed to remember. Even on the one occasion when she finally acknowledged her paralysis, she reverted to full denial when questioned again just a few minutes later! I shall return to this point in a later section.

My results also support Babinski's emphasis (1914) on the relatively normal mental status of anosognosics. He described them as being able to remember events and converse lucidly (without confusion, hallucinations, etc.). Like his patient, my three patients also had some subtle abnormalities in judgment and concentration but their denials and delusions were grossly out of proportion to these abnormalities. In any event, such subtle abnormalities of judgment cannot

<sup>&</sup>lt;sup>2</sup> I recently had the opportunity to try some of these experiments on patient FD. When confronted with a tray of cocktail glasses, her right hand went straight for the right side of the tray. For fear of losing the glasses I grabbed the other end of the tray with my right hand and we lifted it together. When asked whether she had raised the tray by herself she seemed surprised by the question and answered "Yes, of course."

I then wheeled her chair up right in front of a full-length wall mirror, so that she could clearly watch her own performance, and asked her to point to her own image in the mirror using her left hand. Seeing her failure to point had no effect whatsoever on her anosognosia; in fact, she insisted that she could clearly see her left hand in the mirror, pointing as it was supposed to. It remains to be seen how general this effect is. (I would emphasise that Mrs. FD had no visual hemineglect, no apraxia, no autotopagnosia, and no radiological evidence of left hemisphere damage).

possibly explain why the patient *consistently* chose bimanual task (instead of responding randomly), why they were able to describe the tasks to us clearly, and why they persisted in choosing these tasks despite repeated frustrated attempts.

## Experiment 2: The Virtual Reality Box

I will now describe a novel technique that may eventually prove useful in demonstrating "tacit knowledge" of paralysis in patients with anosognosia. The technique is similar to one originally developed for studying intersensory conflict in *normal* subjects (Nielsen, 1963), but I realized that it might provide a valuable tool for probing the depth of anosognosia in patients with right parietal lesions (Ramachandran, 1994a,b).

The "virtual reality box" was constructed out of cardboard and mirrors. The patient's gloved (paralyzed) left hand is inserted through a window in front of the box and she peeks into the box from a hole in the top to look at what she thinks is her own hand. Unbeknown to the patient, an accomplice inserts his gloved right hand through another opening in the box so that its mirror image is optically superimposed on the patient's left hand and she is "tricked" into thinking that she is looking directly at her own left hand. After being given some practice with the right hand, the patient is instructed to move her (paralyzed) left hand up and down to the rhythm of a metronome. The accomplice then moves his hand to the same rhythm so that the patient is "fooled" into thinking that her hand is indeed obeying her command! The question is would the patient be surprised to see her own left hand come to life even though she doesn't explicitly acknowledge the paralysis? The surprise might manifest itself explicitly (i.e., a verbal exclamation) or at least a change in the patient's expression and/or a galvanic skin response (GSR).

For practical reasons I was able to study only one patient (Mrs. FD) properly, using this procedure. She had sustained a right middle cerebral artery stroke with left hemiplegia and anosognosia (see under Patients for clinical details). Mrs. FD was seen by me on three successive occasions: 8 days, 10 days, and 15 days after her stroke. Even on the first session she spoke slowly but fluently and had no obvious signs of aphasia or dementia; in fact, her clarity of thought and mentation were exceptionally clear. She performed accurately on serial subtraction by twos and was able to give us detailed and accurate information about her family (e.g., her son's job and occupation). Furthermore, she had no hemineglect (line bisection and line cancellation), no visual extinction, and mild tactile extinction. Yet she was densely anosognosic, denying that her left hand was paralyzed. When I grabbed it, raised it toward her nose, and asked her whose hand it was, she said "It is your hand, doctor". (This cannot be ascribed to confusion; when I gripped my student's hand and held it under her nose she said "that is her hand" pointing to my student).

When asked whether she could point to my nose with her left hand she said she could. I then actually asked her to point and asked "Mrs. D, are you now pointing to my nose?" She said, "Yes I am." "Can you clearly see it pointing?" I asked. "Yes I can, it is about 2 inches from your nose," she replied.

Last I asked Mrs. D to clap her hands and she proceeded to do so with gusto,

making clapping movements with her right hand alone, as though "clapping" an imaginary left hand! When asked whether she was clapping successfully she said "yes," without hesitation. (Thus, we may, at last, have an answer to the Zen Master's riddle "What is the sound of one hand clapping?" Mrs. D. obviously knew the answer!).

During the following testing session I tried the virtual reality box on Mrs. FD. When she saw her left hand appear to move to the rhythm of the metronome she expressed no surprise whatsoever and when queried specifically, replied "yes I see it moving." I then repeated the experiment with her normal hand, asking the stooge to keep her gloved hand absolutely still. Mrs D then started to move her right hand up and down to the metronome, but the view afforded her inside the box was of a perfectly stationary hand (the stooge's). When she looked inside all she could see was a hand that looked perfectly stationary. Yet when questioned again, she maintained that she could clearly see the hand move up and down! This simple experiment demolishes all "neglect" theories of anosognosia since there was certainly no neglect of the right visual (or somatic) field and yet she was producing confabulations about her right hand! Clearly, what is critical is the presence of a discrepancy in sensory inputs; it is not critical whether the discrepancy arises from the left or from the right side of the body.<sup>3</sup>

# Experiment 3: Repressed Memories: Preliminary Evidence from a Single Case Study

The two experiments that we have discussed so far provided no evidence for "tacit knowledge" of paralysis in these patients. Is it possible, though, that the information is there but simply not accessible? And in the case of the unimanual/bimanual choice experiments, to what extent does the selective anmesia for the failed attempts arise because the relevant memory was never acquired in the first place, given the denial, and to what extent from "repression" causing a failure of retrieval? (This question can be asked of other types of amnesia too, of course, but is especially relevant to the amnesia for paralysis described here.) Certain remarks made by patient LR suggest that the latter explanation might be correct. For example, on one occasion when my student asked her (in my absence) about the previous testing session, she remembered, "That nice Indian doctor . . . he asked me to tie shoe laces," and added without being prompted, "I did it successfully using both hands" (my italics). The vehemence of this assertion is reminiscent of what Freud calls "reaction formation" and it obviously implies tacit

<sup>&</sup>lt;sup>3</sup> Similar confabulations occurred when the patient was asked to keep her right hand still while the accomplice's hand moved up and down to the rhythm of the metronome. This time the patient insisted that she was not seeing her hand move, that it looked perfectly stationary. We are still a long way from understanding the neural basis of such delusions, but the important recent work of Graziano, Yap, and Gross (1994) may be relevant. They found single neurons in monkey supplementary motor area that had visual receptive fields which were "superimposed" on somatosensory fields on the monkey's hand. Curiously, when the monkey moved its hand the visual receptive field moved with the hand, but eye movements had no effect on the receptive field. These hand-centered visual receptive fields ("monkey see, monkey do" cells) may provide a neural substrate for the kinds of somatoparaphrenic delusions I have seen in my patients.

knowledge of paralysis. For if she didn't have such knowledge, why would she actually volunteer the information that she used two hands—something that no normal person would do? Mrs. LR's frequent use of euphemisms when directly confronted with her paralysis also strongly suggests that she "knows" about her paralysis at some level (e.g., "I have never been very ambidextrous," "It isn't very fast today, is it?" or "I am unable to facilitate my arm," etc.).

