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The Evolutionary Biology of Self-Deception, Laughter, Dreaming and Depression: Some Clues from Anosognosia

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Abstract — Patients with right hemisphere strokes sometimes vehemently deny their paralysis. I describe three new experiments that were designed to determine the extent and depth of this denial. Curiously, when asked to perform an action with their paralyzed arm, they often employ a whole arsenal of grossly exaggerated 'Freudian defense mechanisms' to account for their failure (e.g. 'I have arthritis' or 'I don't feel like moving it right now'). To explain this, I propose that, in normal individuals, the left hemisphere ordinarily deals with small, local 'anomalies' or discrepancies by trying to impose consistency in order to preserve the status quo. But when the anomaly exceeds threshold, a 'devil's advocate' in the right hemisphere intervenes and generates a paradigm shift, i.e. it results in the construction of a new model using the same data. A failure of this process in right hemisphere stroke would partially explain anosognosia. Also, our model provides a new theory for the evolutionary origin of self-deception that is different from one proposed by Trivers. And, finally, I use anosognosia as a launching-off point to speculate on a number of other aspects of human nature such as Freudian defense mechanisms, laughter, dreams and the mnemonic functions of the hippocampus.

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 the phenomena with which we are obliged to live.

Lewis Thomas

Introduction

More than one hundred years ago, in the year 1895, Sigmund Freud launched off his 'project for a scientific psychology' – a bold and ambitious manifesto for discovering the neurological underpinnings of human nature. Indeed, few people realize that the 'father of psychoanalysis' was originally trained as a neurologist. Since his real interest was in the mysteries of the human mind, however, he quickly became disillusioned with the minutiae of neuroanatomy, which he saw as being somewhat remote from his ultimate goal. (His first paper was on the spinal ganglia of petromyzon, a primitive fish!) As a student of the eminent

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French neurologist Charcot, he became interested in hysteria, hypnosis and eventually, of course, in practically all aspects of human nature; dreaming, human sexuality, religion, humor, laughter, psychoneuroses, anthropology, and even slips of the tongue. And although we are amused today by such ideas as penis envy or the oedipus complex, there is a real danger of throwing the baby out with the bathwater; of failing to recognize Freud's monumental contributions to human thought. His basic idea – that consciousness is simply the tip of the iceberg and that our behavior is mostly governed by a cauldron of emotions and motives of which we are largely unconscious ('the unconscious mind') – is still a perfectly valid concept that is sure to have a tremendous impact on both psychology and neurology. Modern reincarnations of this idea include such phenomena as 'blindsight' (45) or the elicitation of changes in skin conductance (GSR) in patients who have no conscious recognition of faces (9).

Even though Freud's own interests shifted from hard-core neuroanatomy to 'soft' psychology, he never lost sight of his initial goal of providing a neural explanation for psychological phenomena and he continued to pay lip-service to this goal until the end. Surely, had he been alive today, he would have been delighted with what we now know of the syndrome of anosognosia. For, as we shall see, this curious disorder may eventually allow us to anchor the airy abstractions of Freudian psychology in the physical flesh of the brain.

Anosognosia is usually seen in patients who have had a right hemisphere stroke, resulting in a paralysis of the left side of the body.* Some of these patients vehemently deny the paralysis and, in extreme cases (somatoparaphrenia), may ascribe the arm to another person such as a spouse or even the examining physician (5,8,9,11,16,21,22,26,27,38,45).

In this article, I will describe several new experimental approaches to studying patients with this syndrome. And in keeping with the spirit of this journal, I will use this syndrome as a launch-pad to present some highly speculative ideas on a number of seemingly unrelated aspects of human nature – such as Freudian 'defense mechanisms', laughter, dreams, depression, amnesia and creativity. Throughout the essay, my goal will be to provoke and challenge the reader; I certainly do not claim to have any definitive answers to these ancient riddles.

* Throughout this paper, I restrict the use of the word 'anosognosia' to denial of hemiplegia, not the generic use of the word to indicate denial of other types of deficits.

Anosognosia

The syndrome was first described at the turn of the century by the French neurologist Babinsky (1). Since then, there have been numerous fascinating clinical case studies of anosognosia but there has been a tendency to regard them as outlandish or bizarre. My goal, in this essay, will be to try to bring this syndrome into the domain of modern cognitive neuroscience and to argue that it raises some fundamental questions concerning the organization of the human mind.

As an illustration of this syndrome, consider the following conversation we had with one of our patients, Mrs FD. She was an elderly lady who had suffered a right hemisphere stroke eight days previously, resulting in a complete left hemiplegia. (Clinical details for this and the other patients we tested are described in Table 1). She was unable to move without a wheelchair and had no use of her left arm.

VSR: Mrs D, how are you feeling today?
FD: I've got a headache. You know, doctor, I've had a stroke so they brought me to the hospital.
VSR: Mrs D, can you walk?
FD: Yes. *(FD had been In a wheelchair for the past two weeks. She cannot walk.)*
VSR: Mrs D, hold out your hands. Can you move your hands?
FD: Yes.
VSR: Can you use your right hand?
FD: Yes.
VSR: Can you use your left hand?
FD: Yes.
VSR: Are both hands equally strong?
FD: Yes, of course they are equally strong.

This is quite typical of a person with anosognosia. At this point, I wondered what would happen if I kept pushing the patient further. I did so with some hesitation, for fear of precipitating what Kurt Goldstein has called a 'catastrophic reaction', which is simply medical jargon for: 'The patient becomes depressed and starts crying because her defenses crumble.'

VSR: Can you point to my nose with your right hand?
FD: *(She followed the instructions and pointed to my nose.)*
VSR: Mrs D, point to me with your left hand.
FD: *(Her hand lay paralyzed in front of her.)*
VSR: Mrs D, are you pointing to my nose?
FD: Yes.
VSR: Can you clearly see it pointing?
FD: Yes, it is about two inches from your nose.

At this point she produced a frank confabulation, a delusion about the position of her arm. She has no problems with her vision and could see her arm per-

Table 1 Clinical details of patients tested for anosognosia.

