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Unawareness of Deficits in Neuropsychological Syndromes*

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ABSTRACT

Damage to different regions of the brain can cause a variety of neuropsychological deficits, including specific disturbances of language, memory, perception and motor function. A significant number of brain-damaged patients are unaware of their deficits, even when they are profound and have debilitating effects on patients' performance. This article reviews clinical observations and experimental investigations concerning unawareness of deficits, considers methodological issues, and critically evaluates different interpretations of the phenomenon. An integrative theoretical framework is proposed to account for unawareness of deficits in diverse neuropsychological syndromes. Possible directions for future research are outlined.

Damage to different areas of the brain can produce a wide variety of cognitive and behavioral impairments. Selective disorders of language, perception, attention, memory, action, planning, and spatial orientation have all been described and analyzed extensively in the neuropsychological literature. In their most severe form, such disorders can virtually eliminate a patient's ability to perform a specific cognitive or motor function. For example, amnesic patients may be unable to remember a conversation or a salient event after just a few minutes of distraction; certain kinds of aphasic patients are unable to produce even a few words of semantically coherent speech; and hemiplegic patients typically cannot initiate or perform motor activities with their affected limbs.

In view of the debilitating and frequently dramatic nature of the disorders that result from brain damage, it is perhaps surprising to discover that a

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significant proportion of patients are entirely unaware of their deficits: Some amnesic patients claim that their memory is perfectly normal, aphasic patients frequently do not know that their linguistic productions lack coherence and meaning, and hemiplegic patients often do not realize, and sometimes deny, that they have a motor impairment. Yet these same deficits are all too apparent to others and have a profound effect on afflicted patients' everyday lives.

The fact that brain-damaged patients are sometimes unaware of their deficits has potentially important theoretical and clinical consequences. On the theoretical side, observations of unawareness raise questions concerning the nature of the processes and mechanisms that normally permit people to be aware of and monitor the state of their own cognitive functions. On the clinical side, unawareness of deficits represents a difficult obstacle for rehabilitation efforts; if patients are not aware that they have a particular disorder, then they are unlikely to benefit from or even participate in any remedial interventions. Similarly, patients who are unaware of their deficits will likely pose serious problems for family members and other caretakers because they may insist on undertaking activities that they can no longer perform, such as returning to work.

In view of these important theoretical and clinical issues, it seems clear that developing an adequate understanding of unawareness of deficits represents an important task for neuropsychological research, particularly because the phenomenon has been reported in numerous patient groups. This article attempts to contribute to such an understanding by providing a detailed review of existing literature concerning unawareness of deficits in neuropsychological syndromes. Although relevant observations have been made throughout the past 100 years, they derive from diverse and often unrelated areas of investigation; no integrative reviews of the literature exist. However, a growing number of investigators have recently expressed interest in unawareness of deficits in various patient groups. Accordingly, the time appears ripe to bring together relevant evidence and ideas, offer a critical analysis of them, delineate key methodological and theoretical issues, and suggest directions for future research.

The article consists of four main sections. The first considers briefly some of the terminology that is used frequently in the literature and will thus appear repeatedly throughout the article. The second provides a relatively detailed review of empirical observations concerning unawareness of deficits in various neuropsychological syndromes as well as a critical overview of the shortcomings of existing research. The third section turns to theoretical ideas that have been proposed to account for unawareness of deficits, discusses their plausibility, and sketches a general theoretical framework for conceptualizing unawareness. The fourth and final section considers possibly fruitful directions for future research, drawing on findings and ideas from cognitive, social, and developmental psychology.

TERMINOLOGY AND CRITERIA

A variety of terms have been used to describe the phenomena that we shall consider. *Anosognosia,* which was introduced by Babinski (1914), refers to lack of knowledge, awareness, or recognition of disease. This term has been used most frequently in reference to unawareness phenomena observed in hemiplegia and hemianopia attributable to stroke. The terms *lack of insight* and *imperception of disease* have also been used by some investigators to refer to diminished awareness of neuropsychological deficits. Consistent with their usage in the literature, we shall use the terms anosognosia, unawareness of deficits, lack of insight, and imperception of disease interchangeably. The terms *denial of illness* or *denial of deficit* are also encountered frequently, but they have been used in two different ways - first, in a general and theoretically neutral sense that is roughly equivalent to unawareness or anosognosia, and second, in a narrower and theoretically committed sense that implicates the involvement of the psychological defense mechanism of denial. When used in the latter sense, denial signifies a motivated reaction by a patient who may be in some sense "aware" of his or her deficits but is unwilling to confront them. We will use the terms "motivated denial" and "defensive denial" solely in reference to this hypothetical defense mechanism. In contrast, the terms *anosognosia, unawareness of deficits, lack of insight,* and *imperception of disease* will be reserved for those cases in which patients are *unable* to become aware of a neuropsychological deficit. A problem also arises with the use of the terms *indifference* or *lack of concern* in discussions of unawareness. These terms refer to diminished affective responses to a neuropsychological impairment, but are sometimes used interchangeably with anosognosia and unawareness. However, altered affect does not necessarily imply that patients are unaware of their deficits. Therefore, although indifference may suggest some degree of unawareness, it should not be equated with anosognosia.

One of the deficiencies characteristic of many existing studies is that they do not define key terms and concepts explicitly. In addition, much of the evidence that we shall review is based on clinical observation and contains little or no information concerning the criteria that were used to assess unawareness. Indeed, one of the main points that we will make is that much more attention needs to be paid to defining concepts operationally and devising systematic measurement techniques. However, rather than stating repeatedly that individual studies have failed either to define concepts explicitly or to state exactly how they measured unawareness, we will first report the observations of interest and save the critical assessment for the end of each section. When investigators have defined their terms and/or provided explicit measurement criteria, we will describe them. Despite the evident lack of rigor in many studies, we think that they should be included in any comprehensive review, both for historical reasons and because they provide useful qualitative information concerning the phenomena of interest.

REVIEW OF EMPIRICAL EVIDENCE

This section of the article is subdivided into seven main subsections corresponding to neurological and neuropsychological syndromes in which unawareness has been reported frequently: hemiplegia, Anton's syndrome, hemianopia, amnesia, head injury, dementia, and aphasia. The review of evidence in each of these subsections is followed by a brief critical assessment of the relevent research. An additional subsection considers miscellaneous observations on unawareness that do not fit into any of the other categories.

Hemiplegia

Hemiplegia refers to paralysis on one side of the body caused by a lesion in the contralateral hemisphere. Beginning with the observations of Babinski (1914), numerous investigators have reported anosognosia for hemiplegia. Two general stages of research can be delineated. The first dates from Babinski's paper to the publication of Weinstein and Kahn's (1955) classic monograph on denial of illness, and consists largely of clinical observations concerning individual cases and small series of cases. The second phase of research, beginning in the early 1960s, is characterized by larger patient groups and in some instances by the use of more systematic investigative techniques.

Early clinical studies. Gerstmann (1942) provides a general depiction of the clinical presentation of the phenomenon:

> The hemiplegia is usually of the left side of the body. The patient behaves as though he knew nothing about his hemiplegia, as though it had not existed, as though his paralyzed limbs were normal, and insists that he can move them and can walk as well as he did before. Asked to lift up both arms, he naturally moves the healthy one only, but maintains that he has raised the disabled one also. Requests for movements with the paralyzed left arm or leg are performed by him merely with the healthy one, or not at all, but at the same time he is convinced that he has carried out the task. The patient may pay no attention to the paralyzed side, but even refuse to look at it or turn away to the right. If such a patient is shown the affected arm or leg as being attached to his body, he will often remain indifferent or will declare that it is not his or that someone else's is in his bed, and the like. It is as though the patient experienced the paralyzed limbs as absent (p. 891-892).

