Unawareness of Visual and Sensorimotor Defects: A Hypothesis

DAVID N. LEVINE

Neurology Services, Spaulding Rehabilitation Hospital and Massachusetts General Hospital, and the Department of Neurology, Harvard Medical School, Boston, Massachusetts

A theory is proposed to account for unawareness of blindness, hemianopsia, and hemiplegia, and for phantom limb after amputation. It is assumed that interruption of a sensory pathway at any level—from peripheral nerve to primary sensory cortex—is not associated with any immediate sensory experience that uniquely specifies the defect. Instead the sensory loss must be discovered by a process of self-observation and inference. Discovery is easy for defects that create major functional disability, such as total blindness. Hence unawareness of total blindness occurs only in association with severe intellectual impairment, precluding the required self-observation and inference. In contrast, hemianopsia is difficult to discover because several mechanisms automatically compensate the defect effectively. Thus unawareness of hemianopsia is common, even in intellectually normal individuals. Insensate fields are often the source of suggested (false) percepts, because no information from such a field specifies the absence of a sensory stimulus. The most powerful source of suggestion is sensory activity in uninvolved portions of the affected sensory field. Thus hemianopsics may perceive complete geometric forms when only incomplete forms are shown and the missing portion falls in the hemianopsic fields. Such perceptual completion also occurs in hemianesthetic hemiplegics, creating the illusion that there are normally functioning limbs on the affected side. This perceptual completion increases the difficulty of discovery of hemianesthetic hemiplegia, but the disability is still sufficiently obvious that some additional cognitive impairment is invariably present in patients with lasting unawareness of hemiplegia. Phantom limb after amputation is the product of perceptual completion without associated cognitive impairment. The patient with phantom limb is thus aware of the illusory quality of his phantom. Some insight into the neural basis of perceptual completion and of unawareness of sensory loss may derive from considering sensory systems and associative cortex as parallel-distributed processing mechanisms.

© 1990 Academic Press, Inc.

I thank Drs. Raymond Adams, Ron Calvanio, David Caplan, David Diamond, C. Miller Fisher, Carla Grosmann, and Simmons Lessel for reading an earlier version of this manuscript and for making valuable suggestions. Address correspondence and reprint requests to the author at Spaulding Rehabilitation Hospital, 125 Nashua Street, Boston, MA 02114.
Diseases of the brain cause a variety of functional defects, involving the domains of perception, motor skills, and intellect. For over a hundred years it has been known that some patients afflicted with these defects may be completely unaware of them (Von Monakow, 1885; Anton, 1899). A blind man may believe that he can see, and a hemianopsic may believe that his field of vision is normal. The hemiplegic may be unaware of his paralysis, and the aphasic may believe that he speaks normally. A patient with dementia may be unaware that he has lost any of his intellectual skills.

Although many explanations have been proposed, there is still no satisfactory theory of unawareness of neurologic defects (McGlynn & Schacter, 1989). Several of the earlier theories do not accord with later discovered clinical or neuroanatomic facts. Some theories may apply to one disability but cannot be generalized to unawareness of other defects. Other theories postulate hypothetical brain centers of awareness for which there is no clinical or physiologic evidence. No theory allows detailed predictions of (a) the clinical setting in which unawareness of a given functional defect may occur, (b) the rich variety of behavioral consequences of unawareness of a given defect, and (c) the sites of lesions and the nature of the neural dysfunction underlying unawareness of neurologic defects.

This paper is an attempt to present such a theory. It begins with the rare but dramatic syndrome of loss of awareness of blindness. After a review of earlier theories and their problems, a new theory is proposed. Its point of departure is the hypothesis that sensory loss is never phenomenally immediate but instead must be discovered by observation and inference. The consequences of this hypothesis are used to explain why unawareness of blindness occurs only in a setting of severe intellectual loss and why patients unaware of blindness often confabulate visual percepts. The next section of the paper extends the theory to unawareness of hemianopsia, explaining why this condition does not require intellectual impairment and why it is often associated with the phenomenon of perceptual completion. In the third section the theory is further extended from visual to sensorimotor defects, where it predicts a clinical setting for the denial of hemiplegia that accords well with clinical facts and suggests a new explanation of why unawareness of hemiplegia is more common after damage to the nondominant hemisphere. The theory also accounts for many hitherto unexplained clinical phenomena associated with phantom limb after amputation. The fourth section of the paper deals with the neural basis of the theory, assuming that the brain consists of parallel-distributed processing systems. The fifth section briefly sketches extensions of the theory to a variety of cognitive defects. In the final section the assumptions and predictions of the theory are
UNAWARENESS OF SENSORY LOSS

compared with those of two other recently proposed theories of unawareness.

I. UNAWARENESS OF BLINDNESS

A. Review of Prior Theories

Individuals who become blind may be aware of their visual defect or may be oblivious to it. In fact, there appears to be not a dichotomy but rather a continuum of degrees of awareness of blindness (Critchley, 1979). At one extreme, an occasional patient may deny blindness outright, remaining unconvinced by any demonstration of his disability and confabulating visual experiences. Alternatively he may admit that objects appear dim, but he does not acknowledge a handicap, excusing his poor performance by claiming that the lighting is inadequate. Other patients, while not denying blindness, show little concern over it. Although amenable to demonstrations of their blindness and able to acknowledge it, they do not allude to it or inquire about it, so that their handicap is not the focus of their attention or a matter of much interest. At the other extreme, the majority of blind patients are quite aware of their visual defect and show appropriate concern.

This variability in awareness of blindness has been a matter of neurological interest for a hundred years. During the late nineteenth century it was widely believed that awareness of perception was a function of the modality-specific sensory cortex. This belief arose from a variety of sources. Johannes Müller (1838), in formulating the theory of specific nerve energies, had suggested that consciousness of the specific quality of each sensory modality lay not in the sensory organ or peripheral nerve but rather in the central portion of the nerve pathway. As evidence, he adduced the occurrence of sensations when the central stumps of severed nerves were stimulated. Later Helmholtz (1860) distinguished sensation, which depended only on the stimulus, from perception, which depended upon the subject’s previous experience supplementing or modifying the sensation. Sensations could be dissociated from perception if the subject focused his attention inwardly on the percept occupying his consciousness. This technique of introspection was employed widely by Wundt (1862) who called the conscious focusing of attention “apperception”. Still later, Lissauer (1890) localized visual apperception to the visual cortex. Thus, apperceiving a visual object—becoming consciously aware of it as the center of one’s attention—was a function of the visual cortex. It was believed that a patient who became blind from ocular or subcortical brain lesions would experience “vision noire” (visual blackness) and be aware of his blindness. In contrast, if the visual cortex was destroyed, the patient would have “vision nulle” and be unaware of his blindness just as a normal individual is unaware of his physiological blind spot (Dufour, 1889).
This "classical" view was questioned by Anton (1899), who first discussed in detail the psychological aspects of unawareness of blindness or deafness. He mentioned several cases in which patients with unilateral lesions of visual cortex were aware of a hemianopsia, contrary to the theory of Dufour (1889). Anton accepted the belief that visual cortex was the substrate of immediate awareness of blindness. But he believed that even without visual cortex unilaterally, a patient might still become aware of his hemianopsia indirectly. This secondary, indirect awareness depended upon inference. The patient could compare his perception from the right to his perception from the left, his present capacities with his prior abilities, and his own perceptual abilities to those of normal individuals. Anton concluded that although damage to visual cortex would preclude immediate awareness of the visual defect, only additional damage—beyond visual cortex—would also preclude awareness by inference, resulting in an individual without interest in, or attention to, his sensory defect. He localized this additional damage to the association fibers connecting the visual cortex to the rest of the brain.

Redlich and Bonvicini (1908) challenged the classical view more strongly. They found that denial of blindness could occur in patients with lesions of the optic nerves (chronic papilledema associated with frontal lobe tumors) in whom the visual cortex was spared. Thus lack of awareness of blindness did not require damage to the visual cortex or to the occipital association fibers originating from it. Redlich and Bonvicini concluded that lack of awareness of blindness was a manifestation of generalized, severe brain dysfunction in the presence of blindness of any type. They did not believe that defects of memory, hallucinations, or confabulations could explain the lack of awareness, but offered no positive psychological explanation to replace the classical theory.

During the subsequent twenty years there were several attempts to reconcile earlier views with the findings of Redlich and Bonvicini. Albrecht (1918) expanded Anton's concept of association fiber damage to include lesions of the frontal white matter (containing occipito-frontal connections) or of the corpus callosum. He believed that such associative fiber damage, even if accompanied by blindness that was only peripheral in origin, might indirectly compromise the function of the visual cortex itself, and thus cause lack of awareness of the blindness. Pötzl (1924) hypothesized that awareness of percepts depended upon cortical-thalamic interaction. Corticothalamic fibers, independent of the primary sensory pathways, modulated primitive reflex mechanisms, decreasing their strength and transforming it into the substrate of an integrated sensory schema. Damage to this mechanism, often manifest clinically as euphoria, could cause unawareness of blindness of either peripheral or central origin. The patient would be unable to distinguish visual memories
from percepts and would be dominated by the latter much as the normal individual who dreams when asleep.

Subsequent authors have supported the view of Redlich and Bonvicini that unawareness of blindness is due to diffuse brain dysfunction. In attempting to clarify the nature of this dysfunction they were influenced heavily by Goldstein's (1939) concept that brain damage leads to a re-organization of the personality so that the now more limited individual can adapt optimally to his environment. Redlich and Dorsey (1945) considered the possibility that the brain disease causes "regression to more primitive hysterical patterns of behavior in which the denial of their most important defect is of paramount importance". They found that all patients with denial of blindness had defects of intellect, memory, and orientation, and had delusions and hallucinations. They believed that interruption of reverberating circuits between thalamus and sensory cortex, which mediate awareness of self and environment, was the cause of this state. Weinstein and Kahn (1950) also found that denial of symptoms, including blindness, never occurred in isolation but was always part of a general disorder of behavior which included disorientation, confabulation, affective change such as euphoria or unconcern, personality change, illogical thinking, memory disorder, paraphasia, and in some cases misidentifications and hallucinations. Denial of symptoms was related to the premorbid personality, tending to occur in the patient with a "strong drive to perfection and superiority" who reacted with guilt or anxiety to illness, which he considered an imperfection. Weinstein and Kahn believed that the generalized brain disease caused their patients to relate to the environment in a simpler, more stereotyped manner, largely in terms of needs and feelings. In the individual with the above premorbid personality, the need to be well is paramount and could, as a result of the brain dysfunction, transcend the constraints of reality leading to denial of symptoms. Angelergues, Ajuriguerra, and Hecaen (1960) emphasized the ubiquity of disorientation, impaired memory, and euphoria in patients with denial of blindness. They believed that denial of blindness represented a specific form of mental disorganization, related to the unique role of vision in elaborating the cognitive structures relating to space and the affective forces constituting the "dynamic infrastructure" of the personality. The patient with denial of blindness has a pathological alteration of consciousness in which his visual images become divorced from perceptual reality. In addition there is a personality change consisting of euphoria with relaxation of inhibitions. The resultant of the spatiotemporal disorganization and the euphoria is a "visual delirium" in which the patient lives in an imaginary world that satisfies his needs. Although not specific about the necessary lesions, they suggested that damage to association fibers of the occipital white matter, in the presence of blindness from any cause, was important.
B. Problems and Assumptions of Prior Theories

Each of these theories has difficulty accounting for some of the clinicopathologic facts. Some of these difficulties have already been reviewed. The classical theory cannot account for the existence of patients with damage to visual cortex who are aware of their visual defects. Anton's theory cannot account for patients who deny blindness of peripheral origin. The theories of Albrecht and Pötzl fail to explain the ubiquity of severe intellectual impairment in patients with denial of blindness and depend upon unproven and vaguely described neuroanatomic mechanisms. The theories maintaining that denial of blindness is a manifestation of a global disorder of behavior also face difficulties. First, denial of blindness is only an extreme of a continuum of reactions to blindness. There are also patients who are unaware of being blind but accept their defect when it is demonstrated to them. While it is understandable that repression of reality to gratify the need to be well could explain explicit denial of blindness, it is more difficult to accept this explanation for cases of unawareness without denial. Second, these theories encounter difficulty when one tries to extend them from denial of total blindness to denial of hemianopsia. Whereas the large majority of patients who deny total blindness have gross disturbances of intellect and memory, as required by these theories, the same is not true of denial of hemianopsia. In one study (Warrington, 1962), twelve of twenty patients with hemianopsia had no evidence of mental deterioration and, of these, six were unaware of their hemianopsia, stating that they could see equally well to the left and the right sides. Finally, the theories emphasizing that denial represents gratification of the need to be well cannot explain why a patient will deny some of his defects but not others. It is commonly the case that a patient is aware of a hemiplegia but unaware of a hemianopsia (Gassel & Williams, 1963). Presumably, the hemiplegia represents as great an imperfection as the visual defect; yet the patient acknowledges the former, but is unaware of the latter.