Patient	Age, sex	Handedness	Major neurological deficits	Site of lesions
LR	78, F	R	Left hemiplegia and severe neglect	Right frontoparietal CVA (CT scan)
BM	76, F	R	Left hemiplegia and neglect; somaparaphrenia	Right parieto-occipial infarct. (MRI) Small hemoragic infarct in the head of the caudare nucleaus, etc.
QS	65, F	R	Left hemiplegia	Right temperoparietal infarct (CT scan)
LH	77, F	R	Left hemiplegia and neglect	Right middle cerebral artery and left cerebellar artery infarct (CT scan)
FD	77, F	R	Left hemiplegia and severe neglect	Infarct in the territory of right middle cerebral artery and right cerebellar arteries (CT scan)
SM	76, F		Anterograde amnesia of sudden onset	Lesion not confirmed

Patients: The five patients who participated in our anosognosia study were elderly women who had recently sustained right hemisphere stroke causing left hemiplegia. No formal neuropsychological tests (such as WAIS-R or CVLT) were administered, but I conducted a routine neurological work-up, including a mental status examination, on each patient. At the time when our experiments were conducted, they did not have any obvious signs of dementia, aphasia or amnesia and were able to clearly understand my instructions. (MS, LH and BM were sometimes somnolent and/or distractible but I tried to confine my experiments to lucid periods, when they were alert and willing to participate.) Whenever possible, a CT scan and/or an MRI was obtained. The sixth patient (SM) developed all the classical signs of medial temporal lobe amnesia following a 'food-poisoning' episode but her MRI was apparently normal. She had no dementia or aphasia and her IQ was well above average. She participated in our dream-recall study.

fectly clearly, yet she produces a delusion about her own body image. (I had verified previously that she had no left hemi-neglect and also took the precaution of standing on her right side.)

VSR: Mrs D, can you clap?
FD: Of course I can clap.
VSR: Mrs D, will you clap for me?
FD: (*She proceeded to make clapping movements with her right hand as if clapping with an imaginary hand near the midline!*)
VSR: Are you clapping?
FD: Yes, I'm clapping. (*Thus, here at last, we may have an answer to the Zen master's eternal riddle, What is the sound of one hand clapping? Mrs D obviously knew the answer!*)

What we have seen in Mrs D is quite extreme. What is much more common in patients with anosognosia is a tendency to come up with all sorts of rationalizations to explain why the arm doesn't move; they don't usually say that they can actually *see* their arm moving. Consider the following conversation I had with another more typical patient, Mrs LR, who was also suffering from anosognosia. Mrs LR, like Mrs FD, had sustained a right hemisphere stroke causing the left side of her body to be paralyzed.

VSR: Mrs R, how are you doing?
LR: I'm fine.
VSR: Can you walk?
LR: Yes.
VSR: Can you use your arms?
LR: Yes.
VSR: Can you use your right arm?
LR: Yes.
VSR: Can you use your left arm?
LR: Yes, I can use my left arm.
VSR: Can you point to me with your right hand?
LR: (*LR points towards me with her right hand.*)
VSR: Point to me with your left hand?
LR: (*Her hand remains laying in front of her.*)
VSR: Are you pointing?
LR: I have severe arthritis in my shoulder, you know that doctor. It hurts.

These patients often come up with ingenious excuses and poignantly comical euphemisms in order to evade the main thrust of the question.

Student: How come you are not using your left arm.
LR: I've never been very ambidextrous, doctor.

There is a striking similarity between the strategies these patients use and what Sigmund and Anna Freud

have called 'psychological defense mechanisms' (13,14). These are mechanisms used by normal people when they are confronted with disturbing facts about themselves. Examples of such mechanisms are rationalizations, denials, repression of unpleasant memories and 'reaction formation'. I was struck by the fact that people suffering from anosognosia do the same things *but in a grossly exaggerated form* (35,37). For example, if a normal person endures a peripheral nerve damage and is left with a paralyzed arm, he may downplay the extent of his deficit ('Oh, I think I'll recover soon'), but he is unlikely to declare that the arm does not belong to him or that he sees it pointing about a centimeter from your nose. No normal person would ever carry the defense mechanisms to such absurd limits.

One interpretation of anosognosia would be in psychodynamic terms, i.e. the patient is confronted with something unpleasant – her paralysis – and therefore she plays it down or even denies it. This explanation does not work for one simple reason: this phenomenon is rarely seen when the left hemisphere is damaged, which results in right-sided paralysis. This paralysis ought to be just as unpleasant for the patient yet she rarely engages in denial. This asymmetry suggests that anosognosia is a *neurological* rather than a psychological syndrome. Indeed, the reason that anosognosia is so fascinating is precisely because it straddles the borderline between neurology and psychiatry; between brain and mind.

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.

†Contrary to what the term 'neglect' implies, the syndrome must surely involve much more than simply ignoring the sensory input from the left side of the world. To explore this experimentally, K. McClain, E. Altschuler and I recently asked a patient to turn her head to look into a mirror hanging on her right – parallel to the sagittal plane. Standing on her left (neglect) side, I then showed her a candy bar, the reflection of which was clearly visible to her. Remarkably, instead of reaching leftward to accept the candy, she repeatedly reached into the mirror – even though she knew it was a mirror – complaining all the time that the candy was beyond her reach. (Another patient even tried reaching behind the mirror!) Thus, even the patient's ability to make rational deductions had become selectively distorted to accommodate the strange new sensory world that she now inhabits.

The surreal logic of anosognosia

Several earlier attempts have been made to explain anosognosia but most of them are one variant or another of the two theories outlined above – the psychodynamic theory or the neglect theory. Here, I would like to propose a new approach to this syndrome. The correct way to formulate the problem, in my view, is to consider the two following theoretical questions:

1. Why do normal individuals have psychological defense mechanisms? I.e. why should they hold false beliefs about themselves?
2. Why are these mechanisms grossly exaggerated in anosognosia?

A new biological theory of self-deception: was Trivers right?

Freudian defense mechanisms are essentially *false beliefs* about oneself, but what possible benefit could holding false beliefs confer on an organism? Indeed, on the face of it, they actually seem maladaptive.

The most popular theory of self-deception, currently in vogue, is that proposed by the well-known evolutionary biologist, R. Trivers (43). According to Trivers, there are many occasions when a person needs to deceive someone else. Unfortunately, it is difficult to do this convincingly since one usually gives the lie away through subtle cues, such as facial expressions (12) and tone of voice. Trivers proposed, therefore, that maybe the best way to lie to others is to first lie to yourself. Self-deception, according to Trivers, may have evolved specifically for this purpose, i.e. you lie to yourself in order to enable you to more effectively deceive others.

Trivers' argument is ingenious and there is probably some truth to it. However, I don't find it entirely convincing. When you lie to someone else, your purpose is to withhold information that you don't want the other person to know. For example, suppose a chimp (Chimp A) sees where a zoo keeper places a big bunch of bananas. Chimp A now points Chimp B in the wrong direction, so that he can have all the bananas to himself. Now, following Trivers argument, suppose Chimp A wanted to make sure that Chimp B doesn't

detect the lie, he engages in self-deception, i.e. he really believes that the bananas are in the place that he points to. But if this were true, then Chimp A himself would also go look for the bananas in that false location. This would defeat the whole purpose of deception and be obviously maladaptive!