In his original description of anosognosia for hemiplegia, Babinski (1914) emphasized that anosognosia can occur in the absence of mental confusion, confabulation, and hallucinations. He described two stroke patients with left hemiplegias in whom the anosognosia lasted several months and was associated with preserved intellectual functioning for a short period following stroke, although some generalized mental deterioration was later observed. Interest-

ingly, when asked precisely what her problem was, one patient replied that she was troubled by a backache and phlebitis, but explicity denied her left-sided paralysis. Babinski also reported on several hemiplegics who were aware of their deficits but appeared entirely unconcerned about them. He referred to this condition as *anosodiaphoria,* meaning indifference to illness.

Barré, Morin, and Kaiser (1923) described a 60-year old man with left hemiplegia and left hemianopia who had preservation of intellectual function but was totally unaware of his paralysis. Etiology was not provided but the clinical description is suggestive of stroke. The authors concluded that anosognosia is characterized behaviorally both by imperception and denial of the defect: "...there is a real obstinacy to not admit it, a resistance to the recognition that is truly striking and a little disconcerting when it is found in a subject whose intellectual faculties are otherwise well preserved" (p. 501). Barkman (1925) described a 53-year old female stroke patient who was unaware of her severe left hemiplegia, but showed no other clinical signs of mental impairment.

Another case of anosognosia, reported by Joltrain (1924), merits consideration because it concerns a patient whose premorbid mental state had been observed by clinicians for quite some time. She had been monitored for her diabetes and hypertension before sustaining a stroke. Following her stroke the patient had left hemiplegia with anosognosia that was not accompanied by any other noticeable mental disturbance. Joltrain asserted that there was no difference, except for the unawareness, in her psychological functioning compared to before the attack. The patient was able to describe what happened to her, she remembered having fallen and not being able to get up, but said: "It is curious, it is as if I had been paralysed" (p. 638). The anosognosia lasted until death, 20 days later. Von Hagen and Ives (1937) were the first to report a series of patients with anosognosia for hemiplegia. The hemiplegia was on the left in five cases and on the right in one right-handed patient. An autopsy performed in one case of left hemiplegia revealed several cerebral abscesses, one involving the right thalamus and adjacent structures. Anosognosia for hemiplegia was associated with homonymous hemianopia and conjugate deviation of the head and eyes to the side of lesion in four cases, impaired touch and pain perception on the paralyzed side in all cases, and impaired position sense in at least one of the paralyzed limbs in all five cases for which it was examined. Two patients whose anosognosia could be followed to recovery became aware of their disability 15 days and 3 days following onset of hemiplegia, respectively. One notable case in this series concerned a 76-year old stroke patient who was well aware of her severe memory impairment and paralysis of the left upper limb but denied paralysis of the left leg, thereby indicating that anosognosia can be highly specific.

In a subsequent report, Von Hagen and Ives (1938) confirmed their earlier observation of anosognosia following right-hemisphere damage, a finding that was soon confirmed by others (Cobb, 1947; Waldenstrom, 1939). In contrast, several other early case studies reported anosognosia following left-hemisphere

lesions (Denny-Brown & Banker, 1954; Olsen & Ruby, 1941; Paterson & Zangwill, 1944). However, it was difficult in these cases to rule out unequivocally the possibility of right-hemisphere damage.

Ives and Nielsen (1937) offered one of the first distinctions between different forms of unawareness associated with hemiplegia: anosognosia and delusion of an absent body part. Anosognosia was considered a lack of recognition of hemiplegia whereby patients believe that their limbs are functioning normally, whereas in delusion of an absent body part patients deny ownership of their limbs. Ives and Nielsen reported two cases of patients who were aware of their left hemiplegia, but when shown their own left limbs, claimed that they did not belong to them. The authors argued that this type of delusional disturbance can be caused by focal cerebral lesions and is markedly different from anosognosia for hemiplegia. Autopsies were performed on the two patients with delusions. Results showed lesions of the right thalamus, thalamoparietal peduncle and retrolenticular internal capsule in one case, and lesions in the right parietal lobe and centrum with destruction of the thalamoparietal peduncle, sparing the thalamus, in the other.

Nielsen (1938) elaborated on the distinction between anosognosia and delusional disturbance by comparing the anatomical substrates underlying the two phenomena. Nielsen presented five new cases of body scheme disturbances involving inattention to the left limbs or delusion of their absence. All five had damage to the right thalamoparietal peduncle. Nielsen compared lesion sites of previously reported cases of anosognosia with those cases of inattention to or delusion of absence of the limbs (usually on the left side of the body). It was concluded that unawareness of hemiplegia is caused by an intrathalamic lesion or isolation of the thalamus from the frontal, parietal, and temporal cortex, whereas delusion of absence of the limbs results from a lesion of the thalamoparietal peduncle. Similarly, Spillane (1942) discussed a patient who was unaware of his left hemiplegia and experienced delusions about the left side of the body following a ruptured aneurysm in the right parieto-thalamic region. Sandifer (1946) described a patient who showed anosognosia of left hemiplegia and left hemianopia, confusion, neglect of left space as well as the left side of the body, and a delusion that her left limb did not belong to her. Autopsy findings showed softening of the right thalamoparietal region. Sandifer pointed out that the failure to recognize a body part as one's own or the illusion that a part is absent may contribute to the development of anosognosia when that part becomes paralyzed, but are not sufficient conditions for unawareness of hemiplegia. Gerstmann (1942) reported a case involving delusion of absent body parts that illustrates the subjectively compelling nature of the delusion. This patient claimed that:

> ...another person was in bed with her, a little Negro girl, whose arm had been slipped into the patient's sleeve. She felt the supposedly foreign arm and leg as rather warm and heavy against her body, and, because the foreign limbs

were never moved, she thought that her bedmate was constantly asleep. In an effort to awaken her, the arm was pinched and tossed about by the patient's right arm. She felt some pain but did not associate it with the pinching, being entirely unaware of the status of her left side (p. 894).

Many early case reports of anosognosia for hemiplegia were characterized by the apparent absence of generalized confusion and intellectual deterioration, and this issue was pursued further by several later investigators. Cohn, Neumann, and Mulder (1947) reported two cases of anosognosia in which confusion and disorientation were observed. The first patient had a left hemiplegia caused by an expanding tumor in the right temporal region. Case 2 was a stroke patient with a left hemiplegia and left homonymous hemianopia. Autopsy showed a lesion in the right temporal and parietal lobes, insula and corpus striatum possibly caused by an embolism in the right middle cerebral artery. An area of softening was found in the region of the inferior parietal lobule. The thalamus was not involved, but its major cortical connections were destroyed. Bender, Wortis, and Gordon (1949) studied 10 patients with anosognosia, 8 of whom had a left hemiparesis. All patients showed severe intellectual impairment and extreme defects in visual and cutaneous sensations. Eventually, patients became aware of the defect and this awareness coincided with clearing of consciousness.