Before I present an alternative hypothesis that I believe will better account for the clinical facts, I must point out an assumption that is either explicitly stated or implicitly contained in nearly all of the above theories. This is the assumption that awareness of blindness is ordinarily phenomenally immediate. By something being "phenomenally immediate", I mean that it is known directly, as a sensory experience is known, without the need for inference. If blindness is phenomenally immediate, one is instantly and directly aware of its occurrence just as one is directly aware of what he sees when his visual system is intact. The phenomenal immediacy of blindness has been assumed by nearly all previous theories, and each of them postulates that some additional damage to the brain is required for this phenomenal immediacy of blindness to disappear. In the classical theory the visual cortex was responsible for awareness of
perception and—in a blind individual—awareness of blindness. Only when the visual cortex was destroyed did the patient become unaware of his blindness. Anton held the same view, except that he acknowledged secondary, more indirect, or inferential, ways of becoming aware of blindness, such as by comparing past and present capacities. Even the theories asserting that denial of blindness reflects a general disorder of the central nervous system assume that, in the absence of such a disorder, awareness of blindness is phenomenally immediate. Thus, something extra must occur, associated with widespread damage to the brain, to explain the lack of awareness. For Redlich and Dorsey this might be regression to a primitive form of hysterical behavior caused by interruption of reverberating circuits between thalamus and cortex. For Wein- stein and Kahn it is relating to the world in a simpler manner dominated by gratification of the need to be well. For Angelergues et al. it is a euphoric state in which the constraints of reality are relaxed. For each of these investigators there is a reorganization of the personality in which there is a repression of awareness of blindness so that powerful needs or wishes can be fulfilled.

C. A New Hypothesis

I propose a theory that begins with rejection of the assumption that blindness—peripheral or central—is phenomenally immediate. Blindness is not sensed, or experienced, in the same way that visual or other sensory percepts are phenomenally obvious. A sighted individual viewing an object knows it is there without the need for reasoning and deduction. In contrast, the individual who is struck blind lacks immediate knowledge of his state. He must learn that he is blind by observation and inference.

This hypothesis becomes plausible when one considers that there is no unique visual state that constitutes the immediate experience of blindness. It is often supposed that visual “blackness” is such a state. Admittedly, individuals who are suddenly struck blind during daylight commonly report such blackness. For example, in transient monocular blindness, visual blackness is the most frequent, although not the only complaint (Fisher 1952; Marshall & Meadows, 1968). The experience of visual blackness, however, is not unique to sudden blindness. It occurs in a sighted individual when the lights are extinguished in his room, when he walks out into a moonless night, or when he enters a cave. It is therefore not surprising that the first reaction of a suddenly blinded individual may be that the lighting is insufficient. Having been sighted all of his life, the individual frequently does not consider blindness as the initial explanation of the darkness. He becomes convinced of his state only when the evidence excludes other explanations.

Visual blackness, however, is only a transient state. It may occur briefly when a light-adapted individual is suddenly blinded, but it quickly
disappears. The same is true of sighted individuals who enter a dark cave. Within an hour "no one in the party could report that he was experiencing darkness. The visual field was so free from dark experience that it seemed as though objects should be distinguishable. Under ordinary circumstances, when the visual field was as free from darkness as theirs had become, they would have been able to see objects quite distinctly" (Cutsforth, 1933). Blind writers (Cutsforth, 1933; Chevigny, 1946) vigorously dispute the popular conception that the blind live in a world of darkness. The blinded individual with no functional retinas perceives "neither light nor darkness" (Cutsforth, 1933), "no color whatever" (Chevigny, 1946).

The blind experience neither a visual blackness nor an empty void. If blindness strikes an individual after many years of normal sight, his mental economy continues to be transacted in visual terms. Sounds, including the meanings of words, and haptic experiences are transformed into visual images. "... The same mechanism operates in my mind as operates in the mind of anyone listening to a piece of radio drama. The voices I hear at once call pictures to my mind ... " "... I 'see' rooms I have entered and buildings I have passed" (Chevigny, 1946). "The tactual experience derived from placing a passive hand upon a chair back ... expands visually to include some sort of complete chair ... [which] may be complete in the details of color, number, and position of rungs, turned or square legs, square or rounded arms, leather or cane seat, or upholstery" (Cutsforth, 1933). Experimental investigations (Hollins, 1985) confirm this testimony objectively, demonstrating that adventitiously blinded individuals continue to image in visual terms.

Thus, blindness of peripheral origin in a previously sighted individual, is not the "vision noire" of classical theory. Such blackness is, at most, a transient state, experienced only at the outset of sudden blindness in a light-adapted individual. For example, although transient monocular blindness may be experienced as a black screen or a dark cloud, this experience does not persist if the blindness is permanent. A patient (case 2) of Fisher (1952) described several transient episodes of blindness in the right eye as "a fog rolling in", but when examined seven months later and found to have severely reduced acuity in that eye, he "did not realize that vision was so impaired and could not recall the time of onset of the blindness". Moreover, when monocular blindness occurs gradually rather than suddenly, no blackness need occur, and occasionally the individual is completely unaware of the visual loss until it is detected by the physician.

If there is no visual experience that is unique to blindness, then blindness is not phenomenally immediate. Even if an unexpected sensation, such as blackness, should occur, it must be interpreted as blindness rather than as faulty illumination. When no unexpected sensation occurs,
the individual must attempt something that requires vision, take note of his failures, and infer that he is blind. Awareness of blindness is thus not awareness of a *sensation*, but rather knowledge of a nonsensory *fact*. The detection of blindness is not like discovering something by seeing it. It rather resembles a process of diagnosis, in which one notes data and infers from them the most plausible explanation. One learns the fact of his blindness not through introspecting and examining the sensory content of his consciousness, but rather by a process of self-diagnosis.

The cognitively intact individual usually makes this discovery, or self-diagnosis, of blindness quickly and easily. If he senses dimness or blackness, he can readily exclude explanations such as entering a cave or extinguishing indoor lights. Excluding these explanations may require only knowledge of his situation—for example, that he is walking outdoors in his neighborhood on a sunny day—or, at most, one or two simple actions—for example, switching on the light or pulling up a shade if he is indoors. When there is no accompanying sensation of dimness or blackness, discovery of total blindness will still be quite easy for the cognitively intact individual. The visual modality is so dominant, that errors in perceptual identification and navigation will soon occur, and the self-diagnosis of blindness will be evident from simple inference. Thus, unawareness of blindness lasting more than a few moments, does not occur in individuals who have sufficient intellect and memory to make a few simple observations and inferences.

In contrast, the individual with severe cognitive impairment may remain unaware of his blindness. If he senses blackness or dimness, he may misinterpret it as faulty illumination, and he will be unable to make the observations and inferences required to prove this interpretation false. Once the sensation of dimness or blackness passes, or if it never occurs, he has no sensory experience to inform him immediately that he does not see, and he will be unable to interpret his errors in perceptual identification and navigation as evidence of blindness. One of my patients (see Appendix, case 3) gave a retrospective account of delayed discovery of blindness which developed in association with severe cognitive impairment. She was an intelligent physician who had suffered a cardiac arrest causing cerebral anoxia. Eventually she recovered her intellectual functions and memory, but was left with severely impaired visual perception. Even after her intellectual recovery, she remained unaware of her visual difficulty until she was asked to read some names and addresses, found that she could not get the information that was sought, and concluded that her vision was impaired. She had to infer her blindness from her failures, because it was not given in immediate sensation. In another of my patients (see Appendix, case 1) I recorded some conversations in which I attempted to demonstrate to him the blindness that he was denying. These conversations illustrate his difficulty in inferring
his blindness from failures on simple tests of vision and also his poor memory for the inference once I led him to it and he seemed, momentarily, to accept it.

When the cognitive skills necessary to infer blindness are lacking, the patient may report visual experiences that are the result of suggestion, engendered through other sensory modalities. If no sensation of visual blackness occurs, or once this transient sensation passes, auditory and tactile sensations will synesthetically evoke visual images. Judging such visual images as illusory rather than true visual percepts will be difficult for the cognitively impaired, blind individual. Even in normal subjects, suggestion can influence visual perception (Bruner & Minturn, 1955). Ordinarily these effects are small, subordinated to a reality which is strongly constrained by information from stimuli transmitted through the visual sensory channels (Fodor, 1983). Without such visual sensory information, visual experience remains the chief mode of mental representation, but suggestion—based on information from other sensory modalities—is its only source. Even in these circumstances, one’s knowledge of his situation and of the range of his possible experiences will serve to maintain the distinction between visual image and visual percept. If such knowledge is lacking, however, the blind individual may be at the mercy of suggestions, confounding the images they evoke with visual percepts. James (1890) emphasized that states of reduced cognition, such as sleep or hypnosis, facilitate the acceptance of suggested percepts as real. Thus, it may not be difficult to persuade a cognitively impaired blind individual that he can see, and if such an individual, unaware of his blindness, looks at something merely to confirm a strong expectation he may well report seeing what he expects. Pötzl (1924) described a patient of Hirschl with mild general paresis and with blindness caused by tabes dorsalis who first claimed that he could see after a fellow patient with messianic delusions told him that he was healed. Case 2 (see appendix) illustrates the power of suggestion in a patient who was already unaware of being blind. In the course of denying his blindness, this patient claimed to see objects that he had been led verbally to believe were before his eyes, even when nothing was there. Suggestion may thus create expectations strong enough to support the conviction that one sees something if his critical faculties are compromised and if he lacks any visual information that he does not see what he expects. Our patient’s impaired intellect and memory precluded his realizing that he consistently failed visual tasks and prevented his inferring from these failures the fact of his blindness. Hence suggestion had free reign.

Since detection and awareness of blindness require self-observation, inference and memory, a variety of defects may interfere with the acquisition of this knowledge. Attentional defects may make the necessary
self-observation impossible, and defects of intellect and memory may preclude adequate inference from the data of observation.

Defects in spatial attention may be of considerable importance in failure to detect blindness. The cortex and white matter of the dorsal hemispheric convexity, from parieto-occipital to midfrontal lobes, is essential for the prompt and accurate spatial distribution of attention. Patients with extensive lesions of this region have the syndrome of Balint (1909), or visual disorientation (Holmes, 1918). They have relatively little appreciation of the spatial disposition of objects around them and cannot perceive or explore the environment as a single spatially articulated structure in which data from the different sensory modalities cohere. Let us consider what would happen if such a patient also became blind, perhaps because the lesions in the parietal white matter extended sufficiently ventrally to involve the visual radiations. I believe that he would be slow to appreciate his blindness. He would lack a coherent spatial framework against which he could test his capacity to gain visual information. He would thus not gather the evidence necessary to infer his blindness, and would remain unaware of it. In this regard it is noteworthy that some cases with lack of awareness of blindness were in patients with symptoms of visual disorientation. Anton’s (1899) patient had a “striking . . . lack of spatial orientation. She could not find her way about her room, where she had been for weeks. She could not find her cup, always beside her in the same place. . . . She also could not localize sounds, which she clearly perceived”. At autopsy there were bilateral lesions of the parieto-occipital convexities involving the cortex of the first and second occipital gyri and the angular gyri, extending into the subjacent white matter including the visual radiations.

Although the patient with visual disorientation will find it difficult to gain awareness of his blindness, the task is probably not impossible. The normal individual sees even when he is not actively exploring the environment spatially. There are still expectations that visual information can be gained, which can be induced in such a patient by verbal information. Thus, the patient with blindness and visual disorientation should understand a request to name the color of a large object that he is reassured is before his eyes or surrounds him and should realize after failing that something is wrong with his vision. The patient of Graveleau, cited by Angelergues et al. (1960) was blind with “total spatial disorientation”, but was aware of his blindness. Thus, with adequate intellect and memory, and with other sensory systems intact—particularly hearing and touch—loss of spatial mechanisms alone will not guarantee persistent lack of awareness of blindness.

Intellectual skills sufficient to make the necessary inferences are of paramount importance in gaining awareness of blindness. The patient must be able to infer either from a sensation of darkness or from his
failures on tasks demanding vision not only that there is a problem, but also that the problem is that he is blind. A variety of defects in intellect and memory may make such self-diagnosis impossible, and I shall turn now to a discussion of these.

The patient with an amnestic-confabulatory state cannot learn new facts. If blindness develops in association with an amnestic-confabulatory state, the patient will be unable to learn the fact of his blindness. He will generally assume that he sees as he always had, and he will confabulate visual experiences consistent with this belief. An amnestic-confabulatory state is extremely common in patients with denial of blindness, and some investigators, as previously discussed, believe it to be the rule. I interpret the prominence of the amnestic-confabulatory state differently from other authors. I do not believe that it serves to subordinate a phenomenally obvious reality—blindness—to the gratification of a need to be well. Rather, it deprives the individual of the cognitive tools that he needs to learn that he is indeed blind.