The real reason for the evolution of these defense mechanisms (confabulations, rationalization), I suggest, is to create a coherent belief system in order to impose stability in one's behavior. In order to understand this alternative hypothesis, one must invoke the idea of hemispheric specialization. Popular psychology acknowledges the idea of specialization, the left hemisphere is specialized for language while the right hemisphere engages in visual/spatial tasks. Yet there are also many overlooked differences between the two hemispheres. In particular, I suggest that each of us has a tremendous need to impose consistency, coherence and continuity on his/her behavior. In other words, we need a script – a thread of continuity in time. The left hemisphere is primarily responsible for imposing consistency on to the storyline and this would correspond roughly to what Freud calls the ego. At any given moment in out waking lives, our brains are flooded with a bewildering variety of sensory inputs, all of which have to be incorporated into a coherent perspective based on what stored memories already tell us is true about ourselves and the world (44). In order to act, the brain must have some way of selecting from this superabundance of detail and ordering it into a consistent 'belief system', a story that makes sense of the available evidence (41). When something doesn't quite fit the script, however, you very rarely tear up the entire story and start from scratch. What you do, instead, is to deny or confabulate in order to make the information fit the big picture (I prefer this to saying that you try to 'reduce dissonance', which is simply a meaningless tautology.). Far from being maladaptive, such everyday defense mechanisms keep the brain from being hounded into directionless indecision by the 'combinational explosion' of possible stories that might be written from the material available to the senses (37).

Let me illustrate this concept with an analogy. Imagine a military general about to wage war on the enemy. It is late at night and he is in the war-room planning strategies for the next day. Scouts keep coming into the room to give him information about the enemy. They tell him that the enemy has 500 tanks and that he has 600 tanks, so the general decides to wage war at sunrise the next morning. He positions all of his troops in strategic positions and decides to launch battle exactly at sunrise – at 06.00 hours. But imagine further that, at 05.55 hours, one little scout comes running in to the war-room with a report that says that the enemy actually has 700 tanks, not 500! What does the general do? A good general would ask the scout to shut up and instruct him not tell anyone about what had been seen. Indeed, he may even shoot the scout and hide the report in a drawer. In doing so, he relies on the probability that the previous scouts' information was correct and this new information coming from a single source is probably wrong. The likelihood seems to be small that this one source of information is right and so the general sticks to his original position. Not only that, but for fear of mutiny, he might tell the scout to *lie* to the other generals and tell them that he only saw 500 tanks, which would be analogous to a confabulation. The purpose of all of this is to impose stability on behavior and to avoid vascillation because indecisiveness doesn't serve any purpose. *Any* decision, so long as it is *probably* correct, is better than indecision. A perpetually indecisive general will never win a war!

By now the reader will have recognized that the general[‡] is in the left hemisphere and that his behavior is analogous to the confabulations and delusions of both normal individuals and patients with anosognosia. What we have to explain, however, is why these defense mechanisms are grossly *exaggerated* in the patients. And this is where the right hemisphere comes into the picture.

In order to understand what the right hemisphere is doing, we must push the analogy a step further. Suppose that, instead of the scout's report saying that the enemy has 700 tanks, the report said that the enemy had nuclear weapons. The general would be very foolish, if he adhered to the original plan. He must now abandon his previous plan and formulate a new one, for the consequences, if the scout is correct, are just too great. Once a certain threshold is reached, people must have a mechanism for revising their models completely, and that is where the right hemisphere comes into the picture.

The basic idea here is that the *coping strategies* of the two hemispheres are fundamentally different. The left hemisphere's job is to create a model and maintain it at all costs. If confronted with some new information that doesn't fit the model, it relies on Freudian defense mechanisms to deny, repress or confabulate; anything to preserve the status quo. The right hemisphere's strategy, on the other hand, is fundamentally different. I like to call it the 'anomaly detector', for

‡ The general is not unlike Freud's 'ego' or the language-based 'interpreter' (17) that has been postulated to account for the occasional rationalizations of the left hemisphere in 'split-brain' patients. What I have tried to make clear, however, is the *biological* rationale – in Darwinian terms – for having such a mechanism in the brain and I also postulate a complementary mechanism in the right hemisphere that serves as a 'counterbalance.'

when the anomalous information reaches a certain threshold, the right hemisphere decides that it is time to force the left hemisphere to revise the entire model and start from scratch. The right hemisphere thus *forces a Kuhnian paradigm shift in response to anomalies whereas the left hemisphere always tries to cling to the original model.*

In patients suffering from anosognosia, the left hemisphere is doing all of the confabulation and denial as it would in a normal person. The difference is that these patients have lost the mechanism in the right hemisphere that would ordinarily force them to generate a paradigm shift in response to conflicting information. This forces the patient into a delusional trap and he will continue to confabulate without switching paradigms. The patient may therefore glibly explain away *any* anomaly or discrepancy so that he is, on the whole, blissfully oblivious to his dire predicament.

I would like to add, however, a note of caution concerning hemispheric specialization. M. Kinsbourne, D. Galin, J. Bogen and others have warned us of the dangers of 'dichotomania' (15), i.e. of ascribing any given cognitive function entirely to one hemisphere or the other. (Even the 'mute' right hemisphere appears to be linguistically much more sophisticated than previously believed. (46)) We must bear in mind not only that the specialization is probably *relative* rather than absolute, but that the human brain also has a front and back, and an up and down and countless other subdivisions, not just left and right. Thus, my idea is that the right hemisphere is a left wing revolutionary that generates paradigm shifts, whereas the left hemisphere is a die-hard conservative that clings to the status quo, is almost certainly a gross over-simplification. Even so, if it leads to some interesting new experiments, it will have adequately served its purpose.

The notion that mirror symmetric points in the two hemispheres might be specialized for *complementary* rather than identical functions is also attractive for another quite distinct reason. Throughout evolution, the exploitation of duplicate body parts to permit the emergence of new modules is the rule rather than the exception (32). I would be surprised therefore, if the redundancy inherent in the two cerebral hemispheres had not been exploited in this manner.

These ideas on hemispheric specialization are also consistent with the ingenious speculations of Kinsbourne (25); and Gazzaniga (17). However, these authors do not consider the specific evolutionary advantage of having this peculiar dual organization and they do not discuss anosognosia. I will summarize, here, the most novel aspects of the present proposal:

1. Our idea goes well beyond the notion of a language based 'interpreter' in the left hemisphere (17) in that it raises the question of *why* such a mechanism should have evolved in the first place. Why engage in self-deception and Freudian defenses or adopt false beliefs when – on the face of it – they actually seem maladaptive? Trivers has argued, in a different context, that you deceive yourself in order to lie more effectively, i.e., to prevent traces of emotion from 'leaking' through. Contrary to this view we argue that the main biological function of self-deception is to confer stability on behavior (although it is conceivable that the same mechanisms could *sometimes* be deployed to deceive other people).