Roth (1949) claimed that generalized confusion and intellectual impairment are not necessary conditions of anosognosia. He described two patients with anosognosia of left hemiplegia and neglect of the left side of the body. Case 1 had a tumor in the right parietal region. The hemiplegia progressively worsened over a period of several weeks at which time the patient had developed a severe memory deficit. She confabulated to fill in memory gaps, and she had become disoriented and euphoric. Roth conceded that the intellectual impairment and affective disturbance likely contributed to the appearance of anosognosia but he argued that the patient had exhibited anosognosia of her hemiplegia before any clouding of consciousness or mental impairment, except for some indifference, was detected. Case 2 concerned a patient with an extensive fronto-parietal lesion who, in addition to his left hemiplegia, anosognosia, and neglect, also had a left hemianopia, poor memory for recent events, and was disoriented for time. This patient was unaware of his paralysis, but complained bitterly about his other problems. Twelve days following onset of hemiplegia, the patient began to pay more attention to his left side and became increasingly aware of his disability. Roth stressed that this patient remained deluded about his left side for almost 2 weeks, during which time confusion was absent and the only intellectual defect was memory impairment for recent events. Similar observations were reported by Gilliatt and Pratt (1952).

Nathanson, Bergman, and Gordon (1952) further explored the relation between anosognosia and confusion. They found evidence of either partial or complete anosognosia in 28 out of 100 cases of hemiplegia. All anosognosia patients exhibited some degree of disorientation and there was a direct relation

between the two - that is, patients who were unaware of their paralysis when they were extremely disoriented often admitted the defect with clearing of consciousness. It was indicated that, in all cases where spatial disorientation was present, patients reported being at home or somewhere less suggestive of sickness than a hospital. The authors interpreted this as defensive denial rather than lack of awareness and supported their contention with the observation that patients would spontaneously make remarks indicating knowledge of where they were even though, when directly questioned of their whereabouts, they claimed being elsewhere.

Further evidence pointing toward a relation between anosognosia and intellectual impairment was reported in a series of studies by Weinstein and Kahn (1950, 1953, 1955). Weinstein and Kahn (1950) initially studied 22 patients with brain tumors who showed anosognosia and denial of various defects including hemiplegia, visual defects, craniotomy, illness, incontinence, and other problems associated with their condition. Confabulation and disorientation were observed in all 22 patients, all 22 patients exhibited anosognosia for more than one defect, and 15 patients exhibited a marked affective disturbance in the form of euphoria and unconcern. Electroencephalographic testing was performed in 17 cases, 16 of whom showed diffuse bilateral abnormalities. Weinstein and Kahn (1955) confirmed and extended these findings in a later study of 52 brain-damaged patients with various etiologies (primarily tumors and vascular disease). Interestingly, following recovery from anosognosia, most patients were amnesic for their earlier unawareness. Anosognosia and disorientation were reestablished in patients who had seemingly recovered by administering intravenous injection of sodium amytal (similar observations were reported by Guthrie and Grossman, 1952).

Modern research. There was a decline in the number of publications concerning anosognosia for hemiplegia in the years immediately following Weinstein and Kahn's (1955) monograph, but a significant number of new problems and issues have been addressed in various studies during the past 25 years.

Ullman (1962) attempted to delineate features of anosognosic and personification reactions in patients without overt anosognosic syndromes. Sixty-seven patients were questioned about their subjective reactions to their motor deficits. Thiry-five of them described transient abnormal perceptual experiences that had occurred following the paralysis, such as feelings that the limb was separated from the body or as if it did not belong to them. These experiences were referred to as "the potential anosognosic response" because they seemed to represent the "precursor of what, under conditions of greater brain damage and diffuse dysfunction, emerged clinically as either anosognosia or imperception of a bodily part" (p. 91). It was emphasized that the effects described by these 35 patients occurred in association with a clear mental state and "insight into the subjective nature of what they were experiencing" (p. 91). Thus, disorientation and confusion were regarded by Ullman as crucial factors for

producing a full blown anosognosic response. Ullman, Ashenhurst, Hurwitz, and Gruen (1960) had previously reported the "potential anosognosic response" in 15 of 34 stroke patients who did not show unawareness of hemiplegia. Interestingly, 11 of the 15 potential anosognosic patients had right hemiplegias attributable to left-hemisphere damage.

Several studies have reported novel methodologies for studying anosognosia. Cole, Saexinger, and Hard (1968) employed the method of intravenous anesthesia (using lidocaine) to investigate anosognosia experimentally in 22 brain-damaged patients. Following lidocaine injection to an arm, increased loss of sensorimotor function was demonstrated to the patient by neurological examination. Patients were then asked if there was any alteration in the functioning of their limb compared to before the injection. Eleven patients exhibited anosognosia: 5 during the lidocaine experiment, 4 during clinical examination, and 2 in both conditions. Less than one-third of the non-anosognosic group showed intellectual impairment whereas all patients with anosognosia exhibited confabulation, ludic behavior, and delusions, whether the anosognosia was present initially or only following lidocaine administration.

Green and Hamilton (1976) used somatosensory-evoked potentials to study nine patients with anosognosia for left hemiplegia and one patient with anosognosia for right hemiplegia. In the former nine patients, somatosensory-evoked potentials were nonexistant over both hemispheres during stimulation of the left median nerve. These patients all had acute lesions of the right hemisphere, that is, infarction or hemorrhage. The single patient with anosognosia for right hemiplegia (caused by thalamic hematoma) showed no response over either hemisphere on stimulation of the right median nerve. More recently, Mauguíere, Brechard, Pernier, Courjon, and Schott (1982) revealed that, following auditory stimulation, evoked potentials were absent in the right temporal lobes of patients who had previously been anosognosic for their left hemiplegia. In contrast, auditory-evoked potentials were equally distributed over the right and left hemispheres of patients who had always been aware of their motor defect.

Cutting (1978) developed an anosognosia questionnaire to study 100 hemiplegics during the acute stage of a cerebrovascular accident. The questionnaire included some general questions (e.g., Why are you here?) and some specific questions about the affected limb (e.g., Do you feel the arm is strange or odd?). Of the 48 patients with left hemiplegia, 28 (53%) denied the weakness, whereas only 3 of the 22 (14%) right hemiplegics denied their disability. The most significant correlates of anosognosia were visual field defect, visuoperceptual deficit (impaired picture identification), and apathetic mood. Disorientation was evident in all 3 patients with anosognosia for right hemiplegia whereas 19 of 28 patients with anosognosia for left hemiplegia were disoriented and an additional 4 had impaired memory. Abnormal attitudes such as indifference and "nonbelonging" emerged at a later stage in the aftermath of a stroke than did anosognosia. Based on these findings, Cutting argued that the occurrence of

anosognosia is not dependent on right-hemisphere damage. In addition, it was established that a confusional state was not consistently associated with anosognosia and was often present in patients without the disorder. Cutting, consistent with earlier investigators whose assessment techniques were somewhat less systematic, observed considerable specificity in anosognosic phenomena - some patients would admit to a heart attack and even a stroke, but would remain unaware of weakness in half the body (see Bisiach, Valler, Perani, Papagno, & Berti, 1986, and Willanger, Danielsen, & Ankerhus, 1981a, for similar results).

An issue that has attracted considerable attention in recent research concerns the relation between anosognosia for hemiplegia and the phenomenon of unilateral neglect - unawareness of and inattention to the side of space contralateral to their lesion. The very existence of neglect could be construed as a form of anosognosia, inasmuch as unawareness (of the perceptual world) is a defining characteristic of the syndrome. However, although they may appear together in the same patient, anosognosia and neglect have different etiologies and can be dissociated from one another (Cutting, 1978; Frederiks, 1969, 1985a; Hemphill & Klein, 1948; Weinstein & Cole, 1963; Weinstein & Friedland, 1977; Welman, 1969): many neglect patients are aware of the nature of their problem. Nevertheless, Weinstein and Friedland (1977) reported that anosognosia and disorientation were observed significantly more often with severe hemineglect than with mild hemineglect in patients with left-hemisphere and right-hemisphere lesions. This result suggests that there is some association between anosognosia and unilateral neglect.