The effect of an amnestic-confabulatory state on awareness of blindness can be seen in case 1 (see Appendix). In the conversation recorded on January 12, 1984 the patient, who was totally blind, believed that he could see perfectly. This belief was expressed in the context of other confabulations concerning his location and his illness. Common to all of these confabulations was that they expressed a more usual, or habitual situation than the one he was actually in. He believed himself well rather than ill, near home rather than far away, and sighted rather than blind. When confronted with evidence of his poor vision—his failure to count fingers—he did not change his mind and discounted the evidence. Given more evidence, he appeared to back down and to accept that he could not see. However, this "evidence" did not lead, as solid evidence should, to a lasting conviction that he was blind. Instead, within minutes he had forgotten about the evidence completely, as the older and stronger mental set of retained vision returned to dominate his behavior.

The pathologic basis for denial of blindness in association with an amnestic-confabulatory state is often infarction of the ventromedial aspects of the occipital and temporal lobes caused by occlusion of the basilar or of both posterior cerebral arteries. The geniculo-calcarine visual pathways are destroyed bilaterally, accounting for the blindness, and the hippocampal formations are destroyed, accounting for the amnesia. Some authors (Dide & Botcazo, 1902; Dide & Gassiot, 1912) have maintained that occipital lesions alone can cause an amnestic-confabulatory state. However, this claim predated the discovery that lesions of the limbic system cause amnesia, and it is not clear that limbic structures were adequately examined in these early cases. Nevertheless, the cingulate gyrus, a component of the limbic system, extends posteriorly into the anteromedial occipital lobe, and a recent report has suggested
that amnesia may be caused by a lesion of this retrosplenial portion of
the cingulate gyrus (Valenstein et al., 1987).

Other patients with denial of blindness have not necessarily had a
typical amnestic–confabulatory syndrome, although all have had severe
intellectual defects that could account for failure to learn and remember
that they are blind. In the older literature, there are reports of patients
with large brain tumors, particularly of the frontal lobes, who denied
blindness caused by chronic papilledema associated with increased in-
tracranial pressure. In each case severe defects of intellect and memory
were present. In case 1 of Redlich and Bonvicini (1908) the patient was
apathetic, knew little of the history of his illness, and the meager infor-
vation he gave was often self-contradictory. He was disoriented to place
and date, had severely impaired recent memory, and his flow of thought
was slow, poorly sustained, and impoverished. At autopsy a large bi-
frontal and callosal glioblastoma was present, which also involved the
medial portions of the caudate nucleus and the thalamus. In case 2 of
the same authors, the patient was again apathetic with impaired memory
although neither was as severe as in case 1. She contradicted herself
constantly without any appreciation of her inconsistency, and she was
unaware of many aspects of her illness other than blindness. At autopsy
there was a massive meningeoma at the base of the brain compressing
the brainstem and the basomedial temporal lobes. There was also chronic
hydrocephalus and flattening of the frontal gyri. In case 3 of Albrecht
(1918), the patient’s mental state fluctuated considerably. At first apa-
thetic, he was later euphoric and even manic, with flight of ideas, de-
Iusions, and nocturnal agitation. He could not sustain attention to his
examiners, he had visual hallucinations, and he was occasionally dis-
oriented to time. At autopsy there was a large tumor of the left frontal
lobe.

In attempting to explain the denial of blindness in such cases, many
investigators have emphasized the role of the patients’ affective abnor-
malities—particularly their euphoria or unconcern. Pötzl (1924) noted
euphoria not only in these patients but also in blind tabetics who first
denied blindness with the onset of general parcsis. He interpreted the
euphoria as evidence of dysfunction of corticothalamatic connections
which caused a confounding of perception and imagery, such as occurs
in alcohol intoxication or in dreams. Angelergues et al. (1960) concurred
with this view, emphasizing that this “visual delirium” served to gratify
the patients’ needs, particularly the need to be well (Weinstein & Kahn,
1950).

I suggest, however, that it is the cognitive effects of these large frontal
tumors that account for the patients’ failure to become aware of their
blindness. Several investigators of patients with even milder damage to
the frontal lobes, without the additional complication of increased intra-
cranial pressure, have disclosed a variety of cognitive defects that could—if more pronounced—prevent the self-diagnosis of blindness. These patients have difficulty adopting new mental sets or hypotheses (Milner, 1963, 1964; Teuber, 1964). They lack the mental perseverance or effort that is required to examine evidence conscientiously and to weigh alternatives to make sound judgments. They may lack the initiative to make any inferences, or they may make quick, rash judgments unsupported by evidence. If severe, such inability to change outlook—such disregard of evidence that one’s beliefs are wrong—will clearly hamper the self-diagnosis of blindness.

Finally, the present theory is distinguishable from others with regard to the role of the premorbid personality in denial of blindness. For Weinstein and Kahn (1950), who believe that gratification of the need to be well is paramount, denial is most likely to occur in individuals who strive for perfection and consider illness to be weakness and imperfection. According to the present theory, discovery of blindness requires the ability to change mental set or outlook, based on evidence from the external world. Denial would be more likely in individuals who are rigid and who tend to trust their own feelings about things rather than the statements of others. Future research may determine which of these alternatives is correct.

II. LACK OF AWARENESS OF HEMIANOPSIA

Unawareness of hemianopsia usually occurs in a clinical setting that is very different from unawareness of total blindness. First, unawareness of hemianopsia is a far more common disorder. In one study (Gassel & Williams, 1963) 31 of 35 hemianopsics had some degree of unawareness of their visual field defects, and in another (Warrington, 1962) 11 of 20 hemianopsics were unaware of their visual field defects to the extent of affirming that they saw equally well to the left and the right. Second, unawareness of hemianopsia frequently occurs in the absence of severe defects of intellect and memory, which invariably accompany unawareness of blindness. As mentioned earlier, of the 20 hemianopsics studied by Warrington (1962), 6 of the 12 who had no mental deterioration were unaware of their hemianopsia. Finally, hemianopsics do not confabulate visual experiences in their defective fields to verbal suggestion as do many patients with denial of blindness. Yet they may be subject to perceptual “completion” in which an incomplete object may be seen as complete if the missing portion is confined to the hemianopsic fields.

Because of such differences in the clinical contexts, it has been difficult to extend previous theories of unawareness of blindness to unawareness of hemianopsia. For example, the repression theories (Redlich & Dorsey, 1945; Weinstein & Kahn, 1950; Angeleigues et al., 1960) require severe intellectual and memory loss for pathological repression of the visual
defect to occur, but in the patient unaware of his hemianopsia intellect and memory may be normal. In contrast, the present theory can be readily extended from unawareness of blindness to unawareness of hemianopsia and can also account for the different clinical settings of the two disorders.

The underlying hypothesis of the present theory is that a visual defect is not phenomenally immediate but must rather be discovered by observation and inference. Like the patient with total blindness, the hemianopsic does not become aware of his problem by an immediate sensation that is unique to hemianopsia. Instead he must discover his defect by observing his defective performances. Applied to hemianopsia, the hypothesis becomes identical to the conclusion of Gassel and Williams (1963) that "the hemianopic field is an area of absence which is discovered rather than sensed; it is a negative area whose presence is judged from some specific failure in function rather than perceived."

From this theoretical perspective, the major difference between hemianopsia and total blindness is that discovery of hemianopsia is much more difficult than discovery of total blindness. In hemianopsia there are several automatic mechanisms of compensation that make discovery relatively difficult. The most important of these is that the eyes frequently move, so that objects outside of the field of vision are brought into it by refixation. Even within a single fixation there are unconscious compensatory adjustments. The direction of the visual axis may be shifted slightly toward the hemianopsic field (Gassel and Williams, 1963; Sergent, 1988), residual vision is present in most hemianopsic fields, and it is possible to focus attention into such hemianopsic fields without shifting the position of the eyes (Gassel and Williams, 1963). In contrast, no comparably effective, automatic mechanisms are available to compensate total loss of vision, which instantly causes severe functional limitations that are readily discoverable.

This difference in the ease with which the two defects can be discovered accounts for some of the differences in the clinical settings of unawareness of hemianopsia and unawareness of total blindness. Unawareness of hemianopsia is common, because the majority of patients cannot fully solve the difficult task of discovering the nature of their visual defect. Intellect and memory can be intact, because discovery is so difficult that even cognitively normal individuals will be challenged. In contrast, total blindness is easily discovered by intellectually normal individuals. Therefore unawareness of blindness is rare and is always associated with severe cognitive impairment.

Although cognitive impairment is not essential for unawareness of hemianopsia, the present hypothesis predicts that unawareness will be more frequent in cognitively impaired individuals because they will be unable to make the observations and inferences necessary to discover
their hemianopsia. In fact, there is evidence that defects both in visuo-spatial capacities and in intellect and memory can aggravate unawareness of hemianopsia by making the task of discovery even more difficult than it already is.

The visuo-spatial defect that accompanies hemianopsia most frequently is unilateral spatial neglect of the hemianopsic side. There is strong evidence that a patient whose hemianopsia is accompanied by unilateral spatial neglect is more likely to be unaware of the hemianopsia than a patient with hemianopsia but no neglect. In Warrington's (1962) study, for example, awareness of hemianopsia and unilateral spatial neglect were highly negatively correlated. Only one of nine patients with neglect was aware of his hemianopsia, whereas nine of ten patients without neglect were aware of their hemianopsias. The present hypothesis accounts for this correlation as follows: The patient without neglect has the opportunity to learn that he cannot identify items on one side of his point of fixation with the same facility as items on the other side, because he is capable of deliberately directing his gaze or attention to the right or the left. By contrast, the patient with unilateral spatial neglect is constantly directing his attention or gaze to the other side. This affords him no opportunity to compare his vision to the right with his vision to the left. It is therefore more difficult for him to discover his failure to acquire information from the neglected side.

Impaired intellect and memory also impede the discovery of hemianopsia. Gassel and Williams (1963) found that insight into hemianopsia decreased when level of consciousness decreased in three patients. Although Warrington (1962) found that three of eight patients with "mental deterioration" were aware of their hemianopsia, it is clear that none of her patients had pronounced cognitive impairment. All were described as "fully cooperative, able to carry out instructions, and able to maintain concentration. . . ." In my experience patients with acute confusion and hemianopsia associated with recent temporo-occipital infarction are usually unaware of their hemianopsias even in the absence of unilateral spatial neglect.

The phenomenon of "perceptual completion" in patients with hemianopsia can also be explained by the present theory. Poppelreuter (1917) first reported that when a geometric figure, part of which fell into the blind hemifield, was exposed tachistoscopically to hemianopsic individuals, some reported seeing the whole figure. The same "completion" could be observed when the stimulus was in fact incomplete and the missing portion fell in the hemianopsic field. Although some investigators (Bender & Teuber, 1946; Torjussen, 1978) have doubted the completion of such objectively incomplete figures, the observations of Poppelreuter have been confirmed repeatedly in numerous hemianopsic patients (Pollack et al., 1957; Warrington, 1962; Gassel and Williams, 1963). War-
rington, and Gassel & Williams found that completion occurred regularly only in patients who were unaware of their visual field defects.

Before attempting to account for perceptual completion in terms of the present theory, I should like to point out both the similarities and the differences between perceptual completion in hemianopsia and confabulated percepts of the totally blind. The two phenomena are generically similar in that both involve reports of visual experience from blind portions of the visual field. Some degree of unawareness of the visual defect is a prerequisite both for confabulated percepts of the totally blind and for perceptual completion in hemianopsia. The two phenomena are also similar in that the patient's expectations are of critical importance in determining whether the phenomena will occur. The role of verbal suggestion in the confabulated percepts of the totally blind has been discussed previously. Expectation also plays a vital role in perceptual completion of the hemianopsic (Gassel & Williams, 1963). When Sergent (1988) reduced expectations by first showing incomplete items entirely within the intact visual hemifield, by using "incomplete" stimuli that were meaningful in themselves as well as when completed, and by utilizing a response of copying the stimulus form rather than naming it, completion was reduced to a minimum, although not entirely eliminated.

There are also important differences between the confabulated percepts of the totally blind and perceptual completion in hemianopsics. The confabulated percepts of the blind are induced synesthetically—that is, by nonvisual stimuli, and the percepts may consist of complex objects. In contrast, perceptual completion in hemianopsia is induced by an intramodal stimulus—a visual pattern in the intact visual field. The completion is limited to perceiving a simple symmetric geometric figure from an incomplete one, or a whole familiar object from one that is not quite complete. Finally, confabulated percepts in the totally blind occur only in patients with severely impaired intellect and memory, whereas completion can occur in intellectually intact individuals, although its occurrence is facilitated by impaired intellect and memory (Pollack, Battersby, & Bender, 1957; Gassel & Williams, 1963; Sergent, 1988).