Would it be possible, however, to demonstrate more directly that these patients do indeed repress unpleasant aspects of their experiences? A remarkable piece of evidence for such subconscious knowledge comes from another experiment we did on patient BM. The reader will recall that this patient denied her paralysis even upon repeated questioning and she finally asserted confidently that the arm belonged to her son. Now ordinarily, one thinks of this syndrome as arising from destruction or lesions in the right parietal lobe but, if this is true, how does one explain the fact that parietal lobe syndrome is one of those neurological disorders which show a very high rate of spontaneous remissions within a few weeks? One argument might be that as in the case of phantom limbs (Ramachandran, 1993), some other part of the cortex (e.g., the other hemisphere) might "take over" some of these functions but a remarkable discovery made by Bisiach and his co-workers (Bisiach, Rusconi, & Vallar, 1992) and by Cappa, Sterzi, Vallar, & Bisiach (1987) suggests an alternative, more exciting possibility. Bisiach et al. studied a patient who had sustained a right hemisphere stroke and was suffering from the delusion that his left arm belonged to someone else. They found, to their surprise, that when they stimulated the vestibular system by irrigating the patient's left ear canal with ice cold water, there was a complete disappearance of symptoms! (Unfortunately, a few hours after the caloric stimulation had worn off, the symptoms returned and the patient once again started denying ownership of his arm.) The important implication of this discovery is that denial and neglect may result from a temporary dysfunction of certain neural circuits in the right hemisphere, rather than from a permanent destruction of neural tissue.

I decided to try a caloric test on patient BM. After she had repeated several times that she was not paralyzed and her arm belonged to her son, I administered 10 cc of ice-cold water into her left ear and waited until nystagmus appeared. My main interest was not only in replicating Bisiach et al.'s observation, but also in specifically asking her questions about her *memory*, an issue that had never been studied directly before on a systematic basis.

- E: Do you feel okay?
- P: My ear is very cold but other than that I am fine.
- E: Can you use your hands?
- P: I can use my right arm but not my left arm. I want to move it but it doesn't move.
- E: (holding the arm in front of the patient) Whose arm is this?
- P: It is my hand, of course.
- E: Can you use it?
- P: No, it is paralyzed.
- E: Mrs. M, how long has your arm been paralyzed? Did it start now or earlier?

P: It has been paralyzed continuously for several days now.

After the caloric effect had worn off completely, I waited for ½ h and asked:

- E: Mrs. M, can you use your arm?
- P: No, my left arm doesn't work.

Finally, the same set of questions was repeated to the patient 8 h later, in my absence, by one of my colleagues.

- E: Mrs. M, can you walk?
- P: Yes.
- E: Can you use both your arms?
- P: Yes.
- E: Can you use your left arm?
- P: Yes.
- E: This morning, two doctors did something to you. Do you remember?
- P: Yes. They put water in my ear; it was very cold.
- E: Do you remember they asked some questions about your arms, and you gave them an answer? Do you remember what you said?
- P: No, what did I say?
- E: What do you think you said? Try and remember.
- P: I said my arms were okay.

These observations have several remarkable implications. First, they confirm Bisiach et al.'s (1992) observation about remission from anosognosia and delusion following caloric stimulation. Second, they also allow us to draw certain important new inferences about denial and memory repression. Specifically, her admission that she had been paralyzed for several days suggests that even though she had been continuously denying her paralysis, the information about the paralysis was being continuously laid down in her brain, i.e., the denial did not prevent memory consolidation (Ramachandran, 1994a,b). Part of this, of course, could have been simply a confabulatory "filling in" of the gap between her present knowledge of paralysis and the memory of her admission to the hospital but her use of phrases such as "continuously" or "several days" suggests that this was not the case. (And, in any event, the filling-in hypothesis does not explain why she should confabulate rather than simply plead ignorance or admit that she had been feeling normal prior to the caloric testing. In this regard, Mrs. BM's apparent lack of surprise at her own admission of paralysis is also noteworthy for it implies that she was now repressing the denial she had been engaging in just 10 min earlier!). We may tentatively conclude, therefore, that at some deeper level she does indeed have knowledge about the paralysis. Notice also that the insight gained during the caloric stimulation seemed to last at least for ½ h after the stimulation had ceased, but that when she was questioned again 8 h later, she not only reverted to denial, but also repressed the admission of paralysis that she had made during her stimulation!

Contrary to the frequently expressed view that memory repression is not a

<sup>&</sup>lt;sup>4</sup> On the previous day I had done the control experiment of irrigating Mrs. BM's right ear with water. This produced no remission from anosognosia whatsoever. Thus, the remission certainly cannot be attributed to nonspecific "arousal" produced by cold water.

real phenomenon (Holmes, 1990), my findings provide compelling experimental/ clinical evidence that it is indeed a robust psychological process. Furthermore, these results have important implications for our understanding of how normal memory processes work for they suggest that the separation of memory processes by black-boxologists into "acquisition," "consolidation," "storage," "recognition," "recall," etc., may be somewhat artificial and misleading. The reason Mrs. BM "forgot" her earlier admission of paralysis may be that it would have been very difficult for her to deny her *present* paralysis and yet admit the insight she had acquired 8 h earlier, while at the same time maintaining an integrated self. Instead of simply retrieving a file from the past, it was almost as though she was completely rewriting the script, for this was the only way she could avoid falling apart completely. We may conclude, therefore, that remembering something even from the recent past entails a reordering of one's conscious experience to accommodate current demands (Ramachandran, 1994a,b). Right parietal patients provide a valuable opportunity for seeing these phenomena in amplified form but it is probably something we all engage in when trying to remember something.

But when exactly does the "reconceptualization" of experiences and selective repression seen in these patients occur? Does it happen during consolidation or only during subsequent split-second retrieval? Surprising as it might seem, we have to consider the possibility that as soon as the cold water begins to act, the restructuring of experiences occurs almost immediately by altering synaptic weights to accommodate the patient's current ensemble of beliefs. So, in a sense, we are able to create two mutually amnesic conscious beings—the regular Mrs. BM whom we saw in the clinic every morning and the "cold-water Mrs. BM" produced by caloric stimulation.

# A Darwinian Theory of Defense Mechanisms

Why do psychological defense mechanisms exist? Freud's suggestion that they serve to defend the ego, although not incorrect, is too vague to be useful since it doesn't tell us why the ego needs to be defended, in the first place. On the face of it holding false beliefs actually seems maladaptive and we must therefore look for a functional or Darwinian explanation to account for their origin.