Heilman and Valenstein (1972) studied the relation between auditory neglect and anosognosia. Patients were considered to have auditory neglect if they consistently identified unilaterally presented auditory stimuli but always missed one side with bilateral simultaneous stimuli. Anosognosia was assessed during a neurological examination and was defined as unawareness of either hemiparesis or illness. Ten patients with auditory neglect were included in the study. Brain scans revealed that in nine cases the right inferior parietal lobule was involved and in one case the left frontal lobe was damaged. Five of the nine patients with right-hemisphere damage showed anosognosia in addition to the neglect whereas the case with a left-hemisphere lesion did not exhibit anosognosia.

Willanger et al. (1981b) assessed anosognosia for hemiplegia and unilateral neglect in 55 stroke patients with right-hemisphere lesions. A strong association was reported between unawareness of hemiparesis and visual neglect. Of the 14 patients with denial of hemiparesis, 10 showed visual neglect. On the other hand, visual neglect can occur without denial/neglect of hemiparesis as it did in 10 of the 43 patients with hemiparesis.

Two more recent studies concerned with behavioral abnormalities following right-hemisphere stroke and recovery from the disturbances (Hier, Mondlock, & Caplan, 1983a, 1983b) provide information concerning the relation between anosognosia and neglect, as well as other impairments. Hier et al. examined 41

patients within 7 days of onset of stroke and reexamined them at 2-4 week intervals until recovery was complete. Anosognosia correlated most highly (.82) with motor impersistence, which was tested by timing the interval that the patients could keep their eyes closed on command. Anosognosia correlated significantly with degree of leg weakness (.53), implying that patients with more severe hemiplegia were more likely to be unaware of disability, and with neglect (.42), indicating that the two disturbances tended to appear together. Significant correlations were similarly found between anosognosia and dressing apraxia (.51), inability to name familiar faces (.46), left-sided extinction on double simultaneous stimulation (.46), constructional praxis (.42), and arm weakness (.36). A significant association was found between anosognosia and extent of injury to the frontal, parietal, and temporal lobes, as well as the deep structures. Neglect of hemispace significantly correlated with extent of injury to the parietal lobe, but it was noted that these parietal lesions tended to be quite large. Thus, neglect of left hemispace and anosognosia only occurred consistently following more extensive lesions. Follow-up examinations revealed that the median duration to recovery for patients with anosognosia was 11 weeks and with neglect was 9 weeks. Patients with anosognosia tended to recover more quickly when the neural dysfunction resulted from hemorrhage rather than infarcts. Finally, recovery from neglect was more rapid in patients whose right frontal lobe was spared, whereas this was not a significant factor for recovery from anosognosia.

The relation between anosognosia for hemiplegia and unilateral neglect was also addressed in a recent study by Bisiach et al. (1986). They examined 97 right brain-damaged patients for unawareness of hemiplegia, unawareness of hemianopia, unilateral neglect, and several other neurological disturbances. Anosognosia was assessed on a rating scale from 0 to 3. The lowest score of 0 was given if the patient spontaneously reported or mentioned the defect following a general question about his difficulties and the highest score of 3 was given if the patient did not acknowledge the defect even after it was demonstrated to him. Intermediate scores reflected levels of awareness between these two extremes. Of particular interest in the present context was a double dissociation reported between unawareness of motor impairment and unilateral neglect. Thirty-two patients with moderate and severe anosognosia showed little or no unilateral neglect and four patients with moderate or severe neglect were not anosognosic. This finding demonstrates that anosognosia for hemiplegia is not simply a reflection of unilateral neglect. Some patients who completely ignore the affected side of the body may be fully aware of their motor defect. Others who continue to deny their hemiplegia, even when confronted with evidence to the contrary, may attend normally to the left side. Another striking finding was a double dissociation between anosognosia for hemiplegia and somato-sensory impairment and the latter was observed in two patients with no evidence of anosognosia. This result suggests that a somato-sensory disturbance is not an essential condition for the development of anosognosia.

Cappa, Sterzi, Vallar, and Bisiach (in press) have further explored anosogno-

sia and hemineglect by investigating the effects of vestibular stimulation on the two syndromes in four patients with severe unilateral neglect and anosognosia. All four patients were alert and cooperative, were inattentive to the left side of body and space, showed a left homonymous hemianopia and left hemiplegia, and denied the presence of any motor or visual defect. Remission of personal neglect (inattention to one side of the body) was found in all patients following vestibular stimulation. This finding could not be accounted for by an oculomotor explanation, that is, that a larger gaze field is induced by vestibular stimulation, since the reduction of the disorder was evident in both an eyes open and eyes closed condition. Extrapersonal neglect (inattention of one side of space) was temporarily attenuated following caloric stimulation in all four patients. Remission of anosognosia after vestibular stimulation was also observed, but only in two patients.

Critical Assessment

Several problems exist in the literature on anosognosia for hemiplegia that prevent the drawing of firm conclusions regarding the nature of the disorder. First is the frequent lack of conceptual clarity concerning anosognosia. Many authors have failed to define the disturbance or have only provided vague phrases to desribe the phenomenon. Second, many investigators have relied solely on their subjective observations of the patient to determine the presence of anosognosia; only a few investigators have developed objective methodologies for assessing the presence or degree of anosognosic disturbance (e.g., Bisiach et al., 1986; Cutting, 1978). Third, little attention has been paid to distinguishing explicitly between anosognosia and defensive denial, and no attempt has been made to develop objective methods for separating them. Fourth, observations concerning anosognosia often lack appropriate control conditions. For example, authors may attribute the anosognosia to particular lesions without studying the behavior of patients with comparable lesions who do not deny their motor defect. Autopsies were often not obtained in the early reports, and since accurate neuroimaging techniques were not available, the critical lesions in many case descriptions were inferred from clinical diagnoses, the accuracy of which are questionable. Fifth, although the relation of intellectual impairment and generalized confusion to anosognosia is an important issue, the nature and severity of these deficits were seldom assessed in a systematic fashion. Accordingly, about all that can be said presently is that such deficits are more prominent is some hemiplegic patients than in others.

Anton's Syndrome

One of the most striking forms of anosognosia is unawareness of blindness or Anton's syndrome. Patients with Anton's syndrome deny their visual defects even though their behavior clearly indicates visual difficulty. Anton (1899, cited in Redlich & Dorsey, 1945) was the first to provide a detailed description of the syndrome. He reported the case of a 56-year old women who was completely

blind yet unaware of her disability. Autopsy revealed bilateral lesions of the angular gyrus, occipital association cortex, and the splenium of the corpus callosum. Anton attributed the anosognosia phenomenon to the destruction of association fibers between the occipital lobes and other cortical areas.

Von Monakow (1885, cited in Redlich & Dorsey, 1945) had briefly mentioned unawareness of blindness in two patients several years before Anton's report. The first case was a 70-year old man with left hemiplegia and blindness. He was unaware of his blindness and often thought he was in a dark hall. Autopsy showed bilateral damage in the cuneus, the lingular gyrus, and the superior temporal gyri, as well as extensive lesions of the left occipital lobe, right thalamus and right geniculate body. The second case was a 50-year old man with epileptic seizures in whom bilateral hemianopia developed. He was totally unaware of his blindness. Autopsy showed bilateral lesions involving the lingular gyrus and the third frontal gyrus.