The assumption that hemianopsia is not phenomenally immediate can be the basis for an account of perceptual completion in hemianopsia. The patient who is unaware of his hemianopsia has no sensory experience of blindness to one side. No information from the blind side enters into his percepts, which are solely the product of stimulation of the seeing hemiretinas. Having neither information that the missing part of the object is there nor information that it is missing, the patient will "perceive" whatever is suggested to him by the stimulus configuration in the intact field and, to a lesser extent, by other experimental conditions. He will report the object most likely to have produced the pattern of activation on the seeing part of his retinas. Thus he will complete an in-
complete geometric pattern to form a symmetric or familiar shape, or he will complete a picture with a missing part so that a meaningful item is reported. In contrast, the patient who is aware of his hemianopsia knows that he cannot see to one side, much as a normal individual knows that he cannot see behind his head. He has established a new subjective visual field and will be more resistant to suggestions that imply perception in the excluded blind field. Intellectual and memory impairment, by preventing the discovery of and memory for hemianopsia, keep the patient unaware of his visual defect and facilitate completion.

The differences between visual confabulation in the totally blind and perceptual completion in hemianopsics derive from the character of the suggestion that induces the false percept in each case. The synesthetic induction of visual percepts by verbal means is a relatively weak form of suggestion. It generally does not occur in normal sighted individuals; they do not report seeing nonexistent objects just because someone suggests verbally that they are present. Even in the adventitiously blinded individual, synesthetic visual images—although they occur—are readily distinguished from true visual percepts as long as intelligence and memory are normal. Visual images suggested by verbal means become delusional percepts only when intellect and memory are so severely impaired that the blind patient cannot weigh the evidence that the suggested percepts are illusory and that he is blind. He is thus led to deny his blindness and the verbally suggested synesthetic images assume a perceptual reality.

In contrast, the induction of a complete visual form by one that is slightly incomplete, under conditions of brief tachistoscopic exposure, is a very powerful means of suggestion. Unlike verbal synesthetic suggestion, completion occurs even in normal subjects without sensory defects. In Warrington's (1962) study of completion in hemianopsics the normal control subjects reported seeing complete figures on up to 20% of trials. In the hemianopic who has not discovered his visual defect, the completion effect is even stronger, as he has no information from the hemianopic field that the missing portion was absent. Thus, completion—with the full impact of perceptual reality—can occur in the absence of intellectual impairment. However, the completion is constrained to the most probable of complete objects that would entail the pattern of stimulation on the intact field. A nearly complete symmetric figure or a nearly complete familiar object will be perceived as complete. If the inducing stimulus is highly incomplete or unfamiliar, too many possible completions of nearly equal probability exist. The force of the suggestion decreases, and completion is less likely to occur.

The neurologic implications of the present hypothesis, as applied to unawareness of hemianopsia, are straightforward. There is no segment of the primary visuo-sensory system (retino–geniculo–calcarine path-
way) that holds a special position with regard to awareness of a visual defect. Isolated lesions of the optic tract, lateral geniculate body, visual radiations, or primary visual cortex will all cause hemianopsias that are equally difficult to discover. Nevertheless, lesions in some locations will cause unawareness of hemianopsia more frequently than others because of damage to adjacent areas of the brain, which impair the capacity to make observations and inferences needed to discover the hemianopsia. A lesion of the visual radiations which involves the overlying parietal lobe will cause contralateral spatial neglect, rendering discovery of the accompanying hemianopsia more difficult. A lesion of the calcarine cortex—if accompanied by damage to the hippocampus, as frequently occurs in posterior cerebral artery territory infarction—may be associated with an amnestic syndrome which—if sufficiently severe—will also preclude discovery of the hemianopsia.

III. UNAWARENESS OF HEMIPLEGIA AND THE PHANTOM LIMB—AN EXTENSION OF THE HYPOTHESIS

The hypothesis that interruption of a sensory pathway—whether from peripheral or from cortical damage—does not cause immediate phenomenal awareness of the sensory defect, and that insenate fields are subject to suggestion, either as completion or as synesthesia, can be extended from the visual system to the somatosensory system. The hypothesis can then serve as a basis for an account of clinical phenomena such as denial or unawareness of hemiplegia and “phantom limb” after amputation (Mitchell, 1872).

Extension of the hypothesis from the visual to the somatosensory system is both straightforward and plausible. There is no unique sensation corresponding to interruption of somatosensory input just as there is no unique sensation corresponding to blindness. Admittedly, a patient with sudden somatosensory loss may report feeling as if his affected limbs were absent. Like visual “blackness”, such a feeling of absence is transient, and it generally does not occur at all when the sensory loss is more gradual. Instead, the affected limbs are felt to be in some position and state of movement, although neither may correspond to reality. Furthermore none of these feelings depends upon damage at a specific level of the somatosensory pathway. The transient feeling of absence can occur with cortical lesions—most often as the aura of a seizure—or with brainstem lesions (Frederiks, 1969). The feeling that a limb is in an objectively false location can occur not only with peripheral lesions such as amputation, but also with lesions of the spinal cord (Bors, 1951) or of the cerebral cortex (Von Hagen & Ives, 1937).

Because there is no unique sensation corresponding to somatosensory loss at any level of the neuraxis, such loss cannot be phenomenally immediate. The patient must learn of the loss through observation and
inference. He must learn that he cannot detect stimuli applied to the skin of the affected limbs, and that the limb is not in the position he thought. If there is accompanying paralysis, he must discover it, because there is no afferent information that the limb is not moving. And, if the limb has been amputated, he must discover that too.

These discoveries are fairly easy for the cognitively intact individual. Loss of the effective use of even one limb creates major functional limitations that are not quickly and automatically compensated. The affected individual can see the poorly functioning limb or the amputated stump and can infer the nature of the problem. Thus, the amputee, the patient with brachial plexus injury, and the paraplegic do not remain unaware of their problems for more than a few moments if they are cognitively intact. With regard to ease of discovery, loss of somatosensory input from one or more limbs is much more like total blindness than like hemianopsia.

The cognitively impaired individual, however, may be slow to discover somatosensory loss and accompanying paralysis or amputation because he lacks the ability to make the proper observations and inferences. For example, one of my patients rapidly became paraplegic from a spinal epidural abscess associated with back pain, chills, and fever and did not become aware that his legs were paralyzed until the examining physician later demonstrated the paralysis to him. Another patient who suffered cranial and spinal trauma did not become aware of her paraplegia until several weeks after she was objectively awake and visually and verbally responsive. Only when her intellect and memory returned did she discover her paraplegia when she noted her failure to move her legs on request.

As there is no phenomenally immediate knowledge of somatosensory loss, the affected individual will be influenced by suggestion with regard to the location and mobility of the affected limbs. If the sensory loss affects only a part of the body, sensorimotor patterns involving the unaffected parts of the body can be "completed" over the insensate portion in a manner similar to visual perceptual completion in hemianopsia. Because completion is a powerful form of suggestion, the "sensed" feelings and movements of the affected segment may be experienced vividly. Synesthetic suggestion, i.e., suggestion originating in other sensory modalities, may also play a role, particularly if the individual is cognitively impaired, but it is weaker than completion and its role is a secondary one.

A. Anosognosia for Hemiplegia

The hypothesis that sensory loss and accompanying paralysis will remain unknown to the patient until he discovers them can now be applied to cases of unawareness of hemiplegia—the anosognosia of Babinski
(1914). The patient with anosognosia is unaware that he cannot move his paralyzed arm or leg. As in the case of blindness, there is a continuum of degrees of awareness of hemiplegia. At one extreme the patient may obstinately deny his paralysis, even when it is demonstrated to him. If asked to move his limb, he may say that he has done so, even if no movement has occurred. Chancing to catch sight of his paralyzed extremity, he may believe it to belong to someone else and attempt to throw it out of bed. Another patient may be unaware of his paralysis, but will accept it when it is demonstrated to him. However, a short time later the same patient may again be unaware of the paralysis when questioned. Still another patient may not mention his paralysis when questioned about his health, but will acknowledge some degree of weakness of the limbs if specifically asked. Yet he may show little concern and a tendency to underestimate the degree of disability. Finally, other patients with sensory loss and accompanying paralysis are fully aware of their disability.

Early investigators (Babinski, 1918, 1924; Barré, Morin, & Kaiser, 1923; Barkman, 1925) emphasized the importance of somatosensory loss—particularly loss of proprioception—in the pathogenesis of anosognosia for hemiplegia. Yet they acknowledged that sensory loss could not fully account for the lack of awareness primarily because some patients with severe sensory loss and hemiplegia were quite aware of their paralysis. This situation led other investigators (Schilder, 1923; Gerstmann, 1942; Roth, 1949) to postulate a disorder of the "body image", a hypothetical focal cerebral mechanism mediating one's mental image of his own body, located in the parietal lobe but distinct from the somatosensory system itself. Still others (Sandifer, 1946; Weinstein & Kahn, 1950; Nathanson, Bergmann & Gordon, 1952) emphasized the importance of general mental impairment in anosognosia for hemiplegia. In particular, Weinstein and Kahn believed that anosognosia for hemiplegia never occurred in isolation but was rather part of a generalized disturbance of behavior reflecting "a reorganization of brain activity in which the patient denies whatever he feels is seriously wrong with him".

The present theory attempts to integrate previous views of the importance of both proprioceptive loss and mental impairment in the pathogenesis of anosognosia for hemiplegia. In the absence of somatosensory input, particularly proprioception, the patient has no immediate knowledge that his limb has, or has not, moved. He must discover his paralysis by observing his failure in tasks requiring movement of the affected limbs. As previously mentioned, this discovery is ordinarily quite easy for the intellectually intact individual. Thus, prolonged anosognosia requires sufficient intellectual impairment to make the ordinarily easy discovery more difficult or impossible.

Sensory loss, particularly loss of proprioception, appears to be a nec-
essary element in anosognosia for hemiplegia (Critchley, 1952), as emphasized by Babinski (1918, 1924) and other early investigators (Barré, Morin, & Kaiser, 1923; Barkman, 1925). Gerstmann (1942) acknowledged its presence "in the great majority of cases" and Weinstein and Kahn (1950) believed sensory loss to be "invariably present". Willanger et al. (1981) found loss of position sense in 80% of patients with denial of hemiparesis, but the techniques of testing were not described, and it is not clear whether the exceptions were valid. Bisiach, Vallar, Peroni, Papagno, and Berti (1986) found only a small correlation between anosognosia and impaired detection of touch to the paralyzed side and a slightly greater—but still small—correlation between anosognosia and "personal neglect"—the inability to reach for the paralyzed arm with the unaffected arm. (This test resembles a test of proprioception but differs in being performed with the eyes open). These low correlations between anosognosia and sensory loss appear, at first glance, to be evidence against the present hypothesis, but in fact such an impression is misleading. The hypothesis that sensory loss and associated paralysis must be discovered predicts that sensory loss is a necessary condition, but not a sufficient condition for unawareness of hemiplegia. There are many patients with sensory loss and hemiplegia who do discover their disability. This group of patients will invariably lower the correlation between anosognosia and sensory loss, so that low correlation coefficients are to be expected.

The present hypothesis predicts that cognitive defects impairing the ability to observe and to infer will distinguish those individuals with sensory loss and paralysis who are anosognosic from those who discover and become aware of their paralysis. Defects in spatial attention are important. The large majority of patients with anosognosia for hemiplegia visually neglect the side of space ipsilateral to the paralysis. Such neglect will make discovery of the paralysis more difficult. Nevertheless, visual neglect is neither necessary nor sufficient for anosognosia. I have seen patients with paralysis, sensory loss, and spatial neglect who were aware of their hemiplegia and I have seen an occasional patient with sensory loss and paralysis who was anosognosic in the absence of unilateral spatial neglect. Bisiach et al. (1986) also reported a few instances of anosognosia without spatial neglect and of spatial neglect without anosognosia.

Impaired intellect is also important in preventing discovery of hemiplegia. The anosognosic is unable to assimilate information from a variety of sources to form a consistent and accurate judgment of the reality of his paralysis. Babinski (1914, 1918, 1924) emphasized the "relatively" normal mental status of anosognosics. He found that the patients could remember events and converse lucidly, with no confusion, hallucinations, or confabulation. However, he invariably reported at least subtle ab-
normalities in concentration and judgement. Thus, while emphasizing the preservation of mental functions in an anosognosic patient, Babinski (1924) acknowledged that she talked excessively, was inattentive, and had a tendency to express erotic ideas openly. Later investigators (Sandifer, 1946; Weinstein and Kahn, 1950; Nathanson et al., 1952) emphasized the ubiquity and severity of such cognitive defects in anosognosia. Nathanson et al. found that, in a given patient, denial of hemiplegia would wax and wane in parallel with his degree of disorientation.