I suggest that the various defense mechanisms such as rationalization, repression, etc. arise because the brain/mind tries to arrive at the most *probable* and globally consistent interpretation of evidence derived from multiple sources. By way of analogy, consider a military general in a war room trying to make a major strategic decision, e.g., whether to invade a particular city at sunrise. He would ordinarily collect evidence from a large number of scouts, weigh the evidence, and arrive at a firm decision. Assume, for the sake of argument, that he has decided to launch the attack at dawn, drawn up his plans accordingly, and directed his soldiers to assume certain strategic positions. Now, if one scout were to show up 15 min before sunrise and provide information that was somewhat contradictory to the rest (e.g., he might indicate that the enemy had 10,000 soldiers rather than 5000), the general is unlikely to change his strategy. He may ask the scout to shut up and discard the evidence, and, for fear of mutiny, may even ask the scout to "march in tune" with the others and *lie* to the other officers

in the army. (The former would be analogous to "denial" and the latter to a "confabulation.") A perpetually indecisive general, on the other hand, would be quite incapable of winning a war.

An analogous process also seems to occur when the visual system tries to combine multiple sources of information about relative depth (e.g., perspective, stereo, occlusion, motion parallax, shading, etc.) to yield a vivid coherent impression of depth (Ramachandran, 1988, 1989). The rules that the visual system uses to combine multiple cues are poorly understood but may involve:

- (1) Taking a weighted average. The weighing is important because, statistically speaking, some cues might be inherently more reliable than others and this "wisdom" might be wired into the visual system during ontogeny and phylogeny.
- (2) Looking for consistency, e.g., if six cues yield random values and two yield identical values, then the visual system may "choose" the latter instead of averaging the values. We suggest that the reason for this is that accidental inconsistencies are relatively common in nature (due to noise), whereas accidental consistencies are extraordinarily rare.

Interestingly, once a global interpretation of depth has been reached, the system simply ignores or suppresses the conflicting information. (The purpose of this might be to avoid going into a perpetually indecisive state, i.e., the rule might be that any firm decision is better than none at all.) Now the remarkable thing is that the visual system may, on occasion, even "hallucinate" some of the required evidence in order to preserve consistency, i.e., there appears to be a tendency to actually *impose* coherence. It was as though the general had decided not only to ignore the advice of one of his scouts, but also insisted that the scout actually fabricate the evidence required in order to fit the "big picture." Indeed, he may on occasion even employ "yes men" for the specific purpose of cheering him along!

A striking example of this can be observed with the illusory square (Kanizsa, 1979) (Fig. 1) which is created by simply aligning four disks from which pieshaped sectors have been removed. What people usually see when viewing this display is an opaque white square partially occluding four back disks, rather than four disks that have been elaborately prepared and aligned by the experimenter.

Now it should be obvious that when seeing a square in this display, the visual system has to discard the contrary evidence from the homogeneous paper surface about the absence of an edge (for such an edge would usually be associated with a change of luminance). But instead of discarding the perception of a square, the visual system seems to opt for actually hallucinating an illusory edge, which even has an illusory brightness change associated with it. (In a connectionist model, this would be an example of "vector completion.") So here is a clear example of the visual system distorting the evidence in order to impose a coherent interpretation.

Now in my view, the same sort of thing happens in the cognitive/emotional domain. We have a tremendous need to impose a sense of order and coherence in our lives—we need a "story." Of course, when most of the evidence favors one particular interpretation of the available data, we have no difficulty in simply accepting that interpretation. For example, even when a patient's arm is para-

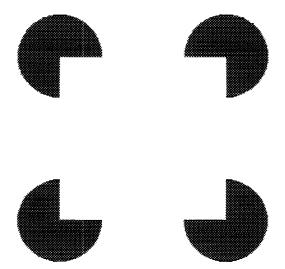


Fig. 1. An illusory square (Kanizsa, 1979). Subjects perceive an illusory white square occluding four black discs in the background rather than four black sectored discs. The illusory perceptual completion seen in these displays may be analogous to the confabulations of right hemisphere patients (see text).

lyzed, her motor cortex sends messages to her limb and there is a comparator in her brain that ordinarily monitors these feed forward signals and informs her "self" that "I am moving my limb." Therefore, her conscious self tentatively accepts this story. When the evidence is conflicting, however (e.g., if the patient's vision tells her that her arm is not obeying her commands), then instead of wasting time in conflict or oscillating between alternate decisions, her cognitive system simply picks one story and adheres to it. Again, in order to do this, it either ignores the conflicting evidence ("denial") or actually fabricates new evidence (rationalization). The evolutionary purpose of such "defense mechanisms" might be that when limited time is available, any decision, however uncertain, is better than an indecisive vacillation—so long as it is the best interpretation of the current data. But to deal with a conflicting source of information that keeps nagging away at the central processor, the latter may actually insert the relevant evidence so that it can go about the rest of its business. What you end up with, therefore, is a "rationalization" or a "denial."

<sup>5</sup> Of course, these mechanisms may have evolved originally in a social context, in conjunction with language. But they may have subsequently become internalised to provide cognitive and emotional stability even though they no longer serve an overtly social function. An interesting empirical question is whether anosognosic patients are equally anosognosic when there is no one watching.

A related question concerns the extent to which patients with denial of paralysis are aware of other people's deficits. I recently had the opportunity to explore this on another densely anosognosic patient, Mrs. LH, who had left hemiplegia following a right middle cerebral artery stroke. Mrs. LH insisted that she could see her left hand pointing to me, on command. I then asked her to watch another hemiplegic patient, in the adjacent wheelchair, while he attempted to point to my nose with

## The Anomaly Detector

And that brings me to my last point. The purpose of a "rationalization" we have seen is to eliminate discrepancy by creating fictitious evidence (or false beliefs). But clearly there must be limits to this process, for otherwise defense mechanisms would soon become maladaptive and threaten the individual's survival. It may be a good thing to repress an extremely traumatic memory in order to avoid being paralyzed with fear. This would be adaptive. It would be maladaptive, however, to repress every memory that was unpleasant since that would defeat the very purpose of having aversive memories in the first place. I suggest, therefore, that there is a special purpose mechanism—an anomaly detector—in the right parietal lobe whose sole purpose is to serve as a "Devil's Advocate" that periodically challenges the left hemisphere's "story," detects anomalies or discrepancies, and generates a paradigm shift if the discrepancy is too large. (You can think of this, if you like, as a mechanism for preserving intellectual honesty or integrity.) Hence, I might be willing to engage in some minor rationalization, i.e., make some small false assumptions to get on with my life but when the false beliefs become too far removed for reality, my anomaly detector kicks in and makes me reevaluate the situation (e.g., if I was a general about to wage war, it would be quite appropriate, usually, to ignore contrary evidence from a single scout, but if he told me that the enemy was waving a white flag or had nuclear arms, I would be foolish to adhere to my original decision). I suggest, further, that the mechanism for imposing consistency (i.e., the small rationalizations, repressions, etc.) is located in the left hemisphere whereas the "questioning" mechanism that monitors the level of discrepancy, discovers large-scale anomalies and reacts with the appropriate paradigm shift-including the appropriate emotion—is in the right hemisphere. This would explain why right hemisphere patients are willing to engage in much more elaborate and fanciful rationalizations than normal individuals or individuls with left hemisphere damage. Conversely, left hemisphere stroke patients may not be able to manage even a minimal amount of denial, rationalization, or confabulatory "gap-filling" and consequently become profoundly depressed. This would be a new interpretation of the common clinical observation that depression is most often associated with left hemisphere (especially left frontal) stroke.