2. In addition to this coherencing mechanism in the left hemisphere that preserves the status quo, I postulate a 'devil's advocate' in the right hemisphere that serves as a counter-balance by performing a global consistency check, challenging the status quo and generating a Kuhnian 'Paradigm Shift' whenever necessary.

3. Without this 'correction' or monitoring mechanism in the right hemisphere that looks for global inconsistencies, the organism becomes progressively more and more delusional and can become hopelessly trapped in them, leading to states such as anosognosia. Indeed, as we shall see, even certain enigmatic aspects of normal behavior, such as humor, laughter and dreaming, may also become more comprehensible when viewed in this context.

Of course, when I speak of a 'general' in the left hemisphere or that the right hemisphere is required for 'paradigm shifts', I am being strictly metaphorical. But the use of metaphors is quite permissible in a field such as ours, that is still in its infancy, so long as one recognizes their tentative status and does not take them too literally. (After all, even the notion of a gene or an electron as a 'particle' was once a metaphor, useful only as an approximation until more accurate accounts could be formulated.) What is exciting to me, however, is that one can even *begin* to experimentally approach such questions as self-deception or Freudian psychology at a neurological level.

Implications for mnemonic functions

Another curious aspect of anosognosia that is rarely commented upon in the clinical literature concerns the patients' *memories* of their failure to use their left hand. When I asked a patient (Mrs OS) on one occasion, 'Mrs. OS, can you use your left hand,' her response was, 'Yes, of course I can use my left hand. In fact, I used it to wash my face this morning.'; and when asked, 'Can you walk?' another patient replied, 'Yes, I can walk; I just went to the rest-room.' I find it quite astonishing that an otherwise mentally lucid and

intelligent person can instantly generate such an absurd confabulation – a false memory – from the recent past. A more intensive study of these phenomena will surely have implications for understanding how memories are 'retrieved' – even in normal individuals – and how they are squeezed into one's pre-existing belief systems.

This amnesia seems to affect only the *actions* of the left hand, but not other aspects of that hand. On one occasion, the patient was surprised to notice that I had slipped a red hair-band on her left hand. After removing the band, I started questioning her about the movements of that hand and, as expected, she vehemently denied the paralysis but, with repeated questioning, she finally admitted that it 'wasn't working'. Yet, a few minutes later, she had no recollection of this 'confession', even though she vividly remembered the hair-band! Surprisingly, even on the one occasion when the patient had a 'catastrophic reaction,' she had no recollection of this a few hours later.

Experiments on anosognosia

Experiment 1: The 'virtual reality box'

Can anosognosia really be conceived of as a failure to generate paradigm shifts in response to an anomaly? If so, would it be possible to test this idea experimentally? An alternate interpretation of anosognosia would be in terms of hemineglect of the left side of the body, i.e. the patient's failure to *orient* to the left. I have already noted that I find this idea implausible, but to test the idea more directly I devised a new experiment. What would happen, I wondered, if one were to temporarily paralyze the *right* arm of a patient that was already suffering from anosognosia in association with left hemiplegia? (Assume the paralysis is achieved by cutting peripheral nerves in the right arm). Would the anosognosia now encompass his *right* arm as well? Note that my theory of anosognosia, outlined above, makes the counter-intuitive prediction that he should now deny that his *right* arm is paralyzed, (since his anomaly detector fails to initiate a paradigm shift), whereas the neglect theory makes the opposite prediction – since there is no neglect of his right side.

To 'paralyze' the patient's (Mrs FD) right arm, I once again used a simple optical trick. The technique is similar to one originally developed for studying intersensory conflict in *normal* subjects, (29), but I realized that it might provide a valuable tool for probing the depth of anosognosia in patients with a right parietal lesion (34,35).

The 'virtual reality box' was constructed out of cardboard and mirrors. The patient's gloved right 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. Unbeknownst to the patient, an accomplice inserts his gloved left hand through another opening in the box so that its mirror image is optically superimposed on the patient's right hand and she is 'tricked' into thinking that she is looking directly at her own right hand. The patient is then instructed to move her right hand up and down to the rhythm of a metronome. The accomplice holds his hand completely steady so that the patient is 'fooled' into thinking that her hand is absolutely still, as though it were paralyzed. I repeated the experiment on two consecutive days and on each day she maintained that she could clearly see her arm moving up and down[§] to the rhythm of the metronome – even though the view afforded to her through the virtual reality box was that of a paralyzed hand!

This simple experiment demolishes all 'neglect' theories of anosognosia, since there was certainly no neglect of the right visual (or somatic) field in this patient and yet she was producing confabulations about her right hand! Clearly, at least in this one patient, 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.[¶]

Experiment 2: Unimanual vs bimanual tasks

When thinking about anosognosia, a second question that arises is, how deeply does the patient believe his/

[§] 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 (19) 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.

[¶] We are currently conducting an experiment to answer a related question, i.e. to what extent can one experimentally alter an anosognosic patient's belief system in order to accommodate the paralysis? For example, what if one were to inject saline into her left arm after telling her, 'As part of your neurological exam, I need to temporarily paralyze your left arm using this local anesthetic'. After the injection, would she say that her arm was paralyzed or would she say, 'Your injection doesn't work. My arm is okay.'? The exciting thing about this syndrome is that it allows you to experimentally address such profound and seemingly intractable questions as the nature of the 'self' or the representation of belief systems in the brain.

her own denials and confabulations? Is it simply a surface facade, or perhaps even an attempt at malingering?

The vehemence of the patient's denials and her quasi-humorous, euphemistic remarks can themselves be taken as evidence that she is 'aware' at some level that she is paralyzed (e.g. 'Yes, of course I can use my left hand. In fact, I used it to wash my face this morning,' or, 'Yes, I can walk. I just walked to the restroom', or, 'I am not very ambidextrous, doctor').

The question then becomes, 'For what sorts of behavior is this tacit knowledge available if it exists?' For example, would it be available for a spontaneous, non-verbal motor response?