Our observations (Levine & Calvanio, unpublished data) confirm the views of these authors. Anosognosic patients often fail to grasp the essentials of their situation. They also are incapable of deliberate and organized behavior to establish or to verify a hypothesis. More specifically, anosognosic patients are often intermittently disoriented, displaying paramnestic delusions as to where they are and what has happened to them. They are apathetic, lacking in initiative and curiosity. When presented with a task such as copying a block design, the anosognosic may show bizarre, non goal-directed behavior. He confuses the copy with the model, attaching his blocks to the model or disassembling the latter. Performance on verbal tasks may be considerably better, but lack of organization in narrative accounts frequently betrays difficulties in concentration and focusing of thoughts.

It remains the case, however, that the degree of intellectual impairment in patients with anosognosia for hemiplegia may be considerably less than that present in patients with denial of blindness, and this difference seems to pose a problem for the present hypothesis. Both the patient with total blindness and the patient with hemianesthetic hemiplegia must discover their disability. For both the task is quite simple if cognition is preserved, since each condition creates a major, easily discoverable defect in function. How is it, then, that the patient with unawareness of blindness must have major cognitive impairment, while the patient with anosognosia for hemiplegia need have only mild or moderate intellectual loss to be unable to discover their respective defects?

I believe that the answer to this question is related to the difference in the nature and strength of the false percepts that prevail in the two conditions. As previously mentioned, false visual percepts in the patient with denial of blindness arise synesthetically, i.e., from stimuli originating in other modalities. This form of suggestion is relatively weak and only achieves the status of perceptual reality in the patient with severe cognitive impairment. In contrast, the anosognosic hemiplegic, like the hemianopsic, still has at least one-half of the relevant sensorimotor field intact. Patterns of somatosensory-motor activity originating in the intact half of the body may be completed across the affected half, since there is no information from the latter to indicate lack of participation in the sensorimotor process. The present theory thus predicts that the ano-
sognosic will have a more or less vivid feeling of a limb that can move and sense normally, and previous investigators (Pötzl, 1924) have reported such phantoms in their anosognosic patients. When the anosognosic patient notices his paralyzed limb, and it does not coincide in position or state of motion with the perceptually completed phantom, he may deny ownership of the affected extremity. It is the presence of this strong, perceptually completed phantom that facilitates the anosognosic's belief that his affected limbs are normal. We shall see, however, that even the most vivid phantom is not sufficient in itself to preclude discovery of the defect (v.i.). Thus, in addition, a certain measure of spatial neglect or impaired intellect must be present for the patient to accept the phantom as reality and to deny that the paralyzed limb is his own.

B. The Phantom Limb

The hypothesis that somatosensory loss and accompanying paralysis are not phenomenally immediate, but rather must be discovered, can also be applied specifically to the case of phantom limb following amputation. In doing so, however, we shall be forced to refine the concept of "awareness" more than was necessary previously.

The patient with phantom limb is aware of the fact of his amputation—aware that a limb or part of a limb has been removed and that motor and sensory function of that part have been lost. If asked whether all of his limbs are functioning equally well, he will readily inform the interviewer of his amputation. Such awareness would be expected from the present hypothesis, because as previously mentioned, the discovery of amputation is quite easy, the amputee is cognitively intact and he should have no trouble making the necessary observations and inferences. In having this form of awareness—i.e., knowledge of his defect—the amputee is quite different from the previously discussed patients, who lack such knowledge because they have been unable to discover it. However, despite being aware that his limb is missing, the amputee may, in another sense, have a false awareness of its presence. He may feel sensations of tingling, numbness, pain, and motion in a phantom limb. I have suggested that this false "perceptual awareness" is the consequence of completion of patterns of sensory and motor activity from unaffected parts of the body across the missing segments, and I shall discuss this further below. In the amputee such false perceptual awareness can coexist with accurate "cognitive" awareness—that is, knowledge of his defect.

Many of the characteristics of phantom limbs following amputation can be accounted for by completion of sensory and motor patterns across the missing segment. The phantom may appear as soon as the patient recovers from anesthesia, because there is no phenomenally immediate awareness that the limb is missing, and even after the patient discovers
that the limb is gone, perceptual completion can create the vivid impression that it is not. The phantom may be painless, mildly paresthetic, or painful. (The percentage in each category varies greatly from one series to the next, depending on both the patient’s tolerance for discomfort, his willingness to disclose it, and also on the investigator’s criteria of classification) (Abramson & Feibel, 1981; Sherman, Sherman, & Bruno, 1987). Much evidence suggests that pain and paresthesia in the phantom are, in most cases, related to afferent nerve activity from the stump (Sunderland, 1978). Pain immediately following surgery may be due to incisional pain at the stump, completed over the missing segment. Later pain may be associated with diffuse reinnervation of the stump scar by immature, hypersensitive nerve fibers, or from focal neuromas, and the paresthesias associated with activity of these abnormal nerve fibers in the stump may also be completed across the missing segment.

Although the stump is the source of much of the perceptual completion causing paresthesia and pain in the phantom, it is not the only source. Sensations in other parts of the body, particularly stimulation of the corresponding limb on the opposite side may also induce sensations in the phantom. Lack of this source of perceptual completion probably explains why phantom experiences are weaker after symmetric double amputations than after amputations of a single limb (Henderson & Smyth, 1948). Many environmental changes and many activities cause sensations in symmetric body parts simultaneously. The presence of one of these parts will facilitate the “sensation” in the symmetric part, even if it is absent. Such facilitation of the symmetric, missing sensory field has been shown in hemianopsics (Torjussen, 1978).

The location in space of phantom limbs can also be understood by assuming that no information about location or movement is available from the amputated segment and that “completion” of normal patterns of sensation and motion will occur. The phantom limb is nearly always perceived as properly aligned with the stump and it maintains this alignment when the stump is moved. It is likely that awareness of the position of the stump in space, mediated by the preserved joint and muscle afferents of the stump, “completes” itself over the missing segment of the limb, so that alignment is maintained. That this is the case is shown by reports that deafferentation of the stump causes dissociation of the phantom from the stump, so that they are no longer aligned (Riddoch, 1941).

Movements of the phantom limbs, whether reflex or voluntary, can be similarly explained. Patterns of motor activity, now incomplete because of the missing limb, are sensed as complete because there is no information from the missing segment to indicate otherwise. The phantom is thus sensed as having moved. The above-knee amputee on crutches may sense his phantom-knee flexing to avoid an obstacle in the path of
his phantom foot. The visual avoidance reflex, although incomplete in several respects, runs its course as though complete, because there is no information that it is not. Asked to move his fingers, the upper extremity amputee may believe that he has done so. There is no sensory information informing him that he has not moved the fingers and there is a pattern of sensory input from the muscles of the stump which ordinarily occurs with the intended movements. This afferent input may even come from proximal muscles that are not prime movers of the fingers but function rather as fixators of the limb girdle during the intended movement. The importance of the still intact partial input in generating "completion" and hence movement of the phantom can be seen from reports that: (a) Voluntary movements of phantom fingers are produced more easily by distal forearm amputees than by proximal upper arm amputees (Henderson & Smyth, 1948). The former clearly have a more complete pattern of remaining sensorimotor activity than the latter and hence a more compelling suggestion of the completed pattern of movement. (b) Denervating muscles of the stump can abolish voluntary movement of the phantom (Henderson and Smyth, 1948). The denervation abolishes muscular activity from the stump that ordinarily occurs with the intended movement. If the stump afferents are unaffected, they will now signal that no movement has occurred. Or, if the afferents themselves are destroyed, the pattern of sensory input is less complete than it had been and the suggested movement is less compelling.

In addition to the compelling suggestions of sensation and movement, generated by completion of partially preserved sensorimotor patterns across the amputated limb segment, there are other, less common phantom limb phenomena that may be caused by synesthetic suggestion, which is ordinarily less compelling than completion of partial sensory or motor stimuli. For example, occasional patients report being able to move their phantom limbs through a much wider range than is usually the case, and such movements occur in the absence of any contraction of the stump muscles (Henderson and Smyth, 1948). Apparently, in these rare individuals, the suggestion induced by a verbal request to move or by other environmental contexts favoring movements, is sufficient to induce phantom movements even without the partial movement pattern induced by contraction of the stump muscles. Another example is the occasional patient whose phantom limb pain appears more related to expectations of pain than to perceptual completion of stump stimuli. Thus Solomon and Schmidt (1978) reported the case of a woman who developed burning pain in her two phantom legs that appeared to be related to her impression that her amputated legs had been disposed of by burning. The pain disappeared with verbal suggestion. Even in the large majority of patients with phantom limb pain, where synesthetic suggestion is not primary, such suggestion may have a facilitating role. Parkes (1973) found that
prolonged illness prior to amputation and ongoing illness after amputation that threatened other limbs or the patient’s life were more likely to be associated with painful phantoms.

C. Neurologic Substrate of Phantom Limb and Anosognosia for Hemiplegia

The present hypothesis predicts that there is no privileged level of the somatosensory system with regard to awareness of sensory loss and of accompanying paralysis. Whether the lesion involves peripheral nerve, spinal cord, brainstem, thalamus, thalamo-cortical radiations, or parietal cortex, interruption of the somatosensory system conveys no phenomenally immediate knowledge of the defect, and the patient will remain unaware of the sensory loss and any associated paralysis until he discovers them by observing his behavior and making the necessary inferences. Nevertheless, the hypothesis does predict some differences in clinical presentation depending on the level of the lesion, which I shall now discuss.

Phantom limb, according to the present hypothesis, is largely the product of completion of sensorimotor patterns, involving the intact parts of the body, across the insensate field. Clearly, if more of the sensorimotor apparatus is intact, the completed pattern is more strongly specified by the incomplete sensorimotor pattern, and the phantom will be more definite and more vivid. Therefore, the hypothesis predicts that the more peripheral the interruption of the sensory system is, the more vivid and definite the phantom will be. A report that distal amputees move their phantom limbs voluntarily more easily than proximal amputees (Henderson & Smyth, 1948) supports the prediction. Extrapolating to yet more proximal lesions, I suggest that the phantom limb will be more vivid after amputation than after spinal lesions with paraplegia or after cerebral lesions with hemiplegia.

The present hypothesis combines and integrates the two major neurologic theories of the origin of the phantom limb after amputation (Sunderland, 1978). The peripheral theory attributes the phantom limb to activity arising from the nerve endings of the stump. The central theory attributes the phantom to activity in the sensorimotor cortex which mediates sensations and movement of the phantom. Each theory, taken in isolation, faces difficulties. The occurrence of phantom limbs after redivision of the nerves of a stump (Henderson & Smyth, 1948), after anesthetic block with no division of nerves (Melzack & Bromage, 1973), and after paraplegia caused by spinal cord injury (Bors, 1951) creates problems for the peripheral theory. The existence of phantom limbs as an aspect of anosognosia for hemiplegia is a problem for both the peripheral and the central theories, because the lesion in patients with anosognosia for hemiplegia may destroy the entire cortex mediating sen-
sation from the affected limbs, including both primary and association cortex of the parietal lobe (Von Hagen & Ives, 1937; Critchley, 1953). The present hypothesis attributes the phantom state to activity involving all of the remaining nervous system—both peripheral and central—on both sides of the body. This includes the nerve endings of the stump, where hyperexcitability of fine regenerating nerve terminals is probably the origin of most instances of phantom pain (Riddoch, 1911). It also involves the parietal cortex, where stimulation in amputees has evoked phantom sensations (Hécaen, Penfield, Bertrand, & Malmo, 1956). Whether the excitation—be it abnormal or physiologic—originates peripherally, as in the stump, or centrally, as in the cortex, neural activity spreads to the remainder of the preserved nervous system and is integrated with other ongoing neural activity to form a pattern corresponding to a sensation or movement. This pattern, although incomplete because of the missing body segment, is completed across the insensible part in the absence of any information that the pattern is not complete.

Before leaving the subject of phantom limb, I must deal with some data that seem to contradict the present theory. It has been reported that cerebral lesions contralateral to a previously amputated limb may abolish the phantom limb if the cerebral lesion compromises somatic sensation (Head & Holmes, 1911; Appenzeller & Bicknell, 1969). It appears difficult to reconcile these reports with the present theory that abolition of sensory function is the basis of the phantom limb after amputation and of phantom-like experiences after brachial plexus injury, spinal cord section, or cerebral damage.

The paradox can be resolved by examining how the onset of sensory loss might be expected to influence a preexisting phantom limb given the importance of sensory completion. Mild dysfunction of sensory nerve fibers is frequently associated with paresthesias (Melzack & Bromage, 1973), which may be perceptually completed, giving rise to paresthesias in a phantom limb. Thus, amputees who develop a peripheral neuropathy or a partial spinal cord injury with paresthesias in the remaining limbs may also develop paresthesias in the phantom (Appenzeller & Bicknell, 1969, cases 12, 13, 24, 26). With the onset of severe sensory fiber dysfunction, sensory loss occurs and paresthesias may disappear (Melzack & Bromage, 1962). There may be a brief “feeling of absence” of the affected part of the body, which the phantom may share by completion. Afterwards, the patient may remain with sensory loss over a wider area of the body than just the phantom limb. Completion of residual sensorimotor patterns—the basis of phantom sensations and movement—will then be more difficult because these residual patterns are less complete than they were before. For example, Appenzeller and Bicknell (1969, case 34) reported that an amputee with a phantom right leg that was perceived as twisted and painful when he bumped the stump, no longer
UNAWARENESS OF SENSORY LOSS

had this sensation when he developed a left hemisphere stroke that caused sensory loss from the right side of the body.