I have not yet adequately dealt with the important question of why anosognosia is apparently *domain specific*; why some patients deny their paralysis and yet admit they had a stroke. One possibility is that this is really a threshold effect,

his left hand. When queried, Mrs. LH insisted that she could indeed see him point! Thus, in this one patient at least, the anosognosia seemed to extend to other peoples' equivalent body parts, while at the same time excluding her own deficits in other domains. This result implies that one may need to access one's own body schema even when making judgments about someone elses body parts. (I might add that Mrs. LH had no apraxia or autotopagnosia and no radiological evidence of left parietal damage.)

<sup>&</sup>lt;sup>6</sup> As with most other types of hemispheric specialization, what we are dealing with here almost certainly is *relative* rather than absolute specialization. J. Bogen (personal communication) has pointed out the dangers of "dichotomania."

i.e., the denial only appears to be domain specific but really isn't. The defenses employed by the left hemisphere may be applied selectively only when there is a disturbing discrepancy such as the patient's feeling that she can move her arm versus the verbal and visual feedback she receives informing her that she is paralyzed. The reason patient BM acknowledged her diabetes, however, may simply be because this wasn't quite as threatening to her complacency as being confronted by the immediate reality of her paralysis. A second possibility is there are indeed separate consistency-imposing mechanisms (or even "anomaly detectors") subserving individual domains such as one's body image. The generation of paradigm shifts in response to inconsistencies, on the other hand, may always require right hemisphere intervention. A more careful study of additional patients might help resolve these issues.

Finally, I emphasize that although the clinical evidence suggests the presence of an anomaly detector in the right hemisphere, the theoretical justification for having a separate mechanism for this purpose is unclear. One possibility is that the mechanism prevents the left hemisphere from becoming trapped in local minima by giving it a periodic jolt. The key difference between the two hemispheres, however, may be not that one hemisphere detects anomalies and the other doesn't, but that the way in which they deal with anomalies is very different, i.e., they have different coping strategies. When a small local anomaly is detected, the left hemisphere tries to impose consistency by ignoring or suppressing the contrary evidence, e.g., by Freudian defense mechanisms, but when the anomaly reaches threshold, an interaction with the right hemisphere forces a complete change in one's world view—a paradigm shift. (And, to that extent, the right hemisphere may be said to be more "sensitive" to anomalies.)8 Until we have a clearer understanding of the underlying neural mechanisms we must accept these metaphorical explanations as a temporary substitute. Meanwhile, if they suggest interesting new experiments, they will have adequately served their purpose.

## Summary and Conclusions

My purpose, in Part I of this article, has been mainly to present some clinical case studies as well as some novel experimental approaches to the problem of anosognosia. The results of these experiments are still very preliminary, but they have already allowed us to probe this syndrome a little more deeply than in the past. They complement the important work of Bisiach et al. (1992), Galin (1992), Critchley (1966), Heilman (1991), and others and serve to remind us of the important role that this enigmatic disorder is likely to play in any future discussions of consciousness and awareness of self—issues that have traditionally belonged to the province of philosophers.

<sup>&</sup>lt;sup>7</sup> Such exquisite domain specificity is, of course, not unique to anosognosia, and it shows up in many areas of neurology. What are we to make of selective loss of vegetable names with sparing of fruits? Or, of loss of inanimate object names but not of animate ones? Such findings pose a serious challenge for any theory of knowledge representation in the brain.

<sup>&</sup>lt;sup>8</sup> This notion is, of course, quite compatible with the traditional view that the right hemisphere sees the "big picture" or gestalt. Seeing the big picture may be a prerequisite for seeing an anomoly.

## PART II: SOME THEORETICAL SPECULATIONS

The previous section of this article was concerned mainly with some new empirical findings on patients with anosognosia, together with brief discussions on their theoretical implications. In this section, I would like to use these findings as a starting point to speculate on a wide range of topics such as dreams, humor, REM sleep, and psychoanalysis, topics that are of enduring interest to psychologists studying consciousness and representation of self. I might add that although these ideas are speculative, at least some of them lead to clear, testable predictions.

# Humor and Laughter: A Biological Hypothesis

Consider how essentially the same dichotomy between the two hemispheres may help explain another major biological puzzle—the origin of humor and laughter. Theories of humor and laughter go all the way back to Kant (1790) and Schopenhauer (1819), two singularly humorless German philosophers. Typically, humor involves taking someone up along a garden path of expectation so that the left hemisphere (in our scheme) is allowed to construct a story or model and then introducing a sudden unexpected twist at the end. Of course the twist is necessary but certainly not sufficient to generate humor; e.g., if my plane were about to land in San Diego and one of the engines failed unexpectedly, I would not regard this as very funny. The key idea here is that the twist has to be novel but *inconsequential*. Thus, we may regard humor as a response to an inconsequential anomaly.

Incongruity theories of humor have a long history (e.g., see Gregory, 1991). I would like to take these early ideas a step further by invoking hemispheric specialization and by proposing a specific explanation for the loud, explosive, stereotyped quality of the sound associated with laughter. I suggest that humor emerges when a dialogue between the consistency imposing tendencies in the left hemisphere and the anomaly detector in the right hemisphere leads to a premature paradigm shift. Imagine you are in a dimly lit room late at night and hear some annoying sounds. Ordinarily you interpret this to be the wind or something equally innocuous. If it gets a little louder you continue to ignore it, following the left hemisphere's strategy of ignoring evidence contrary to its preexisting model. But now the sound gets really loud and your right hemisphere forces a paradigm shift; you decide it must be a burglar and orient to the presumed anomaly. Your limbic system is activated so that you are both aroused and angry—preparing to fight or flee. But then you discover that it is in fact your neighbor's cat and so you laugh and harmlessly displace the emotion that has been built up.

But why laughter? Why the particularly loud, explosive, repetitive sound? Freud's view that then you discharge pent-up psychic energy doesn't make much sense without recourse to an elaborate hydaraulic metaphor. I suggest, instead, that laughter evolved specifically to alert others in the social group that the anomaly is inconsequential; i.e., they need not bother orienting. For example, if someone slips and falls and hurts herself you don't laugh; in fact, you rush to her aid. But if she doesn't get hurt then you do laugh (the basis of all slapstick

humor), thereby signaling to others that they need not rush to the fallen person's aid. Thus laughter is nature's "false-alarm" signaling mechanism.