Redlich and Bonvicini (1907) described three patients with Anton's syndrome. The first case was a 21-year old patient with a brain tumor. In addition to his visual deficit, he was disoriented, amnesic, demented, apathetic, and euphoric. In conversation, the patient did not complain about anything, but "he could gradually be brought to realize that his 'eyes were bad', and then to admit, that he was blind. However, immediately thereafter, the patient had forgotten and claimed to be completely healthy. In a similar manner, the patient talked about his ability to move about despite the fact that he could not walk (p. 947)." Case 2, a 42-year old woman with a brain tumor, was similar to Case 1. Case 3 was a 74-year old stroke patient who admitted to seeing poorly but blamed it on circumstances such as poor lighting. This patient apparently had excellent visual imagery which was possibly confused with sense impressions or conscious perceptions, providing him with enough visual information to believe that he could see. The patient was completely disoriented and slightly demented, but the authors contended that the dementia was not substantial enough to account for the patient's behavior. In a later paper, Redlich and Bonvicini (1912) reported another patient who exhibited similar symptoms.

Bychowski (1920) presented two cases of war injury patients who had sustained a bullet wound through the back of the head and were found to be completely blind as a result. Both patients maintained that they could see everything and would not admit their blindness. One patient, a 30-year old man, was extensively observed and numerous examples of his anosognosia were recorded. The patient claimed to read the newspaper. When asked about what was in them, he always gave the same answer: "As always, about the war" (p. 354). When asked to read aloud from a newspaper or book or to give a more precise answer, the patient would say that he was not in the mood for reading, that he had a headache and would like to sleep, that the room was too dark, or that he would like a pair of good glasses (he had no need for them prior to his injury). He knew he had sustained a brain injury but seemed unaware of its consequences. When pressed, the patient would occasionally admit quietly, and

almost in matter-of-fact manner, that his vision was poor, but upon subsequent questioning he would again revert to giving excuses. Sometimes he attempted to walk alone and bumped into objects in his path but he would blame this on darkness in the room or other circumstances. No defect of intellect, memory, or attention was noted in this case.

A striking example of anosognosia for blindness and other disabilities was reported by Weber (1942). The patient had a right-sided cerebral embolism resulting in complete blindness and left hemiplegia. He could not get around and remained in bed with his eyes and head directed to the right. He was completely unaware of both his blindness and hemiplegia and he persistently denied both defects. He claimed that he could see everything and in broad daylight thought it was night. Guthrie and Grossman (1952) later reported a similar case. Raney and Nielsen (1942) described a patient who exhibited complete denial of blindness for 1 year, but then suddenly became aware of her deficit, exclaiming: "My God, I am blind! Just to think, I have lost my eyesight" (p. 151).

Redlich and Dorsey (1945) reported six cases of Anton's syndrome. They described a complex of five symptoms in their cases that was also consistent with others reported in the literature. First, the patients were unaware of their blindness, behaved as though they could see, reported visual experiences, and denied their blindness when confronted with it. Second, all patients showed at least a moderate amount of intellectual deterioration. Third, the patients were generally disoriented, had impaired memory, and tended to confabulate. Redlich and Dorsey noted that this pattern closely resembled that of Korsakoff's syndrome. Fourth, all six of Redlich and Dorsey's patients had amnestic aphasia (word-finding deficit). Fifth, the blindness was usually, but not always caused by bilateral hemianopia due to occipital or temporoparietal lesions. Redlich and Dorsey emphasized that, although all patients with Anton's syndrome showed intellectual deterioration, the deterioration alone did not provide a satisfactory explanation of anosognosia for blindness. The authors also indicated that Anton's syndrome is not a static phenomenon. Rather, there may be fluctuation in patients' perception of their blindness.

Two cases of Anton's syndrome were reported by Brockman and Von Hagen (1946). The authors' observations and conclusions were identical to those of Redlich and Dorsey (1945). Stengel and Steele (1946) reported unawareness of peripheral blindness in a case of frontal-lobe tumors. Blindness was caused by pressure atrophy of the optic nerves. The patient showed euphoria and memory defect. He was unaware of his blindness but was fully aware of other more trivial complaints. Interestingly, there was initially a difference in the degree of unawareness for the two visual fields. He denied any visual defect on the right side, where he was completely blind, even when it was demonstrated to him; however, he would occasionally admit impairment of vision on the left side, where some residual vision existed. This case was atypical in that most previously reported cases of Anton's syndrome concerned loss of vision attributable to occipital-

lobe damage. Stengel and Steele assumed that gross damage was restricted to the prefrontal areas in their patient suggesting a possible frontal role in awareness of blindness. However, they noted that the euphoria and memory defect commonly associated with unawareness of blindness are suggestive of diffuse neural dysfunction.

Sandifer (1946) described two cases of young children with cerebellar tumors, optic atrophy, and total blindness which was was not admitted. There were no clinical signs of intellectual impairment or disorientation. Sandifer ascribed the lack of insight into the blindness in these children to an "intellectual immaturity coupled with vivid visual imagery characteristic of childhood" (p. 126). He also described a case of anosognosia of total blindness that was peripherally determined. The patient was a 35-year old woman who was aware of the early symptoms of visual impairment but became unaware and denied the final state of total blindness, which coincided with mental confusion. Sandifer concluded that anosognosia of blindness depends on a certain degree of intellectual impairment which may be produced either by loss of functions or by intellectual immaturity.

A significant role for intellectual deterioration in the development of Anton's syndrome was clearly illustrated in a case reported by Hemphill and Klein (1948). This case concerned a 35-year old woman who had been blind for 14 years from bilateral optic atrophy, and whose lower limbs were immovable as a consequence of severe ataxia. Her mental state was normal until just prior to admission with tabo-paresis and she was completely aware of her condition. Following admission, mental deterioration of the Korsakoff type (e.g., confusion, memory loss) was observed, although its etiology was not specified. The patient then denied both her blindness and the motor defect. She insisted that she could see, and would describe people or objects which she believed were present. Similarly, she would tell of walks she had taken, and refused to believe there was anything wrong with her legs. This unusual case demonstrates that anosognosia is not limited to defects of recent onset. Rather, in the presence of profound intellectual deterioration, unawareness of longstanding disabilities may occur. Bergman (1957) also observed an association between unawareness of blindness and intellectual impairment in a patient who denied blindness when he was most disoriented and admitted his blindness when he was oriented. Postmortem examination revealed bilateral lesions of the occipital cortex and cortical atrophy in the parietal and frontal regions.

Nobile and Dagata (1951, cited in Bisiach et al., 1986) described four types of unawareness phenomena which may be associated with cortical blindness. The first type is demonstrated by patients who do not explicitly deny their visual defect but never mention it spontaneously and appear unconcerned about it. The second type is observed in patients who actively claim that they are not blind and attribute their inability to see to other causes (e.g., darkness in the room). The third form is seen in patients who are unaware of their blindness and lucidly describe what they apparently believe they can see. Their visual expe-

riences are frequently related to actual events in their past. Finally, the fourth type involves anosognosia for blindness accompanied by confusion and mental deterioration.