Thus a cerebral lesion affecting somatosensory function can cause a variety of phantom limb phenomena depending on the circumstances. A prior phantom limb due to amputation may be attenuated or abolished if a wide area of sensory loss disrupts the previous completions of residual sensorimotor patterns. Or, a previous repertoire of phantom sensations may be altered if the cerebral lesion results in paresthesias, which are then completed in the phantom. Finally, the cerebral lesion may cause a phantom where none was previously present, as in cases of anosognosia for hemiplegia.

The neurologic substrate of unawareness, or denial of paralysis, is also predictable from the present hypothesis. Lack of awareness requires both cognitive impairment and proprioceptive loss. The cognitive defects consist of unilateral spatial neglect and impaired intellect. A single lesion causing both the sensory and the cognitive defects will generally be located in the parietal lobe, involving either the cortex itself or the white matter interconnecting the parietal lobe and thalamus. Such a lesion interrupts transmission of sensory information from thalamus to somatosensory cortex and, if it extends slightly anteriorly, can cause paralysis by interrupting corticospinal pathways. In addition the lesion can involve multimodal association cortex to cause the requisite cognitive defect. The lesion may involve the cortex itself, its association fibers within the hemispheres, or its projections to thalamus and possibly basal ganglia. Nearly all cases with anosognosia for hemiplegia have thus involved the parietal lobe, either at the cortical surface or in the subjacent corona radiata (Critchley, 1953). The present hypothesis also predicts, however, that unawareness of paralysis can be caused by other lesions, but that they must be multifocal. For example, the somatosensory system might be interrupted more peripherally, such as in the brachial plexus, spinal cord, or caudal brainstem. A second lesion is then required involving multimodal association cortex of parietal, temporal, or frontal lobes to cause the required cognitive defect.

The present theory suggests an alternative to previous explanations of why anosognosia for left hemiplegia, associated with right cerebral lesions, is more frequent and more severe than anosognosia for right hemiplegia following left cerebral lesions. One explanation (Schilder, 1923) is that the left side of the body is subordinate to the right and that a right cerebral lesion exaggerates this asymmetry, whereas a left cerebral lesion mitigates it. In terms of the present hypothesis, paralysis of the right side of the body creates a greater functional defect and hence is more readily discovered. However, this theory cannot explain why peripheral paralysis—such as occurs after brachial plexus injury—is not discovered more quickly when it occurs on the right than when it occurs
on the left. Nor are phantom limbs after amputation more vivid or persistent on the right. It is thus likely that a functional difference between the two cerebral hemispheres, rather than between the two sides of the body, is the basis for the greater frequency of anosognosia for left hemiplegia. In this regard, some investigators have suggested that the right hemisphere is dominant for the spatial distribution of attention (Heilman, 1979; Mesulam, 1981) and include anosognosia for hemiplegia within the group of "neglect" symptoms following damage to this dominant hemisphere. This theory, however, cannot account for some of the consequences of cerebral disconnection by section of the corpus callosum.

Dominance of the right hemisphere for spatial attention, if it is to be understood analogously to dominance of the left hemisphere for speech or praxis, would imply that the right hemisphere has sole responsibility for directing attention to the left and at least partial responsibility for directing attention to the right. The latter might or might not be mediated by callosal transfer of information to the left hemisphere. A callosal lesion would thus be expected to produce right spatial neglect if it interfered with callosal transfer of information mediating rightward attention. Or it would cause no neglect if callosal transfer is unnecessary for rightward attention. Yet callosal interruption consistently causes left spatial neglect in the acute phase after surgery or injury, even when there is no evidence of right hemisphere damage (Dimond, Scammell, Brouwers, & Weeks, 1977). In the chronic phase after callosal disconnection it has been shown that the right hemisphere contributes only to motor activity of the left hand, while the left hemisphere controls the right hand exclusively and also contributes to a small degree to motor control on the left (Gazzaniga, Bogen, & Sperry, 1967). More recently Plourde and Sperry (1984) found that the left hemisphere of split-brain patients could direct the right hand to touch body parts on the left side as well as the right, while it was not at all clear that the right hemisphere could direct the left hand consistently to either side. Plourde and Sperry attempted to reconcile the bilateral spatial capacities of the left hemisphere with the theory of right cerebral spatial dominance by postulating that the left hemisphere could be inhibited by the right after a partial lesion to the latter. Such inhibition, mediated by the remaining callosal pathways, would prevent the left hemisphere from expressing its full potential. However, their explanation predicts that left neglect should be more severe after a small lesion of the right hemisphere than after a larger lesion. In the extreme, after total right hemispherectomy, no inhibition of the left hemisphere should be present, and there will be no left neglect. This prediction has not been borne out. Patients with large right hemisphere lesions have more severe neglect than patients with small lesions when time since injury is equated for both groups (Levine, Warach, Benowitz, & Calvanio, 1986). In any case, it appears that the left hemi-
UNAWARENESS OF SENSORY LOSS

isphere can activate movements of the ipsilateral limbs and distribute attention to ipsilateral space at least as capably as the right hemisphere and probably more so, i.e., it is the left hemisphere that is partially dominant.

The present hypothesis suggests a means to reconcile the apparent superiority of the left hemisphere with the greater incidence and severity of anosognosia for left hemiplegia. A dominant left hemisphere, able to influence gaze in the ipsilateral direction and movement of the ipsilateral limbs better than the right hemisphere, may be more effective in inducing "phantom gaze" and phantom limb movements after damage to the opposite hemisphere. Phantom limb movement has already been discussed, but "phantom gaze" is a new concept that requires explication. One can imagine a (cyclopean) eye, yoked by antagonistic muscles that pull either to the right or to the left. If the rightward pulling muscles are activated chiefly by the left hemisphere and the leftward pulling muscles are activated chiefly by the right hemisphere, there is an analogy to the right-sided limbs and the left-sided limbs, respectively. Just as a right hemisphere lesion may result in a mobile phantom left arm when the left arm is actually paralyzed, it may also cause a phantom leftward pull on the eye when in fact there is no leftward pull. The eye will therefore be directed rightward of where it seems to the patient to be. The greater severity of left neglect may thus reflect the greater power of the left hemisphere to induce sensorimotor completion on the ipsilateral side because of its capacity for ipsilateral oculomotor control. Similarly, the left hemisphere may be more effective in inducing sensorimotor completion in the left limbs, so that left-sided phantom limbs caused by right hemisphere damage may be more vivid than right-sided phantoms caused by left-hemisphere damage. Thus right-hemisphere damage causes greater spatial neglect and more vivid phantom limbs not because it is dominant but rather because it is subordinate to the left hemisphere with regard to ipsilateral motor control. The presence of a phantom limb and neglect of the paralyzed side will make paralysis more difficult to discover. Hence anosognosia will be more frequent following right hemisphere damage. The greater frequency of anosognosia for left hemiplegia may thus be a reflection of the partial dominance of the left hemisphere for spatial distribution of attention and for motor control.

IV. NEURAL MECHANISMS OF PERCEPTUAL COMPLETION AND UNAWARENESS OF DEFECTS

A. Perceptual Completion

An important aspect of the present hypothesis is that insensate fields, which provide no information as to their state, are often taken to be the source of false percepts. These false percepts are induced by activity in still functioning sensorimotor pathways. The modality of the inducing
pathway may be the same as that of the false percept, as in the visual completion of hemianopsics or the phantom limbs of amputees. Or, the inducing activity may be different from the modality of the false percepts, as in the confabulated visual percepts of the totally blind. I wish to discuss the pathogenesis of such "completion" or "phantom" phenomena more fully.

The large repertoire of highly coordinated, precisely adaptive sensorimotor activities of the adult is the product of a long development. At birth the infant employs relatively simple and poorly coordinated sensorimotor reflexes, but these quickly become more differentiated and at the same time more coordinated with one another (Piaget, 1952). With further maturation, nurtured by experience, sensorimotor capacities become more refined and complex. Somatosensory-motor development allows motor skills that coordinate more and more widely separated body segments into more complex, specific adaptive actions. Visual development allows more rapid and refined discrimination, or categorization of stimuli, coordinating different regions of the visual field.

The mature set of sensorimotor patterns, each highly complex and coordinating wide areas of sensory and motor fields, is highly conducive to the phenomenon of completion across insensate fields. If each somatosensory-motor act involves muscular contraction and proprioceptive afferent activity in a pattern involving much of the body, loss of one segment of the body still allows the portion of the sensorimotor activity pattern in the remainder of the body to develop. This pattern, although incomplete, may nonetheless be sufficiently specific to determine the movement. More precisely, the incomplete pattern will be consistent with a subset of actions and sensations and will specify the most probable of these given the available data.

If this view is correct, perceptual completion should be stronger in adults, who have mature sensorimotor patterns, than in young children and infants, whose patterns of sensorimotor activity are not as complex. Studies of phantom limbs in children with amputation or with congenital aplasia of limbs support this view. Phantom limbs are less frequent after amputations in infancy than after amputations in late childhood and adult life (Simmel, 1962). In children with congenital aplasia of limbs, phantoms occur in less than 20% of cases. When they do occur, they tend to be poorly differentiated anatomically and incapable of "voluntary" movement, and they appear only briefly and intermittently (Weinstein & Ser sen, 1961; Weinstein, Cole, Mitchell, & Lyerly, 1964). Exceptions, however, do occur. Poeck (1964-1965, case 1) reported an 11-year-old girl with congenital absence of both forearms and hands, who had highly anatomically differentiated phantoms of both hands and could voluntarily "move" the individual phantom fingers. In this exceptional individual it is possible that completion was induced by patterns of sensorimotor
activity in the feet and toes, possibly reinforced by synesthetic suggestion
induced by visual observations of the hands of others. In this regard it
is notable that the phantom hands did not appear until age 6, when
sensorimotor patterns of other body segments and visual perception were
relatively mature and able to sustain the necessary completion.

The neural mechanism of perceptual completion is unknown, but the
structure of the nervous system in general and of the sensory systems
in particular is conducive to its occurrence. Neural connectivity is char-
acterized by marked divergence and convergence. Each point of the
sensory field is represented over a wide region of each of several grey
structures such as thalamus and cortex, and each small region of these
grey structures affords the possibility of interaction from a large area of
the sensory surface. A percept begins with excitations of many sensory
transducers which signal their states to the central nervous system in
parallel. A vast number of central neurons are excited or inhibited by
each percept, and no one neuron is critical to the successful completion
of the perceptual act. It is also the case that one and the same neuron
participates in many different percepts, so that a retinal ganglion cell
might be excited by many different objects in the visual field. Thus each
percept is represented by a pattern of excitation and inhibition distributed
over many neurons, and patterns corresponding to different percepts
overlap to varying degrees. At each stage of the serially connected neural
structures in a given sensory system, a subset of the neurons transmit
the output over many parallel fibers to the next stage where another
pattern develops. The patterns of connectivity of neurons within each
stage and perhaps between the stages is altered by experience, so that
at any time the entire set of neural connections instantiates the rela-
tionship between the set of possible stimuli and the set of possible dis-
criminable percepts.

Artificial systems constructed along the lines just described are known
as "parallel distributed processors". These systems have been studied
intensively in recent years, because they demonstrate, in a rudimentary
fashion, many features typical of human perception (McClelland, Ru-
melhart, & Hinton, 1986). For our purposes the most important of these
features is resistance to partial damage—"graceful degradation". If the
system is disturbed by destroying some of the intermediate "neurons"
or by burdening the input with random noise, the system tends to main-
tain input--output relationships that are highly similar to those that were
operative in the undamaged state (Hinton & Sejnowski, 1986). The
greater the damage, the greater the error rate, but the systems are sur-
prisingly tolerant to injury. The parallels between such graceful degra-
dation and perceptual completion are obvious. The amputee has had a
segment of his input neurons rendered inoperative. Nevertheless, the
input--output relations of his somatosensory system remain fairly con-
sant. The now incomplete input still is transformed into a complete perceptual output—a phantom limb. If the sensory systems of the brain can be identified with parallel-distributed processors, the phantom limb is a natural consequence of partial damage.

B. Unawareness of Disability and False Reality Judgements

Another important aspect of the present hypothesis is that unawareness of an easily discoverable defect such as hemiplegia or total blindness requires cognitive impairment. One aspect of this cognitive difficulty is a defect of judgement, so that the patient believes that he is normal rather than disabled and that percepts in his insensate field are real rather than illusory.