Notice, however, that although this view explains the logical structure of humor, it doesn't explain why humor itself is sometimes used as a psychological defense mechanism. One possibility is that, jokes are an attempt to trivialize what would otherwise by genuinely disturbing anomalies. In other words, when an anomaly is detected, it is ordinarily dealt with by orienting or—when appropriate—by denial or repression but if, for some reason, it becomes more conspicuous and starts clamoring for attention, then an alternate strategy would be to pretend that it is a trivial anomaly by using a joke (i.e., you set off your own 'false alarm' mechanism). Thus, a mechanism that originally evolved specifically as an ethological signal to appease others in the social group, has now become internalized to deal with cognitive anomalies—in the form of a psychological defence mechanism. (Hence the phrase 'nervous laughter.')

# A Theory of REM Sleep and Dreaming: Nature's Own Virtual Reality

One of the most curious findings I have considered in this article is the remission of anosognosia and the reactivation of associated memories produced by caloric/vestibular stimulation. Why does vestibular stimulation produce these remarkable effects? I shall suggest three explanations that are not mutually exclusive.

- (1) The cold water may produce a nonspecific "arousal" of the right hemisphere, and this (in our scheme) allows the anomaly detector in the right hemisphere to become functional again. This hypothesis would be consistent with physiological work on animals that demonstrate a powerful cerebral activation (Fredrickson, Kornhuber, & Schwartz, 1974) during vestibular stimulation.
- (2) The vestibular stimulation may alter the patient's spatial frame of reference by allowing the right hemisphere to "orient" to the left side so as to eliminate the neglect of the left side of the body as. This may make the patient more "aware" of the left side of the body and thus, indirectly, more aware of the paralysis. The fact that we could induce Mrs. D to produce confabulations concerning her *right* hand argues against this interpretation (which is not to say that it doesn't contribute to the anosagnosia at least in some patients).
- (3) A third, more intriguing possibility is that the vigorous nystagmus itself is somehow causally linked to the chain of events that leads to the remission from anosognosia and the derepression of memories that we observed. This idea is not as farfetched as it sounds. After all, the link between eye movements and reactivation or "derepression" of unconscious memories can also be seen in dreaming associated with REM sleep and it is known that bursts of spontaneous vestibular neuronal activity occur in animals during REM sleep (Pompeiano, 1974). One way to test this hypothesis would be to elicit nystagmus in these patients using a rotating striped drum to see if there is any remission from anosognosia. Furthermore, the advent of new imaging techniques such as PET, MEG, and functional MRI might eventually allow us to determine the exact cause–effect sequence that underlies this extraordinary phenomenon.

Developing this theme a little further, I suggest that during ordinary waking

life the left hemisphere engages in "on-line" processing of sense data, including the temporal ordering of experiences and the imposition of consistency and coherence. This would necessarily involve the kinds of censoring, repressions, denials, and rationalizations that characterize most of our daily lives. In dream sleep, on the other hand, the mind/brain is allowed to tentatively bring some of the repressed memories out for an "improv" rehearsal on the main stage to see if they can be coherently incorporated into the main script without penalty to the ego. If the rehearsal doesn't lead to a stable organization, however, the material gets repressed again (unless you wake up accidentally, in which case it emerges in disguised form but is not incorporated into your psyche). But if it does work, then it gets incorporated seamlessly into the conscious self in the left hemisphere so that you personality becomes progressively more refined and less encumbered by unnecessary defenses. (This might explain why psychoanalysis is so notoriously difficult. What the therapist tries to do during wakefulness is precisely what nature has evolved to avoid during wakefulness and allows to occur only during REM sleep.) I suggest, therefore, that the eye movements generated by caloric stimulation in Mrs. BM may have produced their remarkable effects by emulating REM sleep and allowing a temporary "derepression" of her knowledge of paralysis in a manner analogous to what happens normally in dream sleep.

At the risk of pushing the analogy a bit too far, let us return once again to our general in the war room. Recall that during the day, in the heat of battle, he simply had no time to consider the contrary evidence from the single scout and therefore decided to shove it into a drawer marked "Top Secret. Do not open." It is now late at night and while relaxing over a glass of cognac he decides, "maybe I should take a second look at that file, after all, to see if I can incorporate into my battle strategy for the next day." Since he is not, at this time, actively engaged in making more important decisions, he can afford a leisurely contemplation of that file to see if its contents can be incorporated into his plans. And this, in my view, is exactly what happens each time you dream—you open your top-secret files and "psychoanalyze" yourself!

If this hypothesis is correct, one would also expect that in marked contrast to Freud's wish-fulfillment theory, patients with anosognosia should actually *dream* that they are paralyzed, e.g., if the patient is woken up during a REM episode, one might expect her to say, "I feel okay now. I can use both hands. But, you know, it's funny—I dreamed that my left hand was paralyzed." This counterintuitive prediction has never been tested directly but the experiment would be easy enough to do. (Recall, also, that in patient BM the effects of caloric stimulation continued for 30 min after the nystagmus had subsided. Hence, it is not inconceivable that for a short period after the patient is woken up from REM sleep, she might continue to admit her paralysis before eventually reverting once again to denial.)

My hypothesis on dreams is different from—although not inconsistent with—Hobson's (1988) well-known proposal that dreams are essentially an attempt to see meaningful patterns in "noise" generated by PGO activity. It is, however, similar to the ingenious suggestion of Winson (1986), who has postulated that dreaming involves a rehearsal and consolidation of both instinctive and learned

patterns of behavior. (It is noteworthy that Winson arrived at his theory starting from a completely different set of initial assumptions based on his physiological work on the hippocampus.) The key difference is that I invoke hemispheric specialization. Furthermore, in my scheme, an important function of dreaming is to allow the vestibular neuronal bursts that occur during REM sleep to selectively derepress disturbing memories so that you are given the opportunity to unburden vourself of unnecessary or maladaptive defenses. One can ask, of course, why the same goal cannot be achieved during wakefulness; why can't you simply try out the rehearsals when awake? It makes sense that you can't enact them literally since that might actually put you in physical danger, but, as pointed out by Jouvet (1975), this is neatly avoided by using a powerful barrage of inhibitory signals to completely paralyze all your somatic muscles during REM sleep. The reason you can't carry out these rehearsals in your *imagination*, however, is less obvious but three possibilities come to mind. First, during wakefulness the brain may be actively engaged in more important activities like our general in the war room. and daydreaming may simply be too costly an option. Second, for the rehearsals to be effective, they must look and feel like the real thing and this may not be possible when you are awake since you know that the images are internally generated. (As Shakespeare said, "You cannot cloy the hungry edge of appetite with bare imagination of a feast.") Indeed, during wakefulness the system that generates imagery may be temporarily disconnected from your limbic system so that imagery can never actually substitute for the real thing (which would make good evolutionary sense), whereas in dreams there may be limbic activation as well, in order to ensure verisimilitude. Third, and most important in our scheme, unmasking disturbing memories when awake would defeat the very purpose of repressing them in the first place and may have a profound destabilizing effect on the system, whereas unmasking them during REM may permit realistic and emotionally charged simulations (so realistic, in fact, that you may actually have an orgasm with your boss's wife and ejaculate during a dream—the so-called "wet dream"). Dreams may therefore be a way of "having one's cake and eating it too"—of reenacting highly realistic simulations without taking any of the associated emotional risks (in our scheme) or physical risks (in Jouvet's). They are nature's own virtual reality.