To find out, we gave four patients a choice between a simple unimanual task or a simple bimanual task which would be impossible to perform with one hand, e.g. in one case the choice was to either thread a light bulb into a socket that was mounted on a heavy base (unimanual) or to tie a shoelace (bimanual) (See Table 2 for a complete list of unimanual/bimanual task pairs). If the patient threaded the light bulb, she would receive a $5 reward and if she tied the shoelace she would receive $10. Remarkably, the patients start trying to tie the laces and keep at it for several minutes without showing any signs of frustration. Even when the patients were then given the same choice 10 minutes later, they *again* went invariably for the bimanual task. In one case (Mrs LR), the tests were repeated on several consecutive occasions – always with the same result. Indeed, it looks as though the patients have no memory of their previous failures, a 'selective amnesia' for their previous failed attempts.

Table 2 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 a large box	A large box of candy
Use scissors to cut a paper circle	
Unimanual	
Screw the nut on to the bolt (mounted on wood to remain perpendicular)	$2.00
Stack five blocks	Bar of scented soap
Pick up objects with a clamp and put them into a bag	Small box of candy
Pick up toy octopus with a fishing hook and put it into a cup	

Note: The tasks (left-hand column) were paired randomly with different rewards (right-hand column) on different trials. Each patient was confronted 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. Before each trial, she was first given careful demonstrations of both tasks and told that 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 and the smaller or less valuable prizes with the unimanual tasks.

Student: Mrs R, do you remember a short while ago when we did some tests on you?
LR: Yes I do.
Student: What did you do?
LR: That nice Indian doctor... he asked me to tie shoelaces... I did it successfully *using both hands*.

This patient has no problem in carrying on a conversation or remembering me when I come to see her each day, yet she doesn't remember her failure with the shoelace 10 minutes ago. Interestingly, she volunteered the information 'with both hands' when referring to her ability to tie the shoelace. It is difficult to imagine a normal person saying this and it suggests that, in some part of her brain, LR 'knows' that she is paralyzed. She was exhibiting what Freud has called 'reaction formation' – the assertion of the opposite of what one believes to be true. (A notable example comes from Macbeth, 'Methinks the lady doth protest too much'.)

An interesting question concerns the *domain specificity* of anosognosia. Do the patients only deny paralysis of body parts or do they deny other disabilities as well? In my view, this would probably depend on how anterior the lesion is; lesions that are towards the front may tend to produce a more global denial, whereas lesions in the parietal may generate denials that are confined to one's body image.** Indeed, there

** 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.

I would like to emphasize, also, that the specific theory of hemispheric specialization I am proposing certainly isn't the only explanation for all forms of 'anosognosia', e.g. the anosognosia of Wernicke's aphasics probably arises because the very part of the brain that would ordinarily represent 'beliefs' about language is itself deranged. The unawareness of the blind spot (in normal people) and scotomas caused by damage to areas 17 and 18 is probably different, yet again, since the subject certainly doesn't deny the blindness *once it is pointed out to him.*

is already some clinical evidence illustrating that patients with right frontal strokes tend to be blissfully indifferent to the gravity of their predicament, i.e. they suffer from a global anosognosia. Left frontal patients, on the other hand, are often depressed (35), perhaps because they lack even the minimal coping mechanisms that they would need to get on with their lives.

Experiment 3: Denial of other peoples' deficits

Another interesting question about anosognosia concerns the extent to which the patient is aware of *another person's* paralysis (37). I explored this and in three patients found, to my amazement, that two of them showed no awareness of the hemiplegia of the patient in the adjacent bed even when the hemiplegia was demonstrated to them! (All three patients were mentally lucid and none of them had any autotopagnosia, apraxia or evidence of left-hemisphere involvement.) Thus, in these two patients at least, the anosognosia seemed to extend to other peoples' equivalent body parts while, at the same time, excluding their own deficits in other domains. The result implies that some patients may need to access their own body schemata even when making judgments about another human being's body parts (37). The third patient (LR), however, was very surprised when we asked her, 'Is that other patient moving his arm properly or is he paralyzed?' 'Of course he is paralyzed;', she said, 'he is not moving his arm', even though she vehemently denied her own paralysis. It is noteworthy also that, when Mrs LR watched her failure to move her arm in the mirror, she continued to insist that she was not paralyzed (35,37).

Experiment 4: Repressed memories in anosognosia

Our experiments with anosognosia seem to suggest that the information that the patient is paralyzed is being held somewhere in the brain but that access to this information is blocked. Would it be possible to demonstrate this more directly? To do so, we took advantage of an ingenious experiment performed by an Italian neurologist Edwardo Bisiach on a patient with neglect and anosognosia (2). Bisiach took a syringe filled with ice-cold water and irrigated the patient's left ear canal – procedure that is usually used for testing vestibular nerve function. Within a few seconds, the patient's eyes started to move vigorously (nystagmus). Bisiach then asked the patient if she could use her arms. Surprisingly, the patient replied that she had no use of her left arm. The cold-water irrigation of the left ear brought about an admission of her paralysis.

I tried this same experiment on my patient Mrs BM, an elderly woman who suffered a right parietal stroke which resulted in left-side paralysis. My purpose was not only to confirm Bisiach's observation but also to ask questions specifically to test her memory – something that hadn't been done before on a systematic basis.

After going through the usual sequence of questions and eliciting a vehement denial of paralysis, I irrigated her *left* ear with the cold water. I waited until the eyes started moving and then questioned her once again about her paralysis.

VSR: Do you feel okay?
BM: My ear is very cold but other than that I am fine.
VSR: Can you use your hands?
BM: I can use my right arm but not my left arm. I want to move it but it doesn't move.
VSR: Mrs M, how long has you arm been paralyzed? Did is start now or earlier?
BM: It has been paralyzed continuously for several days now. . .

About eight hours later, my assistant tested her.

Assistant: This morning, two doctors did something to you. Do you remember?
BM: Yes. They put water in my ear; it was very cold.
Assistant: Do you remember they asked some questions about your arms, and you gave them an answer? Do you remember what you said?
BM: I said my arms were okay.

Thus, even though BM had denied her paralysis on every occasion that I had seen her in the clinic since the stroke, the information about her failed attempts had been, nevertheless, getting into her brain. It seems as though the *access* to these memories is ordinarily blocked, but the cold water removes the block. The memories then come to the surface and the patient 'confesses' her paralysis. And yet after the effect from the water wears off, the patient flatly denies her earlier admission of paralysis – as though she were completely rewriting her 'script'. Indeed, it was almost as if we had created two separate conscious human beings who were mutually amnesic: the 'cold water' Mrs BM who is intellectually honest – who acknowledges and is disturbed by her paralysis and the Mrs BM without the cold water, who has anosognosia and completely denies her paralysis!