The judgement that an illusory percept is real occurs for several reasons. Vividness of the percept contributes to its being taken for real, and so does the fact that having the percept is more in accord with the patient’s past experience than not having it. The anosognosic’s phantom arm, for example, is consistent with his lifetime of having a mobile limb. However, neither vividness nor habit are sufficient to guarantee that the percept will be judged real. The amputee with a phantom limb has an even more vivid phantom than the anosognosic hemiplegic, and both are impelled by an equal force of habit, a lifetime of having four mobile limbs. Yet the amputee does not believe that his phantom limb is real. He appreciates it for the illusion that it is.

The most important condition for judging a percept as real is that it is not incompatible with other percepts or ideas whose reality is more certain (James, 1890). For each individual there is a set of percepts and ideas whose existence and truth are nearly incontrovertible. These and everything compatible with them are taken as reality. Everything incompatible with them is judged as unreal and illusory. For the amputee, the phantom limb, although vivid and carrying the force of habit, is incompatible with the memory of his recent history, the knowledge of his present situation, and the evidence from his sense of vision. These predominate over vividness and habit, and the phantom is no more than an illusion. In contrast, for the anosognosic hemiplegic vividness and habit are not so easily vanquished. His visual attention may be displaced away from his affected limbs, and his memory for the recent past and orientation to his present situation are weakened sufficiently for the vivid phantom, representing his habitual state, to be taken for real. Before discussing the neurologic foundations of this error I shall first mention the parallel error of judgement—the patient’s denial or unawareness that he is disabled.

Awareness of a sensorimotor defect, according to the present hypothesis, is not an immediate sensory phenomenon. It instead is the result of discovery, the product of observation and inference. The in-
ference that one is disabled represents the judgement that is most consistent with the individual’s perceptions and other knowledge. The hemianesthetic hemiplegic may note his failure to perform tasks requiring the use of his affected limbs. He may also be told of his paralysis by family, friends, or physician. In these circumstances the most reasonable inference, the one most consistent with these observations and information, is that he is paralyzed on one side. The anosognosic hemiplegic does not make this inference. His attention is deviated to his healthy side, and on the paralyzed side he is armed with a more or less vivid phantom, which he considers mobile and sensible because his limbs have always been that way. He is unaffected by the events that should compel the inference that he is paralyzed. He is not shocked or moved to inquiry by contradiction. He makes no serious attempt to resolve the incompatibility between his confinement to bed on the one hand and his assumed lack of paralysis on the other. His reasoning is shallow. If he “rationalizes” his confinement to bed by offering an excuse, it is usually an ad hoc solution that is often not consistent from one occasion to the next, and does not explain the variety of situations that he has experienced as a result of his paralysis. If his disability is demonstrated to him, he either dismisses it, or more often, accepts it with surprisingly little dismay, only to entertain the entirely contradictory notion that he is normal moments later. The same shallow reasoning and insensitivity to contradiction is present in the patient with denial of blindness, as illustrated by the conversations recorded in case 1 of the Appendix.

Faulty inference and poor reality judgement are difficult to explain in neurologic terms. It is clear that these cognitive defects are related to damage to parts of the nervous system that are neither exclusively sensory nor exclusively motor. When the damage involves cerebral cortex, it affects the associative cortex rather than the primary sensory areas or the motor cortex. This is understandable if one considers judgements as intermediate states between sensory stimuli and motor responses. Judgements reflect a tendency to act in a specific manner in response to a particular pattern of events perceived by the senses. The associative cortex of parietal, temporal, and frontal lobes likewise is neither sensory nor motor, but is intermediate in nature. It receives input, often via several connecting links, from all of the sensory areas, and it sends output, again over several connecting links, to motor cortex (Jones & Powell, 1970). It is therefore not unreasonable to identify an individual’s judgements with the states of activation of his association cortex and its subcortical connections. Because judgements reflect tendencies to act under given circumstances, they have an internal consistency, which is presumably reflected in the compatibility with regard to motor tendencies between successive states of activation of associative cortex. But how can one account for the faulty judgement associated with damage to this
system? How can we account for the tolerance of contradiction—the acceptance of two incompatible judgements only moments apart and the lack of any drive to resolve the contradictions?

Some insight into the pathogenesis of faulty judgement with damage to the associative cortex may follow from again considering association cortex as a parallel-distributed processing system. The essence of such a system is that when it is excited, it equilibrates, or settles, into a state, or “solution,” that is compatible with multiple constraints, or “boundary conditions”, simultaneously (McClelland et al., 1986). If damaged, the system does not cease functioning altogether, as many serially constructed mechanisms do. Rather, it continues to arrive at solutions, but if the damage is sufficient, these solutions may be incorrect. That is, a solution will satisfy some, but not all of the constraints, or boundary conditions, of the problem. Drawing an inference means coming to a judgement, a conclusion that is compatible with a variety of observations, or premises. The conclusion represents the “solution” of the problem, subject to the “boundary conditions” represented by the premises. When the reasoning mechanism—the association cortex—is defective, judgments will still be drawn, but they may no longer satisfy all of the constraints. They will thus not be “logical”. The affected individual will not be shocked by contradiction or motivated to resolve it, because such behavior would be expected only if association cortex were undamaged and able to reach its solution satisfying the full range of constraints.

The anosognosic hemiplegic is thus unable to integrate into a coherent historical structure his premorbid status, in which his limbs moved normally, and his present status of hemiplegia. To do so would be to realize his disability and would require a relatively undamaged association cortex. Instead, his premorbid status and his present disability remain disconnected. The former predominates most of the time. It would appear that the “solutions” of the damaged association cortex violate most frequently those constraints that have been most recently established. The patient’s failures on present tasks do not carry much weight against the force of habit, and he remains unaware of his disability. His phantom limb, supported by the force of habit, is judged as real because it is not effectively contradicted by his present circumstances.

V. FURTHER EXTENSIONS OF THE THEORY

The hypothesis that sensory defects are not phenomenally immediate but rather must be discovered, and the corollary that insensate fields are subject to false percepts, has proven to be a basis for understanding four distinct disorders: denial or unawareness of blindness, unawareness of hemianopsia, anosognosia for hemiplegia, and phantom limb after amputation. The clinical settings in which these occur and many of these features of each condition can be understood if two questions are an-
UNAWARENESS OF SENSORY LOSS

swered: (a) How much of a functional disability does each disorder create, and therefore how easy is the task of discovery? (b) What are the sources of induction of false percepts and therefore how strong are they likely to be?

The hypothesis can be extended to other neurological defects of which patients may be unaware. These include loss of specific skills such as aphasia and constructional apraxia, as well as broader mental defects such as impaired intellect and memory. In the case of skill defects the present theory predicts that disruption of the afferent systems which ordinarily provide immediate knowledge of the act is necessary for unawareness of the defect. Such perceptual loss, however, may not be sufficient. Defects that are easily discoverable by indirect means may become known to the patient even in the presence of such loss. In such cases impaired judgement and inference are also required. These cognitive defects are both necessary and sufficient for unawareness of intel-lectual and memory impairments. I shall discuss briefly a few of these other neurological disabilities of which patients may be unaware.

Aphasics may be unaware of their difficulty in speaking. Because the auditory and the oral proprioceptive systems ordinarily provide immediate sensory knowledge of what one has said, the present theory predicts that severe disruption of these systems is necessary for unawareness of aphasia. Discovery of aphasia when these systems are acutely damaged is probably difficult, for unawareness of aphasia is common in the acute phase following extensive temporoparietal damage to the left hemisphere. With the passage of time, however, many aphasics who were initially unaware gain some degree of awareness of their speech errors. It is possible that such recovery reflects the use of partially recovered auditory and proprioceptive pathways in the left hemisphere, perhaps in combination with the undamaged afferent pathways in the right hemisphere. Alternatively, it is possible that initial unawareness may reflect impaired capacities for observation, judgement, and inference combined with damage to the auditory and proprioceptive systems of the left hemisphere. Gaining of awareness may then reflect recovery from these intellectual defects. Weinstein et al. (1964) supported the latter mechanism, but further studies of the role of intellectual defects in unawareness of aphasia are needed.

Some patients with constructional apraxia are unaware of performing poorly in copying various designs. The reason for this unawareness becomes clear if we assume that defects in spatial perception are not phenomenally immediate. The patient must learn that he cannot construct figures properly. The most obvious means of doing so is to compare his construction with the model he is copying. However, to be successful, such a comparison requires relatively good spatial perception. The present hypothesis predicts that—other things being equal—patients with
defective spatial perception (visuospatial agnosia) will be more unaware of their constructional difficulties than patients without visuospatial agnosia. It also predicts that in patients with visuospatial agnosia, those with additional defects in judgement and inference will be more likely to be unaware of their constructional apraxia than patients without such cognitive defects.

The hypothesis that sensory defects are not phenomenally immediate but must be discovered can be extended to intellectual defects. Self-observation and inference, not merely introspection or some other form of immediate awareness, are required to become aware of defects in intellect and memory. The patient with impaired judgement, who is unable to weigh evidence and draw inferences, will be unable to discover his intellectual impairments and will be unaware of them. Thus the patient with an amnestic-confabulatory syndrome will remain unaware of his defects in memory and judgement; and the patient with frontal lobe disease who cannot shift mental set to entertain new hypotheses will be unable to discover his loss of mental flexibility.

VI. COMPARISON WITH OTHER THEORIES OF UNAWARENESS

At this point, the theory of unawareness has been developed sufficiently to compare it with other theories, particularly with regard to its assumptions and predictions. I shall conclude by discussing two general theories of unawareness that have been proposed in recent years—that of Bisiach et al. (1986) and that of McGlynn and Schacter (1989).

Bisiach et al. (1986) emphasized the modality-specificity of unawareness, and their data confirm that lack of awareness of one defect—for example, hemianopsia—may coexist with full awareness of another—for example, hemiplegia. Bisiach et al. suggest that such dissociations argue against a supramodal organ of awareness in the brain. Instead, they suggest that unawareness of defective function implies a disorder "at the highest level of organization of that function." They suggest that the required damage is in modality-specific association cortex or its connections.

In contrast McGlynn and Schacter (1989) postulate that immediate awareness requires activation of a supramodal system which is located in both the parietal and the frontal lobes. The parietal branch, called the "conscious awareness system" (CAS) receives input from activated function-specific cortex and mediates awareness of the activating stimulus. A disconnection of function-specific cortex from the CAS causes function-specific unawareness, whereas damage to the CAS itself causes global unawareness. The frontal branch receives input from the CAS and mediates the "executive functions" of initiating, organizing, and monitoring complex behaviors. Damage to this system, or disconnection from
UNAWARENESS OF SENSORY LOSS

The present theory is fundamentally distinct from either of the above views, but it does share some features with each of them. Like previous theorists, Bisiach et al. and McGlynn and Schacter assume that sensory loss is phenomenally immediate, and that unawareness supervenes only with additional damage to an "awareness mechanism" in the brain. For Bisiach there are distinct, modality-specific mechanisms in each sensory association cortex. For McGlynn and Schacter there is a single supra-modal mechanism in the parietal and frontal lobes. In contrast, the present theory assumes that sensory loss is not phenomenally immediate but rather must be discovered or inferred. There is no specific awareness mechanism in the brain. Like Bisiach, the present theory emphasizes the modality-specificity of unawareness, but this is because the sensory systems themselves are anatomically distinct, not because there is an awareness mechanism at the cortical end of each sensory pathway. The present theory asserts that unawareness is the immediate consequence of damage to any portion of a sensory system—from transducer to cortex—and that awareness follows if the neural apparatus for discovery is preserved. Like McGlynn and Schacter, the present theory emphasizes the importance of polymodal parietal and frontal cortex, but this is because that cortex is essential to attentional and intellectual processes needed for discovery of a sensory defect, not because it constitutes a special awareness mechanism. For McGlynn and Schacter the parieto-frontal cortex is essential not only for awareness of sensory loss but also for awareness of sensations themselves. Both forms of awareness are equally immediate. The present theory recognizes a distinction between the phenomenal immediacy of sensations, and the discovery required for sensory loss. The parieto-frontal cortex is more important for the latter than for the former.