### A Hypothesis Concerning Classical Anterograde Amnesia

I will consider now how our ideas concerning motivational repression might also help explain bilateral mesial temporal lobe amnesia (Squire, 1987), a syndrome characterized by normal immediate memory (e.g., digit span), a profound anterograde amnesia (failure to acquire new "long-term" memories) and intact memories for events that preceded the onset of the disease (i.e., no retrograde amnesia). The currently popular view is that the syndrome results from a failure

<sup>&</sup>lt;sup>9</sup> In a disorder called cataplexy, the patient's somatic muscles become suddenly paralyzed in a manner analogous to the paralysis that accompanies in REM sleep, even though the patient is fully awake. Curiously, the attacks are often precipitated by either laughter or surprise, providing yet another link among dreams, REM sleep, and laughter.

of consolidation resulting from damage to the hippocampal system, rather than a failure of acquisition or retrieval. The old idea that amnesia (e.g., Korsakoff's amnesia) might result from "motivational repression" fell into disfavor because it fails to explain why even emotionally neutral memories (e.g., a list of words) are quickly forgotten, whereas the patient has no difficulty remembering the emotionally disturbing circumstances (e.g., alcoholism) that led to his hospitalization.

I would like to revive a modified version of the old view in the light of our hypothesis and suggest that the basis of the classical amnesic syndrome is a faulty hippocampal gating system that causes the patient to *indiscriminately repress* all memories rather than just disturbing ones. In other words, the memories are all there but they are locked away in the wrong drawers labeled "don't open." This would explain why the patient has no retrograde amnesia since the older memories were laid down before the repression mechanism became faulty. <sup>10</sup> In addition, it would also explain the preservation of "priming" effects and procedures or skills in these patients, since these do not require conscious retrieval and are, therefore, unlikely to be repressed. The "blocked-access" hypothesis makes two simple predictions: (a) That caloric stimulation might help the patient access some of the repressed memories that were apparently forgotten. (b) If woken during REM sleep, the patient might actually report some of the events that occurred during the day, i.e., his "day residues" may be just as vivid as that of normal nonamnesic control subjects. Such experiments would be illuminating whether one believed in the particular theory being proposed here.

If my general argument is correct, however, then the derepression of "lost" memories should be seen not only in amnesia but also during routine caloric testing of any neurological patient. One reason such effects have not been reported might be simply that vestibular stimulation only partially mimics the unmasking of memories that occur naturally during REM episodes. Consequently, the only memories that would surface would be the ones that were recently repressed, whereas long-lasting traumatic memories that have been strongly censored for many years may not be activated sufficiently to reach threshold.

# Summary and Conclusions

The theory of human nature that I have proposed in this article has much more in common with biologically based theories of cognition and perception (Crick, 1994; Edelman, 1989) than it does with the central tenets of classical AI. As we have pointed out in the past (Ramachandran, 1990), classical AI ignores the relevance of the neural machinery in the brain and the evolutionary history of the organism, both of which can provide vital clues to understanding the functional organization of complex biological systems such as the human brain. No engineer in his right mind, for example, could have foreseen a link between the inner ear and cognitive functions such as memory retrieval, dreams, or Freudian psychology.

<sup>&</sup>lt;sup>10</sup> An alternate formulation of this hypothesis would be that the hippocampal/limbic gating system is needed primarily for attaching a "value" to an object or event and in its absence, the default option is forgetting.

My purpose, also, has been to provide a new framework for linking several seemingly unrelated phenomena such as anosognosia, REM, amnesia, and laughter. I made two sets of key assumptions (A and B) that are logically independent of each other:

## Assumption A

- (1) The coping strategies of the two hemispheres are fundamentally different.
- (2) When a small incongruity is detected that doesn't fit the preexisting model or framework, an attempt is made to force a fit. In the cognitive domain this involves denial, rationalization, confabulation, dissociation etc. The purpose of doing this is to ensure consistency and stability of behavior and to avoid indecisive vascillation. (The argument here is that *any* decision, even a partially flawed one, is better than indecision.) Although these psychological defence mechanisms may have originally evolved in a social context they have now become internalized and provide emotional/cognitive stability at the individual level.
- (3) This particular coping strategy, i.e., discarding or distorting the evidence to preserve the status quo, is adopted largely by the left hemisphere (It is important to stress that one is probably dealing with partial rather than exclusive specialization.). I would argue that this is done not just for language but for cognitive consistency in general, although language is the most obvious external manifestation.
- (4) When the anomaly reaches threshold a "devil's advocate" in the right hemisphere intervenes and forces a paradigm shift, i.e., instead of discarding or distorting the evidence an attempt is made to question the status quo and to construct a new model. The paradigm shift also leads to a strong "orienting" and emotional response and to the production of a novel set of behaviors appropriate to the new model.
- (5) A dialogue between these two opposing tendencies of the two hemispheres may help account for many enigmatic aspects of human nature including the genesis of humor, laughter, and dreams.

## Assumption B

- (1) Vestibular/caloric stimulation eliminates not only anosognosia but also appears to reactivate (or "derepress") the patient's memories of her previous failed attempts.
- (2) Caloric stimulation may produce its remarkable effects by mimicking the reactivation (or derepression) of memories that is presumed to occur during REM sleep.

These two sets of assumptions (A and B) form the cornerstone of our theory and they lead to several novel predictions. I would emphasize, however, that even if one of these assumptions turns out to be wrong, e.g., the view that vestibular stimulation mimics REM sleep, the experimental predictions may still have some heuristic value since they do not depend *critically* on this assumption. For example, the question of whether amnesics remember their "day residues" during REM sleep would be interesting whether or not one believed the particular

theory being advanced here. And so, also, would be the outcome of the "virtual reality" experiment or the experiments attempting to elicit tacit knowledge or repressed memories in anosognosia. Indeed, these experiments might have implications for memory research that go well beyond their immediate implications for understanding anosognosia. As well as demonstrating, vividly, that memory is an active, constructive process (as emphasized by Edelman, 1989), the study of patients with this disorder may allow us to insert an experimental lever into the mental machinery that merges new memories seamlessly into one's preexisting conceptual categories. Such experiments would be especially easy to conduct in conjunction with the caloric-induced "reversible hyperamnesia" -- if that effect is confirmed on additional patients. However, since patients with this syndrome tend to recover spontaneously over a period of several days, the experiments could also in principle be carried out, even without the caloric testing, by simply interviewing the patient repeatedly about her memories as she gradually regained insight. 12 Finally, we would emphasize also that our ideas on the biological function of humor and dreaming depend only on the validity of the assumptions described under (A) rather than (B) and they can be tested directly by a careful study of patients with lateralized focal brain lesions.