Only a few cases of Anton's syndrome have been reported in recent years. Morley and Cox (1974) reported a patient with bilateral occipital-lobe infarction who was anosognosic for his blindness. A transition from anosognosia to simultaneous visual agnosia was observed in conjunction with partial recovery of visual function. Other impairments included a severe memory deficit and disorientation. Cusumano, Fletcher, and Patel (1981) described a head-injured patient who appeared to be unaware of his blindness. When questioned about his visual defect the patient would respond that the lights had been turned off in the room or that he was still asleep. A brain scan revealed bilateral occipital-lobe infarctions. Swartz and Brust (1984) reported a case of Anton's syndrome in a blind patient during alcohol withdrawal. The patient, who was admitted to the hospital 3 days after cessation of alcohol intake, experienced visual hallucinations, some of which he recognized as unreal. During these hallucinatory periods, he believed that his vision had recovered and he fabricated descriptions of his surroundings. Despite his alcoholism, the patient had normal intellectual abilities. The hallucinations and anosognosia for blindness disappeared within 6 days. Given the absence of clouded consciousness and cortical damage, Swartz and Brust noted that anosognosia of blindness may have been a consequence of the hallucinations.

Critical Assessment

Although clinical observations have established the existence of unawareness of blindness and depict some of its major features, there is an absence of objective quantitative data. The case studies that have been reported generally do not clearly delineate the etiology of brain dysfunction and make no attempts to define operationally or assess objectively unawareness of blindness. The nature of the relation between unawareness and the various cognitive deficits observed in conjunction with it has yet to be systematically explored. Furthermore, the possible contribution of motivated denial to Anton's syndrome, and its relation to neurologically based unawareness, has yet to be addressed. Thus, even though it is clear that Anton's syndrome represents a striking form of anosognosia, little can be said regarding the nature and characteristics of the phenomenon.

Hemianopia

Anosognosia for visual defects is not limited to cases of complete blindness. Critchley (1949) documented the existence of unawareness in patients with hemianopic field defects and delineated several of its characteristics. Awareness of the field defect does not seem to depend upon the integrity of patients' mental processes and is not influenced by the rate of onset of the field defect; unawareness of hemianopia is often associated with cortical or subcortical lesions; and

different degrees of unawareness can be observed, with only one quarter of hemianopics exhibiting total lack of awareness of their visual defects. Critchley suggested that a rare phenomenon called 'optic alloaesthesia' may explain unawareness in some cases. Patients with optic alloaesthesia perceive objects presented in the intact visual field but the perception is inaccurately projected onto a corresponding point in the blind field of vision. Consequently, the patient experiences vision in the blind visual field and remains unaware of any defect. Occurrence of visual hallucinations within the hemianopic field may also contribute to the lack of awareness.

Battersby, Bender, Pollack, and Kahn (1956) examined 122 neurological patients for unilateral "spatial agnosia" (neglect) and its correlates. The neglect syndrome was significantly associated with hemianopia, unawareness of visual defect, disorientation, and severe somato-sensory and motor defects. Of particular interest for the present purposes is the tendency to deny or minimize visual impairment in the presence of unilateral neglect and the virtual absence of such symptoms in patients without neglect. However, it should be noted that two patients exhibited anosognosia for visual difficulties in the absence of any spatial neglect, thereby suggesting that the two phenomena may be dissociated. Qualitative and quantitative observations provided evidence that: (a) nondominant lesions do not always result in unilateral neglect and anosognosia, (b) lesions of the left parietal area can produce the inattention syndrome as well as unawareness of deficits, and (c) the two disorders can occur following lesions not directly involving the parietal lobe. Thus, the lesion site did not appear to be directly involved in producing the spatial deficit or the lack of awareness.

Warrington (1962) demonstrated a strong association between unawareness of hemianopic field defects and completion of visual forms. The study involved tachistoscopic presentation of figures to patients with homonymous hemianopia. The figures were presented in such a way that only half of each form fell within the intact half field of vision. Subjects were instructed to fixate on a cross in the center of a screen and report what they saw. Results on this task were evaluated with respect to patients' awareness of their visual field defect. Awareness was assessed by simply asking subjects whether they saw equally well to the left and right. Eleven of the 20 hemianopic patients were not aware of their visual defect while nine showed adequate insight into the field defect. All patients who were unaware of the deficit showed marked completion of figures; that is, they reported seeing complete forms even though it was impossible for them to have actually done so. In contrast, no patient with adequate awareness showed this tendency to complete forms. There was also a close association between the presence of parietal-lobe lesions in either hemisphere and presence of completion, suggesting that unawareness of visual disability is related to parietal-lobe disease. No significant relation was found between completion and the rate of onset or duration of illness, degree of general mental impairment, laterality of field defect, or unilateral neglect (although this final relation approached significance). Warrington attempted to link the completion effect

to other unilateral neglect phenomena and argued for its usefulness as a localizing sign of parietal-lobe disease (see Zangwill, 1963, for further discussion of these findings).

Gassel and Williams (1963) investigated further the completion phenomenon in patients with homonymous hemianopia and related their findings to degree of awareness determined from a history of the patient's visual complaints. Complete lack of awareness was present in 10 of 35 patients, all of whom demonstrated significant completion on a confrontation task. This involved reporting whether the examiner's whole face was seen while fixating on his nose. Five of the patients with absence of awareness also showed a high level of completion of figures (squares and circles). Patients with a high level of awareness showed very little completion for either faces or figures. In all cases of unawareness the hemianopia was repeatedly demonstrated to the patient at each examination, but this rarely resulted in a change of attitude toward the disability. The possibility that memory deficits contributed to patients' failure to alter their beliefs following demonstration of the defect was not discussed. Gassel and Williams described one patient who was unaware of both her right hemianopia and hemiplegia. The authors indicated, however, that other patients who lacked awareness of their visual defect had normal awareness of other physical defects. In contrast to Warrington's (1962) findings, however, the lesion site was not associated with lack of awareness or with completion. Gassel and Williams also found a strong association between awareness of the visual field defect and the patient's actual function in everyday life. Patients with no insight into the defect generally had little or no functional impairment in everyday life situations, whereas those with insight had significant disability. As noted earlier, however, cases have been reported in which the patient is constantly bumping into objects while remaining completely unaware of any visual problem.

Willanger et al. (1981b) found that most patients with field defects following right-hemisphere damage also exhibit defective awareness. Koehler, Endtz, Te Velde, and Hekster (1986) examined the CT scans of 41 patients with homonymous hemianopia to determine the relation between lesion site and awareness or unawareness of visual defect. On a confrontation task like that of Gassel and Williams (1963), patients were asked: (1) "Can you see well?" (2) "Can you see as well on your left as on your right side?" and (3) "Has your vision changed recently?" (p. 256). Patients responding positively to the first two questions and negatively to the third question were classified as unaware of their hemianopia ($n = 25$), whereas those admitting visual difficulty were considered partially ($n = 9$) or fully ($n = 7$) aware of the visual defect depending on the accuracy of their responses. Three patients whose brain scans were normal (2 unaware, 1 aware) were omitted from the analysis. Of 23 unaware hemianopic patients, 14 had parietal lesions and 9 showed no evidence of damage in the parietal region. There was no significant difference in lesion sites between the partially aware and fully aware groups and they were therefore considered as one group of patients aware of the hemianopia. In this group, only 1 of 15 patients had a

parietal lesion. Lesions in the aware patients tended to be smaller and restricted to the occipital lobe whereas lesions in the unaware patients were more extensive and more anterior involving the occipito-temporal and occipito-parietal regions. The side of hemianopia was primarily left in the unaware group. In contrast, there was no difference between left- and right-sided hemianopia in the aware patients. The left-sided predominance in unaware patients was attributed to the exclusion of aphasic patients in this study, some of whom may have been unaware of their right hemianopia.