It should be possible to distinguish the present theory from the others with careful clinical and experimental observations. The theory of Bisiach et al. predicts that: (a) Modality-specific unawareness cannot occur following peripheral sensory loss, even with superimposed generalized brain disease. (b) Modality-specific unawareness of any defect can occur with perfectly normal intellect. (c) Unawareness of hemiplegia can occur with intact proprioception. Similar predictions follow from the theory of McGlynn and Schacter. Since modality-specific unawareness requires disconnection of modality-specific cortex from the CAS, it cannot be caused by peripheral sensory loss. Because the disconnection is highly specific for awareness, modality-specific unawareness need not be accompanied by other cognitive defects. Since the disconnection spares the sensory system itself, unawareness of defects such as hemiplegia requires no sensory loss. In addition McGlynn and Schacter's theory
predicts that (d) damage to the parietal lobes should cause unawareness of sensation from an intact sensory system just as easily as it causes unawareness of sensory loss from an interrupted sensory system. The present theory does not support these predictions: (a) It allows for modality-specific unawareness of peripheral sensory loss, as reported by previous observers (Redlich & Bonvicini, 1908; Pötzl, 1924) (b) It predicts that additional cognitive defects are required for some forms of unawareness. Specifically, easily discovered defects such as blindness can only occur with severe defects of intellect, but defects that are hard to discover—such as hemianopsia or the normal blind spot—may escape awareness even in an intellectually normal subject. (c) The present theory also predicts that defective proprioception is a necessary but not a sufficient condition for anosognosia for hemiplegia. (d) Finally, damage to frontal and parietal cortex will cause unawareness of loss of function in interrupted sensory systems far more easily than it will cause unawareness of function in sensory systems that are intact.

APPENDIX

Case 1

CD, a 68-year-old retired pipe-fitter, was vacationing with his wife in Florida on December 21, 1983, when she noted that he had suddenly become blind and confused. A neurologist found him to be cortically blind and recommended hospitalization, but the patient refused. He returned home to Maine and was hospitalized on January 1, 1984. He was disoriented to time and place and completely blind with no light perception. His speech was fluent but there was paraphasia and difficulty following some spoken commands. His affect was euphoric. CT scan showed bilateral occipital infarctions. Over the next 10 days his aphasia improved, he was better oriented, but he remained blind. He was transferred to Spaulding Rehabilitation Hospital in Boston on January 11, 1984.

His past medical history was notable only for mild hypertension and migraine headaches. The general physical examination was normal. On neurologic examination he was awake and he spoke fluently with occasional paraphasia. He could repeat well, and forward digit span was 6. He could follow simple spoken commands but had difficulty once the command involved more than two steps. He erred on some oral calculations (37 − 12 = 27; 8 × 11 = 44) but not others (10 + 10 = 20; 5 × 5 = 25). Right-left discrimination was usually correct. He usually designated named fingers correctly, but occasionally confused the ring and index fingers. He identified common objects by touch and was able to use them properly.

His memory was very poor. He was given three words to remember, and after 1½ min mostly occupied by counting by threes, he could not
recall that he had been given any words. He could not sustain mental effort: When asked to add threes serially, he repeated the examiner's examples—2, 5, 8—but could not go on. Asked to list animals, he listed four and then stopped. He could not interpret proverbs, and was content to reread or paraphrase part or all of each one.

He was totally blind with no light perception and no ability to detect gross motion. His pupils reacted promptly to light, and ophthalmoscopic examination disclosed normal fundi except for mild arteriolar narrowing. The range of eye movement was full. The other cranial nerves were normal, there was no limb weakness or ataxia, somatic sensation was preserved, and there were no pathological reflexes.

He was unaware of his blindness. At times he explicitly denied being blind, while at others—only moments later—he might admit to some difficulty, but still not fully appreciate the problem. The following conversation took place on January 12, 1984.

What happened to you?  
Do you know where you are?  
What are you doing in the hospital?  
Is anything wrong with you?  
Can you see?  
Perfectly?  
Are you weak?  
How many fingers am I holding up? (4)  
I thought you said you could see well?  
Why can’t you tell how many fingers?  
Why not?  
Yet you see perfectly.  
I’ll put this in your hands (a green book). Tell me what color it is.  
So you can’t see the color.  
Can you see my face?

"Nothing. I just had a routine blood examination"  
"Sure . . . the Portsmouth Hospital"  
"Filling in . . . for Mr. Johnson"  
"No doctor, nothing whatever"  
"Yes, I can see"  
"Yes . . . yes" (sighs)  
"No, no"  
"I can't see how many, three I guess . . . I don't know"  
"I can see well!!!" (raising his voice)  
"I can't attempt to tell how many you have"  
"Why not? God I don't know . . . because I can't tell"  
"Yes"  
"I can't. I don't know. All I know is it's a book"  
"If that's the case, I can't"  
"No, not too good"

His imagery for animal shapes, directions, and colors was difficult to assess, because his answers were at times correct and at others grossly
wrong. This was also the case for questions that probed his fund of
knowledge but did not draw upon visual imagery.

He continued under observation. One week after admission he re-
mained blind, but his statements about his vision varied from one moment
to the next. For example, he said once that his eyesight was ‘‘fair, not
perfect . . . or maybe it is.’’ The following conversation took place on

How is your vision, is it okay?  ‘‘Yes’’
Where are you?

‘‘Massachusetts Rehabilitation
Center’’ (correct)

‘‘Boston’’ (correct)

‘‘I don’t know’’

‘‘The third of January’’

‘‘I don’t know’’

‘‘The fifth’’

‘‘The second’’

‘‘Nineteen, oh . . . I’ll say 1905’’

‘‘I’m 1984 . . . same as that is’’

What town is this?

What is the date?

What is it?

What year?

Take a guess.

What year is it?

Nineteen . . . ?

No, it’s 1984. How old are
you?

What is your birthdate?

What year?

Do you see anything?

‘‘November 15’’ (Nov. 14)

‘‘1915’’ (correct)

‘‘No, I can’t see anything, doc. I
can’t see a thing. That bothers
me’’

One week later he had improved mentally. He was usually, but not
always aware of where he was. Calculations were better and sustaining
mental effect was much improved. He was now consistently aware that
he could not see. Ironically, at this time light perception first returned.
When taken into a pitch black room, he could reliably detect a flashlight
being turned on and off in his central vision. On February 1, 1984 we
had the following conversation.

What happened to you?

‘‘I haven’t the slightest idea doc,
I know it’s awful . . . I can’t
see a thing’’

Do you know where you are?

‘‘Yes, in the Portsmouth
Hospital’’

What are you doing in the
hospital?

‘‘What am I doing in the hospi-
tal, doc? I don’t know . . .
trying to get better I guess’’
Is there anything wrong with you?

"Is there anything wrong with me? God only knows, doc. I can only tell you that I can't see and when it gets dark I see even less... I see nothing"

Can you see at all?

"Can I see at all? Right now I can't see a thing"

Are there times that you can see?

"No doc. No there are not. It's all the same"

CT scan on January 31, 1984 showed large infarctions on the medial aspect of the occipital lobes bilaterally.

He was discharged home with his wife on February 1, 1984.

Case 2

JK, a 74-year-old retired truckdriver, was in good health until early 1981, when he developed brief episodes of inability to talk. These occurred every 2 to 3 weeks, but later they increased in frequency, culminating in the sudden onset of more lasting aphasia, mild right hemiparesis, and "confusion" on December 23, 1981. He was admitted to a local hospital. CT scan showed a left parietal infarction. Diabetes mellitus was discovered, and insulin injections were begun. Over the following week the language difficulty and confusion greatly improved. On January 3, 1982, bilateral carotid angiography was performed, revealing extensive intracranial vascular stenoses in the territory of the anterior and middle cerebral arteries. Following the angiogram he was more confused and lethargic. A few days later he was noted to be completely blind, although in retrospect his wife had noticed something wrong with his vision immediately after the arteriogram. An ophthalmologist diagnosed cortical blindness. Over the next 2 weeks his mental confusion improved slightly and it was thought that occasionally he could count fingers or distinguish some colors. He was transferred to Spaulding Rehabilitation Hospital on January 21, 1982.

Detailed neurologic examination was carried out on January 29, 1982. He was awake and alert, but his attention wandered. Speech was fluent with occasional semantic paraphasias and perseveration. He could repeat and understand short statements and questions but not longer, more complex items. Naming objects by touch was impaired with many bizarre answers and much perseveration. His memory for recent events was poor; he could recall none of three items after 5 min. Although aware that he was in a rehabilitation hospital, he thought it was located in Revere instead of Boston. He gave the date as February, 1917. His memory for remote events was also impaired, and he could not recall
his address or when he was married. He could not sustain mental effort in serial tasks such as counting backwards.

He was nearly totally blind. He did not fixate objects with his gaze and often lay with his eyes closed. With eyes opened he could not identify objects or colors, but he did object when a bright light was shined in his eyes. He also appeared to detect gross motion when the examiner waved his hands to either side. There was no reaction to visual threat. The pupils reacted sluggishly to light, and ophthalmoscopic examination disclosed normal fundi. The range of eye movement was full. The remaining cranial nerves were normal. There was mild weakness of the right leg, but strength was otherwise normal. Somatic sensation was preserved. He walked independently with short steps but collided with obstacles. Tendon reflexes were symmetric. There was a marked sucking reflex and there were bilateral grasp reflexes, more intense on the right. Intermittent paratonic rigidity and general restlessness were present.

On this occasion he was aware of his visual difficulty. His main complaint was that he could not see. He claimed to be absolutely blind and that this upset him. He could not estimate how long he had been blind but believed that it was of recent onset.

Six days later his vision had not improved, but he denied being blind. When asked to describe the examiner he confabulated. When asked to describe the lady (who did not exist) in the room, he again confabulated a description. His memory had not improved, but he was less fidgety.

Throughout the hospitalization he was intermittently agitated, with paranoid delusions. By February 12, 1982 his vision had improved slightly. He counted fingers accurately on 3 of 5 occasions and identified a color 3 of 4 times. He managed to avoid several obstacles placed in his path and found his way to the door of his room once but not on a second trial. He could not find his armchair except by feeling about for it.

He was discharged home on February 24, 1982.

Case 3

SW, a 29-year-old physician, was traveling in Europe when she became ill with diarrhea and weakness. On November 23, 1987, while waiting to be examined at an emergency ward in Amsterdam, she suffered a cardiopulmonary arrest and was found unconscious. She was resuscitated but remained comatose for approximately 2 weeks. She left Amsterdam for the United States on December 17, 1987, at which time she was quadriparetic and anarthric. She had a nasogastric tube for feeding and an indwelling urinary catheter. She was admitted to St. Raphael's Hospital in New Haven and was transferred on January 4, 1988 to a rehabilitation hospital. At that time she could speak and eat, and she could urinate spontaneously. Her limbs were stiff and movements were
awkward. She was treated with serial casts to lengthen shortened muscles.

On April 25, 1988 she sought consultation because of poor vision, which she considered the main residue of her illness. She said that her memory had initially been poor but had improved to near normal. She recalled the flight from Amsterdam to the United States and most subsequent events. When asked if she had been totally blind at any time, she said that she did not know—that she “didn’t even think about it” until she was at St. Raphael’s Hospital in New Haven. There, at one point, her brother asked her to read some addresses on a sheet. She could not see them and realized there was a problem with her vision. At first she could not identify colors and textures, but later she could. She still had much difficulty identifying objects, analyzing each object piece by piece until she could infer its identity. She could not read and had difficulty identifying letters.

As a child she had been precocious and achieved academic distinction. She had been a gifted amateur artist, specializing in charcoal portraits. She was fluent in four languages and felt that her ear for music and languages had not suffered in her recent illness.

On examination she was alert and oriented to place and date. Speech was clear and coherent with no paraphasia. She could repeat and understand speech normally. There was some difficulty spelling: although she correctly spelled “table”, “floor” and even “majority”, she made errors with “ceiling”, “museum” and “physician”. Simple mental calculations were slow and usually correct, but she could not solve $7 \times 9$. She could name her fingers properly when touched, including discrimination between left and right.

Her memory for recent events was quite good. She was slightly slow in her flow of thought and was able to name only twelve animals in one minute. Reasoning by analogy was good, and she interpreted proverbs accurately, only occasionally showing slight imprecision.

The major difficulty was her visual perception. She could not identify most common objects although they clearly caught her gaze, and she tracked the objects if they moved slowly. She often guessed at the identity of an object based on one or two details. Identification by touch was much better but not normal. She could not count black dots on a white page or the examiner’s fingers before her eyes reliably. She quickly identified the surface colors of large objects, however. In reaching for objects that she had fixated, she frequently reached past the object but usually didn’t miss by much. Visual pursuit was inaccurate as the target’s velocity increased.

In contrast to her perception, visual and spatial imagery were quite good. She described faces and animals accurately, knew the direction
of travel between cities, and knew the colors of all objects named by the examiner.

Peripheral visual fields were full. The pupils reacted to light. Ophthalmoscopic examination disclosed no abnormality. The eyes moved conjugately and fully. Her face was rather inexpressive but was more animated when she discussed an idea that concerned her. Movements of her hands and arms were slow and somewhat awkward, and she had difficulty manipulating objects. She could stand with her feet together for only a few seconds before she needed support. When she walked, her legs overadducted and she needed assistance. She localized tactile stimuli accurately, and there was only mild proprioceptive difficulty.

REFERENCES


UNAWARENESS OF SENSORY LOSS