Many individual fragments of this puzzle have been around for a long time, e.g., Gazzaniga's idea that in split-brain patients the left hemisphere has a language-based "interpreter" that provides a running commentary of events (Gazzaniga, 1992), Bogen's novel suggestion (Bogen, 1975) that the right hemisphere may be involved in "appositional thinking," that laughter involves a deflation of built-up expectation (Spencer, 1860; Kant, 1790), or that there may be specialized mechanisms for "reality checks" (Neisser, 1967) or "monitoring" (Galin, 1992). What I have tried to do, however, is to come up with a unified theory from a Darwinian perspective that can help link a number of seemingly unrelated phenomena, ranging from Freudian defense mechanisms and somatoparaphrenic delusions to REM sleep and laughter. The theory also makes several predictions that would be easy to test; indeed, some of them have already been partially confirmed. For instance, right parietal patients should have a diminished capacity for appreciating humor but paradoxically their remarks should seem funny to the examining physician (e.g., recall Mrs. LR's remark, "I was never very ambidex-

<sup>&</sup>lt;sup>11</sup> Virtual reality may turn out to be a promising technique for studying illusions of body image. We recently used the technique on a patient with a phantom arm to convey the visual illusion that his arm had been resurrected and was obeying his commands. Remarkably, 15 sessions (10 min each) of this "therapy," distributed over 2 weeks, resulted in a permanent "amputation" of the phantom limb with a disappearance of pain in the phantom elbow (Ramachandran, 1994b).

<sup>&</sup>lt;sup>12</sup> Such sequential interviewing of patients with anosognosia—to elicit memories—has rarely been done on a systematic basis. Four months after her repeated denial of paralysis and repeated failure with bimanual tasks (e.g., tying shoelaces), Mrs. OS had recovered completely from anosognosia and she now complained that her left arm was paralyzed. Her memory for various irrelevant details of the early testing sessions were quite vivid but when asked if she had always been paralyzed, she said "yes." When asked whether she remembered denying the paralysis, she said, "Well, if I was, I must have been lying and I don't usually lie."

trous"). Conversely, when the left hemisphere is dysfunctional, there might be a *diminished* tolerance for discrepancies or anomalies along with an inability to deploy psychological defense mechanisms.

Anomaly detection may be a prerequisite for seeing the paradoxial nature of certain visual illusions such as Penrose's impossible triangle. (Consistent with this prediction, we found that when one of the original Sperry-Bogen series of split-brain patients, LB, was shown the triangle either in the left or right visual field, he saw the anomaly only in the left visual field, i.e., when the right hemisphere was viewing the display.) Indeed, one could almost argue that our aesthetic appreciation of Escher's engravings arises from a dialogue or "tension" between the oppressing tendencies of the two hemispheres. And in the language domain, right parietal patients should have difficulty in interpreting fables such as the "sour grapes" story that illustrates the fallacy of rationalization. (The fox who couldn't reach the grapes said to himself, "Oh, the grapes are probably sour anyway.") It is known that literal mindedness is a characteristic of right hemisphere disease (Gardner, 1993), but to our knowledge no one has specifically tested their interpretation of fables or stories that illustrate Freudian defense mechanisms (as opposed to any fable or proverb).

My scheme would explain the curious delusions experienced by patient FD when she failed to see "her" right hand respond to her commands. When confronted with contradictory information from her different sensory systems, her left hemisphere tries to impose consistency by simply inserting the required evidence, i.e., the visual appearance of a moving right hand. Since she has a malfunctioning anomaly detector in her right hemisphere, this bizarre delusion goes unchecked and, consequently, she reports that she can actually see her hand moving even though this belief is contradicted by the visual appearance of a stationary hand. The result is so surprising, however, that it needs to be repeated carefully on a large number of patients.

Finally, my scheme also provides a way of linking vestibular activation with dreams, REM sleep, and anosognosia. Specifically, I suggest that the *overt* orienting that normally occurs during wakefulness in response to environmental disturbances or anomalies (including vestibular input) becomes replaced with *covert* orienting toward disturbing anomalous, memories that are derepressed in conjunction with eye movements during REM sleep. The involvement of the vestibular system in all this makes perfect evolutionary sense since it is part of a phylogenetically primitive mechanism that generates "orienting" in response to disturbances—either in the environment or in the body—receiving, as it does, information from many sensory systems. As the brain evolved and became increasingly sophisticated, perhaps it also developed a mechanism in the right hemisphere for monitoring *cognitive* anomalies and disturbances. If so, what better way to generate covert orienting in response to such cognitive anomalies than to hook it up to the vestibular orienting system that is already in place?

<sup>&</sup>lt;sup>13</sup> Physiological similarities between brainstem mechanisms that mediate orienting during REM and orienting during wakefulness have also been emphasized by Morrison (1979).

# Freud and the Inner Ear: A Neurological Approach to Psychotherapy

Talking to a patient with anosognosia can be an uncanny experience. Indeed the reason the disorder seems so peculiar to us is because it brings us face to face with some of the most fundamental questions that one can ask as a conscious human being: What is the "self"? What brings about the unity of my conscious experience? What does it mean to will an action? Such questions are often considered to be outside the scope of legitimate scientific enquiry and neuroscientists usually shy away from them. What I have tried to argue in this essay, however, is that patients with anosognosia afford a unique opportunity for experimentally approaching these seemingly intractable problems. As we have seen, they may even help us answer the eternal riddle "What is the sound of one hand clapping?"

Is it conceivable that there are mechanisms in the right hemisphere that are specialized not only for dealing with anomalies involving the corporeal self but also for dealing with other types of anomalies? According to classical psychoanalytical theory, many forms of neuroses arise because the patient's defense mechanisms have become so extreme that they have actually become maladaptive instead of being simply used as a coping strategy. Providing "insight" into these maladaptive defense mechanisms is, therefore, one of the goals of Freudian psychoanalysis and this is usually achieved by several months or years of intensive psychotherapy. But if I am correct in arguing that that "insight" can be enhanced using caloric left ear stimulation in patients with parietal lesions, is it conceivable that such insight could also be induced in neurotic patients who are neurologically intact, in order to eliminate the maladaptive defenses that are said to produce the psychoneuroses? (Notice that the efficacy of this would not necessarily depend on whether the denial is domain specific or not; all we have to assume is that caloric stimulation can also eliminate other types of anosognosia by activation of the right hemisphere—a proposition that can be tested experimentally.)<sup>14</sup> After all, in patient BM, caloric stimulation not only eliminated denial but also allowed the repressed *memory* of the paralysis to be overcome. What I am suggesting then (tongue-in-cheek!) is that the Barany chair may eventually replace the analyst's couch as a device for reviving repressed memories and for producing insight. 15 Obviously, these are highly speculative and tentative ideas but at least they are testable and they may not only point to new directions of research but also eventually suggest new ways to treat the numerous psychological ailments to which our species is notoriously prone.

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<sup>&</sup>lt;sup>14</sup> For example, would denial of cortical blindness or "Anton's syndrome" be temporarily relieved? And how about the distortions of body image that are said to accompany anorexia nervosa?

<sup>&</sup>lt;sup>15</sup> Soon after I wrote this essay, it was pointed out to me that there is now a fad therapy, popular among some clinical psychologists, that utilizes eye movements to enhance the patients "insight" and to uncover repressed memories. While I would ordinarily be inclined to dismiss this as bizarre, it makes perfect sense from the point of view of my theory!