My experiments suggest, therefore, that anosognosia might provide a new experimental paradigm for studying mnemonic functions in the human brain;

especially the question of how new memories are seamlessly incorporated into one's pre-existing cognitive schemata. Such experiments would be especially easy to carry out in conjunction with the caloric-induced reversible hyperamnesia, if this 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 caloric testing, by simply interviewing the patient repeatedly about her memories as she gradually regained insight over several days. For example, four months after her repeated denial of paralysis and repeated failure with bimanual tasks (e.g. tying shoelaces), another patient (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 did I must have been lying and I don't usually lie' (35).

The question arises as to why the water produces these apparently miraculous effects – acting almost as a 'truth serum', as it were. One possibility is that the cold water 'arouses' the right hemisphere. There are connections from the vestibular nerve projecting to the vestibular cortex in the right parietal lobe as well as in other parts of the right hemisphere. Arousal of the right hemisphere makes the patient pay attention to the left side. Thus, the patient pays attention for the first time to her arm, which is lying lifeless, and she then recognizes that she is paralyzed.

This interpretation is probably at least partially correct, but I would like to consider the alternative hypothesis that this phenomenon is related to rapid eye movement (REM) sleep. People spend one third of their lives sleeping and, 25% of that time, their eyes are moving and it is during that time that people have vivid, emotional dreams. In both the cold-water state and in REM sleep there are noticeable eye movements and unpleasant memories come to the surface and this may not be a coincidence. Freud believed that, in dreams, we pull out material that is ordinarily censored. At the risk of pushing the analogy too far, we can refer back to our general who is now sitting in his bedroom late the next night sipping a glass of cognac. He now has time to engage in a leisurely inspection of the report brought to him by the one scout at 05.55 hours and this is what we call dreaming. If the material makes sense, he may decide to incorporate it into his battle plan for the next day, but if it doesn't make sense or is too disturbing for him, he will put it back into his drawer and try to forget about it. This is what happens 90% of the time in dreaming, which is why we cannot remember most of our dreams. Perhaps the vestibular stimulation partially activates the same circuitry that generates REM sleep, thereby allowing the patient to pull out unpleasant, disturbing facts about herself including her paralysis, which is usually repressed when she is awake.[‡*] This is obviously highly speculative, but it does lead to a simple testable prediction: patients with anosognosia should *dream that they are paralyzed.* Indeed if they are awakened during a REM episode they may continue to admit their paralysis for several minutes before reverting to denial again. (Recall that the effects of caloric-induced nystagmus lasted for at least 30 minutes after the nystagmus had ceased.)

A theory of dreams; nature's own virtual reality

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 conscious lives. In dream sleep, on the other hand, the 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 new script doesn't make sense, 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 is incorporated seamlessly into the conscious

[†*] Not every patient 'rewrites the script' to match his/her current beliefs. For example, I recently interviewed a patient (SM) 12 years after he had recovered form a massive right-hemisphere infarct. He vividly remembered denying his paralysis, asking his wife what 'Ken' was doing in bed with him (Ken, as in Ken and Barbie Dolls) and denying that his left arm belonged to him. Thus, the extent to which memories are accurately recollected by any given patient after the acute effects have worn off may itself depend on the presence or absence of residual neurological deficits.

In my experience, somataparaphrenia is usually recollected accurately by patients (although they may or may not regard it as delusional, e.g. Mrs OS insisted that I *had* shown her brother's arm to her during her stay at the hospital. Anosognosia, on the other hand, is often 'repressed', i.e. the patients deny the denial they had engaged in earlier.

[‡*] Soon after I wrote this essay, it was pointed out to me that there is now a fad therapy, popular among some 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!

self in the left hemisphere, so that your 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.)

The reason you can't carry out these rehearsals in your *imagination* – when awake – is not obvious, but two possibilities come to mind. First, 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.' It makes good evolutionary sense that imagery cannot substitute for the real thing.) Second, 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 simulation in a part of your mind/brain that is informationally insulated from you ordinary conscious mind that is active during wakefulness.

By way of analogy, think of the two hemispheres as equivalent to the two separate heads of a Siamese twin of which the left head is you – the conscious talking self – who has a curious alliance with your twin brother who is a starry-eyed dreamer. You are not only the glib press agent for both twins but also the one who makes all the major decisions. It is only for a few hours each night that your brother is allowed to communicate his outlandish ruminations to you and you are then just as likely to accept them or to reject them as you see fit – in order to ensure the survival and well-being of the whole organism. Life without your brother would not only be boring and predictable but would also lead to your becoming submerged in layer upon layer of self-deception. What keeps you intellectually honest are the illicit nightly liaisons with your twin that shake you up to your very core. Indeed, it is 'language chauvinism' that causes me – the person in the left hemisphere writing these lines – to regard the left hemisphere as the repository of the 'true self' (not surprising, given that it is the left hemisphere that is making this claim!).

A theory of medial temporal lobe amnesia: the hippocampus as a 'librarian'

In the previous section, we argued that, during dreams, there is a tremendous liberation from psychological constraints and defenses so that 'censored' memories – even ones that have apparently been forgotten – can emerge into consciousness. This leads to an interesting new question: what do amnesics dream of?

Consider medial temporal lobe amnesia of the kind originally described in patient HM. It is now generally agreed that hippocampal damage from any cause can lead to a profound anterograde amnesia for all events occurring *after* the accident, intact immediate memory, and a relative sparing of 'old' premorbid memories, i.e. partial retrograde amnesia. The 'retrieval failure' view is no longer widely accepted, since it fails to account for the intact premorbid memories. The most widely accepted current view is the 'consolidation failure' theory, according to which the hippocampus is required for 'consolidation' – 'the gradual transfer of memories into durable long-term traces. So, when that structure is damaged, the information never gets into the brain.

I would like to propose a somewhat different hypothesis that doesn't necessarily contradict the consolidation theory, but goes well beyond it. Specifically, I will argue that, in this type of amnesia, the memories *are* in fact transferred into long-term storage but are stored in such a way that they *cannot be retrieved under ordinary circumstances*.

Our first clue comes from realizing that the hippocampus is part of the limbic system and is intimately linked to emotions. Hence, the main function of the hippocampus might be to serve as a 'librarian' or 'sorting office' in conjunction with information received from the cortex. More specifically, it might serve to tag episodic memories – labelling them with the appropriate 'value' or emotional label. (Perhaps all memories are tagged with such 'significance' labels; there may be no such thing as a value-free memory or a truly neutral event.) If the hippocampus is destroyed, therefore, all the new incoming information does indeed get into long-term storage but it gets *misfiled* – since the items can no longer be labeled. Consequently, it would be impossible to retrieve these items under conditions of ordinary 'cued' recall. (Notice that this is not the same as 'source amnesia'; under the present scheme *no* aspect of the memory can be retrieved since it has been completely misfiled – it isn't just its time tag that gets lost.)