As discussed earlier, Bisiach et al. (1986) studied anosognosia for several neurological disturbances including hemianopia and hemiplegia in 97 patients with right-brain damage. Results concerning the relation between anosognosia for hemianopia and hemiplegia showed that medium or severe anosognosia for visual field defect was far more prevalent among hemianopic patients (28/32) than the same degree of unawareness for hemiplegia among patients with severe motor impairment (12/36). A possible explanation for the higher incidence of anosognosia for hemianopia was proposed. It suggested that the visual pathways may be nearer to the neural region which, when damaged, causes disturbed monitoring of the particular dysfunction. This study also revealed that 4 of 10 patients with severe anosognosia for hemianopia had very little, if any unawareness of hemiplegia.

Critical Assessment

There have been several controlled studies of anosognosia for hemianopia, but these investigations are limited in number and are marked by several methodological problems. First, systematic assessment of anosognosia was lacking in the majority of studies. For example, Warrington (1962) evaluated its presence by asking patients whether they saw equally well to the left and right and Gassel and Williams (1963) determined degree of awareness from a history of the patients' visual complaints. Some patients diagnosed as anosognosic by these methods may have developed special strategies to compensate for their visual defect and therefore responded truthfully that they see as well to one side as the other and fail to report visual difficulties. Similarly, equating anosognosia with denial of the visual defect (e.g., Battersby et al., 1956) may be misleading since a patient could be expressing defensive denial rather than unawareness of the deficit. Second, although several authors have observed that anosognosia for hemianopia may occur in cases of right- or left-hemisphere lesions, there have been virtually no systematic investigations of lateralization of the disorder. Koehler et al. (1986) did report the side of lesion in patients with anosognosia for hemianopia; however, the exclusion of aphasic patients in their study makes it difficult to accurately assess the lateralization issue.

Amnesic Syndromes

Amnesic syndromes occur as a consequence of various types of neuropsychological impairment, including viral encephalitis, anoxia, ruptured aneurysms,

tumors, bilateral strokes, Korsakoff's syndrome, and head injuries (for review, see Cermak, 1982; Hirst, 1982; Schacter & Crovitz, 1977; Whitty & Zangwill, 1977). Lesions to medial temporal or diencephalic brain regions are necessary to produce amnesia (Squire, 1986). Afflicted patients typically have normal or near-normal intellectual, linguistic, and perceptual function, and yet are unable to remember recent events and learn many types of new information. Although a great deal of systematic research has attempted to characterize the nature of amnesic patients' memory deficit, relatively little attention has been devoted to analyzing their awareness of memory impairment. Nonetheless, a variety of clinical observations as well as a few experimental studies have been reported.

Unawareness of memory deficits has been most frequently observed in patients with alcoholic Korsakoff's syndrome. When Korsakoff (1889) initially characterized the syndrome, he noted the apparent lack of concern that patients showed toward their memory defect. For example, a patient who was surprised that he had forgotten seeing the examiner just moments before claimed that his memory had always been poor, so he was unconcerned about it. In contrast, two patients appeared to be aware of their memory impairment and were careful not to let their problem show in the presence of others. Consistent with these observations, Talland (1961) reported that patients who were examined in the acute phase of the disease were extremely confused and disoriented, exhibited confabulation, and lacked awareness of their memory deficit. He also observed that some Korsakoff patients become aware of their disturbed memory function with the passage of time. In a later and more extensive study, Talland (1965) reported that Korsakoff patients are either unaware of their disability or not fully aware of its severity. Most patients examined did not appreciate why they were in the hospital, often attributing their hospitalization to some physical problem: "None realized the full extent of his amnesic disability; some would admit to poor memory for names or dates, others denied any memory disturbance even in the face of the most striking evidence" (p. 29). Talland also reported that one chronic Korsakoff patient who had no insight into her condition responded to hypnosis but failed to demonstrate any awareness of her disability in the hypnotic state.

Zangwill (1966) viewed lack of insight as a primary feature of the Korsakoff syndrome. He claimed that: "Insight into the memory defect is either lacking or, at best, very partial, and when attention is drawn to obvious failures of memory, these are explained away by facile rationalization" (p. 105). Zangwill also noted that failure to appreciate accompanying physical disabilities may also be observed in Korsakoff patients. In addition, he considered lack of insight to be partly responsible for abnormal reasoning and judgment in Korsakoff patients. In an extensive study of the Wernicke-Korsakoff syndrome, Victor, Adams, and Collins (1971) found lack of insight into the memory defect to be evident in the majority of cases. Although patients sometimes acknowledged that their memory was impaired when directly questioned, they often had no appreciation of the severity of the amnesia and no knowledge that memory function had

changed recently: "Many patients who failed to answer a single question designed to test retentive memory refused to admit that they had a memory defect" (P. 55).

Several investigators have discussed the relation between confabulation and awareness of deficit in Korsakoff patients. It has been widely acknowledged that patients are unaware of the inappropriate and often bizarre nature of their confabulations (e.g., Talland, 1965; Victor et al., 1971; Zangwill, 1966). In an early study, Williams and Rupp (1938) reported that confabulation disappears gradually as awareness of deficit develops. Similar observations have been reported by other investigators who found that confabulation decreases as awareness of deficit increases (Mercer, Wapner, Gardner, Benson, 1977; Shapiro, Alexander, Gardner, & Mercer, 1981; Stuss, Alexander, Lieberman, & Levine, 1978; Wyke & Warrington, 1960). For example, Mercer et al. (1977) reported a severe case of amnesia and confabulation following head injury and normal-pressure hydrocephalus. Confabulation diminished after implantation of a shunt. This improvement occurred in conjunction with an increase in the patient's spontaneous verbal self-corrections and latency to respond to a variety of questions probing memory and factual knowledge, suggesting that the patient had become aware of and able to check his incorrect responses. Mercer et al. also examined 10 other amnesic patients with varying degrees of confabulation whose conditions remained relatively stable. Severe confabulators responded immediately to questions and rarely attempted to check their verbal responses, whereas mild confabulators showed significantly more correction behaviors. The authors hypothesized that the short latency to response and the lack of self-corrections exhibited by severe confabulators indicate a defective ability on the part of patients to monitor their own cognitive functioning. Victor et al. (1971) also reported that as patients' awareness of their memory defect increased, they were more likely to admit that they did not know the answers to questions rather than confabulate. It was concluded that confabulation was not employed by these patients as an intentional strategy to hide their memory disability, but was a direct consequence of their unawareness of deficit. However, Talland (1961) argued that the tendency to confabulate diminishes and may stop altogether in many patients who remain unaware of the existence of severity of memory impairment. This finding is not incompatible with the frequently reported co-occurrence of confabulation and lack of awareness, but it does suggest that the presence of confabulation is not a necessary condition of impaired awareness. However, the available evidence is consistent with the idea that unawareness of deficit is a necessary condition of confabulation.

Jarho (1973) described a Korsakoff-like amnesic syndrome in six patients with penetrating brain injuries. Three patients lacked awareness of their memory impairment. Of these three, one patient had sustained damage to both frontal lobes with a large splinter extending through to the left occipital region. A second patient had splinters through the left frontal region directed towards the hypothalamic region, and a third suffered lesions to the right parietal region

and the left orbital and temporal regions. By contrast, three other patients who exhibited good insight into their memory dysfunction had no signs of frontal-lobe damage.