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#### REFERENCES

- Babinski, M. J. (1914). Contribution a l'etude des troubles mentaux dans l'hemiplegie organique cerebrale. Revue Neurologique, 1, 845-848.
- Bisiach, E., Rusconi, M. L., & Vallar, G. (1992). Remission of somatophrenic delusion through vestibular stimulation. *Neuropsychologia*, 29, 1029-1031.
- Bogen, J. E. (1975). The other side of the brain. UCLA Education, 17, 24-32.
- Cappa, S., Sterzi, R., Vallar, G., & Bisiach, E. (1987). Remission of hemineglect and anosagnosia after vestibular stimulation. *Neuropsychologia*, 25, 755-782.
- Critchley, M. (1962). Clinical investigation of disease of the parietal lobes of the brain. Medical Clinics of North America, 46, 837-857.
- Critchley, M. (1966). The parietal lobes. New York: Hafner.
- Crick, F. H. C. (1994). The astonishing hypothesis. New York: Scribner's
- Cutting, J. (1978). Study of anosagnosia. Journal of Neurology, Neurosurgery, and Psychiatry, 41, 548-555.
- Damasio, A. (1994). Descarte's error. New York: Putnam.
- Edelman, G. M. (1989). The remembered present. New York: Basic Books.
- Ehrenwald, H. (1930). Verandertes erleben des korperbildes mit konsekutiver wahnbildung bie linksseitiger hemiplegie. Monatsschrift fur Psychiatrie und Neurologie, 75, 89-97.
- Farrah, M. (1991). Visual agnosia. Cambridge, MA: MIT Press.
- Fredrickson, J., Kornhuber, H., & Schwartz, D. W. (1974). In H. Kornhuber (Ed.), *Handbook of sensory physiology*, (Vol. VI, 1, pp. 565-583). New York: Springer-Verlag.
- Freud, A. (1946). The Ego and the mechanisms of defense. New York: International Universities Press.
- Freud, S. (1961). The standard edition of the complete works of Sigmund Freud (Vol. 1-23). London: Hogarth Press. (Original work published 1895)
- Galin, D. (1974). Implications for psychiatry of left and right cerebral specialization. Archives of General Psychiatry, 31, 572-583.
- Galin, D. (1992). Theoretical reflections on awareness, monitoring and self in relation to anosagnosia. *Consciousness and Cognition*, 1, 152–162.
- Gardner, H. (1993). In E. Perceman (Ed.), Cognitive processing in the right hemisphere. New York: Academic Press.
- Gazzaniga, M. (1992). Nature's mind. New York: Basic Books.
- Gerstmann, J. (1942). Problem of imperception of disease and of impaired body territories with organic lesions: Relation to body scheme and its disorders. Archives of Neurology and Psychiatry, 48, 890-913.
- Goldstein, K. (1940). Human nature. Cambridge, MA: Harvard Univ. Press.
- Graziano, M. S. A., Yap, G. S., & Gross, C. (1994). Coding of visual space by premotor neurons. Science, 266, 1051-1054.
- Gregory, R. L. (1991). Odd perceptions. New York: Routledge, Chapman, & Hall.
- Haligan, P., Marshall, J., & Wade, D. (1995). Supernumerary phantom limbs. *Journal of Neurology*, *Neurosurgery*, & *Psychiatry*, in press.

Heilman, J. (1991). In G. Prigatano & D. Schacter (Eds.) Awareness of deficits after brain injury. New York/Oxford: Oxford Univ. Press.

Hobson, J. A. (1988). The dreaming brain. New York: Basic Books.

Holmes, D. (1990). The evidence for repression: Examination of 60 years of research. In J. L. Singer (Ed.), *Repression and dissociation*. John, D. & Catherine T. MacArthur Foundation series on Mental Health and Development. Chicago, IL: Univ. of Chicago Press.

Joseph, R. (1993). The naked neuron. New York: Plenum.

Jouvet, M. (1975). The function of dreaming. In M. Gazzaniga & C. Blakemore (Eds.), *Handbook of psychobiology* (pp. 500-524). New York: Academic Press.

Juba, A. (1949). Beitrag zur struktur der ein- und doppelseitigen korperschemastorungen. Monatsschrift fur Psychiatrie und Neurologie, 118, 11-29.

Kanizsa, G. (1979). Organization in vision. New York: Praeger.

Kant, I. (1790). Kritik dev. Berlin: Unteilskraft.

Levine, D. N. (1990). Unawareness of visual and sensorimotor defects: A hypothesis. *Brain and Cognition*, 13, 233-281.

McGlynn, S. M., & Schacter, D. L. (1989). Unawareness of deficits in neuropsychological syndromes. Journal of Clinical and Experimental Neuropsychology, 11, 143-205.

Morrison, A. (1979). Brainstem regulation of behavior in sleep and wakefulness. In J. Sprague & A. N. Epstein (Eds.), *Psychobiology and physiological psychology*. New York: Academic Press.

Neisser, V. (1967). Cognitive psychology. New York: Appleton-Century-Crofts.

Nielson, T. I. (1963). Volition: A new experimental approach. Scandinavian Journal of Psychology, 4, 215-230.

Pompeiano, O. (1974). In H. Kornhuber (Ed.), *Handbook of sensory physiology* (Vol VI, 1, pp. 583-623).

Ramachandran, V. S. (1988). Perception of depth from shading. Scientific American, 269, 76-83.

Ramachandran, V. S. (1989). *The neurobiology of perception*. Presidential Lecture at the Annual meeting of the Society for Neurosciences (USA).

Ramachandran, V. S. (1990). Visual perception in people and machines. In A. Blake & T. Troscianko (Eds.), AI and the eye, Bristol: Wiley.

Ramachandran, V. S. (1993). Behavioural and MEG correlates of neural plasticity in the adult human brain. *Proceedings of the National Academy of Science U.S.A.*, **90**, 10413-10420.

Ramachandran, V. S. (1994a). How deep is the denial (anosagnosia) of parietal lobe syndrome? Society for Neuroscience Abstracts.

Ramachandran, V. S. (1994b). Phanton limbs, somatoparaphrenic delusions, repressed memories and Freudian psychology. In O. Sporns & G. Tononi (Eds.), *Neuronal group selection*. San Diego: Academic Press.

Schopenhauer, A. (1819). Die Welt als Wille und Vorstellung. Leipzig.

Spencer, H. (1860). The physiology of laughter, MacMillan Journals, Ltd., 1, 395-402.

Squire, L. (1987). Memory and the brain. New York: Oxford Univ. Press.

Waldenstrom, J. (1939). On anosagnosia. Acta Psychiatrica, 14, 215–220.

Weinstein, E. A., & Kahn, R. L. (1950). The syndrome of anosagnosia. Archives of Neurology and Psychiatry, 64, 772-791.

Winson, J. (1986). Brain and psyche. New York: Vintage Books, Random House.

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