This theory explains at least five salient aspects of medial temporal amnesia: (1) the failure to retrieve memories that were laid down after the onset of amnesia – since these have no value labels and have been misfiled; (2) the preservation of premorbid memories, since these already have their labels; (3) intact immediate memory; (4) preserved skills; instead of arguing that 'procedural' and 'declarative' memories are fundamentally different, all one has to assume is that 'value labels' or 'filing' is largely irrelevant for skills and hence their immunity from amnesia; (5) the

occasional report of priming effects in amnesia. Again, 'value labels' may not be required for eliciting unconscious priming.

In the light of our earlier speculation on dreams, these ideas on amnesia lead to an intriguing new question: what do amnesics dream about? If a patient has had anterograde amnesia of sudden onset that developed (say) three years ago, and he/she was woken during a REM episode, would all her dreams be only about premorbid events from her life or would she also dredge up events from the recent past – *even though she can't remember them when awake*?

Although I have yet to conduct a formal large-scale study, I recently had the opportunity to explore this question in a single patient, Mrs SM (see Table 1) in collaboration with C. Gillin, L. McLure and D. Mulherin. This patient had developed profound anterograde amnesia following an attack of 'food poisoning' in Mexico that had occurred three years prior to our testing her. She had no obvious dementia and was neurologically normal apart from her amnesia. Her immediate memory was normal, she had no problem retrieving premorbid memories, and yet she couldn't remember the examining physician even 10 minutes after a one-hour interview!

I woke this patient up during three consecutive REM episodes at the University of California, San Diego dream/sleep laboratory. During the first awakening said she had been dreaming of Batman and described the scene vividly. (Since her immediate memory was normal she had no difficulty remembering the dream.) During the second awakening, she said that her son's girlfriend – a certain lady named Faviola – had appeared in her dream. And finally, two hours later, during the third awakening, she once again reported she had been in her kitchen conversing with Faviola about a washcloth – even though she had no conscious recollection of dream two. She added that Faviola had been her 'son's girlfriend several years ago'. (This is interesting for it suggests that amnesics also have 'recurring dreams' like normal individuals – already a hint that the information does indeed get into the brain.)

I had no idea why Mrs SM had dreamed of Batman (had she seen the movie recently?) or who Faviola was and, when questioned in the morning, she had no idea either. Yet when I asked her husband (in her absence) he said, 'yes, of course, we had spent an hour that very morning reading an article on bats in the *National Geographic*' and 'Faviola? Why, yes, she is our daughter-in-law, who has been away for many years and just moved in to live with us two weeks ago. . .'

It is possible, of course, that this is just a remarkable coincidence and the patient may have actually been dreaming of premorbid, rather than postmorbid, episodes. But, taken at face value, the results have two remarkable implications. First, my hypothesis concerning amnesia might be on the right track – perhaps all the memories are indeed recorded but are simply not available for ordinary free recall or even 'cued recall.' Second, these mislabeled and misfiled memories might be spontaneously dredged up in the bizarre, uncensored world of dreams where 'value' labels are largely irrelevant.

Now the reader might wonder why memories should be dredged up in dreams but not during spontaneous 'free association'. What is so special about dreams? The answer to this question is twofold. First, what we call 'free-association' during wakefulness isn't really free – as any analyst will tell you. It is necessarily constrained by the 'value' labels of individual items. Second, sometimes amnesiacs do indeed surprise the investigator by 'accidentally' retrieving apparently forgotten items of information. Dreams might simply be a way of greatly facilitating such accidental retrievals.

Obviously, before any of these speculative ideas can be accepted, additional experiments are needed on a large number of amnesic subjects. Surprisingly, I found that there was only one study, in the literature, on dream patterns of amnesics (44) and this study was done on Korsakoff's amnesics who might have had other neurological problems. Also, no attempt was made to specifically look for dreams of their *postmorbid memories*, which is critical for my argument.

Is the human mind at the 'edge of chaos'?

The dialectic between the two opposing tendencies that we are proposing also bears a tantalizing resemblance to what physicists call the 'edge of chaos' in dynamical systems and the emergence of 'complexity' at the boundary between stability and chaos (23). This basic principle has been invoked to explain a diverse range of natural phenomena – ranging from stasis vs rapid change ('punctuated equilibrium') in biological evolution to economic theory. As noted by Coveney and Highfield (6), 'these ideas on the relationship between dynamics and computation are part of a larger effort, notably in the field of statistical physics, to find complex behavior in "critical regimes" between order and deterministic chaos. The search for complexity in such regimes is also intellectually appealing since living things appear to capture an elusive mixture of yin and yang. Biological life seems to occupy a zone between regularity and turbulent chaos, where randomness coexists with creative adaptation. Organisms combine the ability to change and innovate with the stability of feedback systems that ensure a well-defined structure and metabolism.'

I propose here that the same basic principle might underlie at least some interhemispheric interactions. Chaos arises in deterministic systems which show a highly sensitive dependence on initial conditions and this is not unlike the sensitivity to perturbation (or 'anomalies') that I have postulated for the cognitive style of the right hemisphere. In marked contrast, the left hemisphere is relatively *insensitive* to change and tries to preserve stability. 'Interesting' or 'complex' types of behavior, on the other hand, seem to emerge spontaneously at the boundary between the two – a place where there is just enough novelty to keep things interesting and unpredictable but also just enough stability to avoid complete anarchy and instability. And it is precisely these little eddies of 'complexity'[§§] at the border zone that may correspond roughly to what we call human caprice, innovation and creativity.

These are obviously highly speculative ideas, but perhaps it isn't just a coincidence that science, as a collective human enterprise, also works in exactly the same way: long periods of stasis ('normal science') followed by sudden upheavals of thought in response to anomalies. Indeed, this essential tension between 'conservative' and 'radical' science may be a social manifestation of the same interplay of forces that occurs between the two hemispheres of individual humans. One wonders, also, whether the creative surges that occur in manic depressives, as they see-saw between the two states, are also a manifestation of complexity at the edge of chaos. It would be easy enough to determine whether these individual are truly more creative along the manic phase (as often assumed) or actually only during the transition between the two phases – as our theory would predict. Perhaps the actual surges of creativity occur during the relatively rapid transition but the results are allowed expression mainly during the manic phase.

This basic idea may also help resolve the old debate between free-will and creativity, on the one hand, vs the deterministic 'behaviorist' view of the brain on the other. The answer may lie in 'complexity' emerging at the boundary between the two since, according to this view, novel and interesting patterns of activity can arise spontaneously at the interface without violating any physical laws. (By way of analogy, think of any large company: neither a totalitarian dictatorship nor complete anarchy will work. Nor will a boring compromise. What is needed instead is a dynamic interplay of precisely the kind that we have postulated to occur between the two hemispheres.)

Awareness of death and the genesis of depression: an evolutionary hypothesis

Humans are unique in that they have well-developed frontal lobes that enable them to contemplate and plan for the future – they are not prisoners of immediate circumstances. But the penalty they had to pay for this is the awareness of their own mortality:

> One moment in annihilation's waste
> One moment of the well of life to taste (24)

I suggest that, as soon as the earliest hominids became capable of the contemplation of the future of their own minds, they must also have evolved a special 'value yourself' program, i.e. you are not only aware of your own mind but also *value* your mind – for if you didn't, you would not bother to make elaborate plans for the future. This is different from the mere crude, reflexive avoidance of pain that one sees in lower animals, although it serves essentially the same evolutionary purpose.

But if you value your own mind, then you ought to be terrified by the prospect of its eventual annihilation. Which one of us has not woken up in a cold sweat in the middle of the night wondering about our mortality and pondering the futility of it all, given the ultimate humiliation and finality of Death? Fortunately, such disquieting moments usually pass, but the question remains: why isn't everyone paralyzed by the constant fear of death?

> For who would lose, though full of pain,
> This intellectual being, those thoughts that wander through eternity
> To be swallowed up and lost
> In the wide womb of uncreated night (28)

I suggest, therefore, that, to compensate for the growing awareness of death – of the terrifying annihilation that it portends – our brains also evolved a

[§§] I hasten to add that, although 'complexity theory' (23) has begun to achieve some measure of respectability in the mathematics community (e.g. at the Santa Fe Institute), its relevance to natural phenomena – especially biological phenomena – is not without its critics. The ideas I am proposing should, therefore, be taken with a generous pinch of salt!

A potential objection to my idea is that hemispherectomized patients – who have had the telencephalic structures of one cerebral hemisphere removed completely in early childhood – do not appear grossly abnormal in their cognitive functions. One response to this objection would be that the personality and creativity of these individuals has never been tested on a systematic basis (e.g. a one-eyed person may seem to have normal depth perception unless carefully tested with a stereogram!). A second answer would be that, since the hemisphere was either damaged or removed in early childhood, the kind of dialogue I am postulating could have been set up either between subcortical structures or even entirely within one hemisphere. My emphasis is not so much on the particular anatomical structures but on the rather special kind of 'balance' between two seemingly antithetical tendencies that coexist in any human being. I would argue, also, that this is unique to humans; it is only humans who can generate *counterintuitive* conjectures – so that the status quo can be questioned if necessary. Indeed, it is this ability to tentatively entertain implausible conjectures that may be the hallmark of our species, setting us apart from 'lower' primates.

special purpose mechanism to selectively uncouple it from limbic/emotional centers in the brain, a mechanism akin to denial or 'repression'. As a result, although one is still intellectually aware of the inevitability of one's mortality, one is not ordinarily terrified by it. Indeed, one may 'intellectualize' it – treat it as an intellectual problem (as I am doing now!) rather than respond to it emotionally. The remarkable ability that our brains have to intellectualize a problem for the sole purpose of trivializing its emotional impact is one of many forms of self-deception that we all engage in to 'protect the ego'. It is what Sigmund and Anna Freud (13,14) would have called a 'psychological defense mechanism', although they did not specifically suggest that it might be a coping strategy for avoiding fear of death.

Think not I dread to see the spirit fly
Through the gates of fell mortality
For 'tis living ill that makes a man fear to die (24)

Now, in my view at least, some forms of depression may arise when this particular mechanism goes awry, so that the person's coping mechanisms break down. Consequently, she becomes acutely conscious of her own mortality. A disturbance in the REM/dream sleep mechanism that ordinarily censors forbidden thoughts – such as fear of death – may be partially responsible which would explain why many antidepressants seem to work by blocking REM sleep. Notice that this hypothesis is not incompatible with a chemical theory of depression: indeed biochemical and psychodynamic interpretations should be regarded as complementary rather than mutually exclusive.

Other 'censored' memories, besides fear of death, may also rise to the surface of consciousness as a result of such a faulty REM gating mechanism. Many layers of self-deception ordinarily hide the truth from the conscious mind; as we have seen, the sole reason self-deception originally evolved may have been to create a coherent and internally consistent belief system in order to provide one's behaviour with stability. A flaw in this mechanism may, therefore, have a profoundly destabilizing effect on the system, leading to depression. One way of treating depression in male patients, therefore, might be to take advantage of the nocturnal erections that occur exclusively during REM. For example, one would attach a penile plethysmograph to an alarm bell to produce selective REM awakenings during the night, thereby providing relief from depression without resorting to psychoactive drugs.

Humor and laughter: a biological hypothesis

I will now 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 Schopenhauer (40), a singularly humorless 19th century German philosopher. Typically, humor involves taking someone up along a garden path of expectation so that the left hemisphere (in my scheme) is allowed to construct a story or model, and then introducing a sudden unexpected twist at the end so as to force a *paradigm shift*, i.e. a completely new model has to be invoked to explain the same data. 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 (20). 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 'orienting' mechanisms in the right hemisphere *leads to a paradigm shift whose implications are trivial.* 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 pre-existing 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 particular loud, explosive, repetitive sound? Freud's view that it is during laughter that you discharge pent-up psychic energy doesn't make much sense without recourse to an elaborate hydraulic 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 consequentially 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.[11]

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 be genuinely disturbing anomalies. In other words, when an anomaly is detected, it is ordinarily dealt with by orienting or – when small – by denial or repression but, 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 new type of psychological defense mechanism (hence the phrase 'nervous laughter').

Summary and conclusions

The ideas I have proposed in this article have much more in common with biologically based theories of cognition and perception (7,11) than it does with the central tenets of classical artificial intelligence (AI). As we have pointed out in the past (31,32), 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 (11).

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. I suggest that patients with anosognosia afford a unique opportunity for experimentally approaching these seemingly intractable problems. Indeed, as we have seen, they may even help us answer the eternal riddle 'What is the sound of one hand clapping?'.

[11] Partial evidence for this view comes from a curious neurological syndrome called 'pain asymbolia' seen in patients with damage to the insular cortex. These patients recognize a pin-prick as painful, but do not withdraw from it – claiming that, 'it doesn't hurt'. Ironically, many of these patients also laugh uncontrollably when getting the pin-prick. We suggest that the patient initially begins (CSF) to the painful ('anomalous') stimulus but, when he realizes this case – that it is inconsequential ('doesn't hurt'), he withdrawing his hand. The universal human which is paradoxically aversive and funny at similar origins.

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