Jarho's observations suggest a possible relation between frontal-lobe damage and unawareness of memory deficit. The fact that lack of awareness has been observed frequently in alcoholic Korsakoff patients is consistent with this possibility, because Korsakoff patients are often characterized by signs of frontal-lobe pathology (cf. Moscovitch, 1982; Schacter, 1987; Squire, 1982). An experimental study by Shimamura and Squire (1986) provides further pertinent evidence. They examined Korsakoff patients' ability to monitor their own memory function by investigating feeling-of-knowing judgments - predictions regarding whether or not one feels one could recognize information that cannot be recalled to a specific cue. The accuracy of Korsakoff patients' feeling-of-knowing judgments was impaired relative to that of control subjects, both for questions regarding preexperimentally acquired facts (Experiment 1) and for questions regarding newly learned information (Experiment 2). These findings suggest that Korsakoff patients have difficulty monitoring their memory performance. Shimamura and Squire contended that the monitoring deficit was produced by cognitive impairments specific to Korsakoff's syndrome, perhaps related to frontal-lobe pathology. Significantly, a variety of other amnesic patients who were free of cognitive impairments associated with frontal-lobe pathology showed normal memory monitoring on the feeling-of-knowing task.

A variety of other observations also point to a relation between frontal-lobe damage and unawareness of memory deficits. Luria (1976) described a patient with removal of a massive left frontal tumor who expressed no awareness of his memory deficits, although he did complain of headache and blindness. A second amnesic patient with bilateral frontal damage following head trauma insisted that he was not ill and did not know why he was in bed. When Luria examined him 40 days postinjury, he still asserted that he was not ill and could only conjecture that he was in the hospital because of a toothache.

Patients in whom amnesia is attributable to rupture of an anterior communicating artery aneurysm (ACAA) may also exhibit little awareness of their amnesic disturbance. Frontal-lobe disturbances are frequently, though not always, observed in ACAA patients (Alexander & Freedman, 1984; Vilkki, 1985; Volpe &Hirst, 1983). For example, Luria (1976) described two ACAA amnesic patients who denied any memory defect. A strong association between frontal damage and lack of awareness was revealed in a study by Vilkki (1985). Of five ACAA patients with severe memory disorders, three who were unaware of their memory deficits had damage in the frontal region. Vilkki reported that no frontal lesions were found in the two patients who were fully aware of their memory problems. Alexander and Freedman (1984) studied 11 patients with amnesia after ACAA rupture. They observed a general pattern of recovery following surgery that included an early stage characterized by confabulation and unawareness of deficit. In a later stage of recovery, confabulation and

unawareness decreased substantially, but patients remained unconcerned about their memory problems. Alexander and Freedman noted that unawareness was observed in four patients who did not have CT evidence of frontal damage, although the patients did exhibit other behavioral characteristics of frontal pathology, such as confabulation and confusion.

Schacter, Glisky, and Mc Glynn (in press) studied a severely amnesic ACAA patient who was unaware of his memory defect. For approximately 1 1/2 years following his ruptured aneurysm, this patient denied his gross memory impairment. He would admit a "slight" memory problem upon direct questioning but insisted that he could return to his former management level position and successfully function with the aid of some compensatory strategies such as note taking. We developed several self-report questionnaires to assess awareness more formally. The questionnaires consisted of some general questions about memory function (e.g., Compared to before your illness, how much difficulty are you currently having with your memory?) and some specific questions that required the patient to judge how likely he would be to remember various kinds of information in particular work and everyday situations. The patient's wife was given the same questionnaires and asked to rate her husband. In addition, the patient was asked to rate his wife's memory on the various questionnaires, and the wife was asked to rate her own memory function. Relative to his performance on objective memory tests and his wife's ratings, the patient consistently and substantially underrated the severity of his memory impairment and overrated the likelihood that he would remember successfully in specific situations. However, the patient's assessments of his wife's memory abilities were quite accurate, indicating that his lack of awareness was not attributable to a general impairment of judgment.

A second phase of this study involved an attempt to increase the patient's awareness of his deficit. This was accomplished by giving the patient lists of words and actions to remember, requiring him to predict his own recall performance, and then providing extensive feedback and discussion concerning the discrepancies between prediction and performance. As expected, the patient initially overpredicted his performance on the recall tests. With extensive training, however, his predictions became more realistic and his responses on many questionnaire items reflected increased awareness of memory problems. These findings suggest that, with sufficient repetition, an amnesic patient may be able to develop awareness of his current state of memory function, but it is not yet clear whether the awareness training will have any long-lasting effects. It should also be noted that, although the patient became more realistic when rating himself on general questions about his memory, he generally did not apply this knowledge to specific situations, especially those concerned with the work setting.

Although the foregoing studies indicate that unawareness of deficits has been observed in many ACAA patients, some observations of preserved insight have been reported. Luria (1976) described an ACAA patient with excellent aware-

ness of his memory disorder. Volpe and Hirst (1983) discussed two ACAA amnesics who exhibited awareness of their condition 10 days postsurgery, although they were apparently unconcerned about the consequences of their injury. However, it was unclear whether the patients were aware of their disability during the first 10 days of their illness. Moreover, their reported "unconcern" may indicate some degree of impaired awareness.

Studies of patients with severe head injuries, who frequently sustain significant frontal-lobe damage (Levin, Benton, & Grossman, 1982), have also provided evidence for unawareness of memory deficits. Sunderland, Harris, and Baddeley (1983, 1984) examined the relation between patients' subjective assessments of their own memory function on self-rating questionnaires and their performance on objective memory tests. Results demonstrated a large discrepancy between patients' subjective measures and objective memory tests. Head-injured patients did not rate themselves as different from controls on the questionnaire even though the patients showed significant memory deficits compared to controls on the objective tests. However, relatives' ratings of patients' memory problems were positively correlated with objective measures, that is, were more realistic than patients' ratings of themselves.

More recently, Boake, Freeland, Ringholz, Nance, and Edwards (1987) studied 34 severely head-injured patients and confirmed the lack of association found by Sunderland et al. (1983, 1984) between patients' self-ratings of memory impairment and their performance on objective measures. A relation was found between self-ratings of memory impairment and emotional distress (as measured by anxiety and depression scales), with more distressed patients tending to report greater memory difficulties. Boake et al. also examined the relation between lesion site and memory self-ratings. Patients with left-hemisphere focal lesions were more likely to report memory problems than those with right-hemisphere lesions.

Cockburn, Wilson, and Baddeley (1986) investigated how well people with memory problems attributable to head injury and other forms of traumatic brain insult recognize their difficulties. They divided patients into poor, moderate, and good memory groups according to their performance on an objective test of everyday memory (Wilson, Cockburn, & Baddeley, 1985). All patients were assessed with objective measures of memory and were asked to rate themselves on a memory rating scale for 10 everyday functions. A close relative was also given the memory rating scale to assess the patient. No correlation was found between the self- and relative ratings for the poor memory group, whereas significant correlations were evident for the other two groups. The discrepancy between patients and relatives of the poor memory group was considered indicative of some lack of awareness on the part of patients with respect to their memory function. Overall, the ratings of the poor and moderate memory groups indicated that most of the patients realized they were having more difficulty with their memory in everyday situations than they had before their illness or accident, but were not fully aware of the severity of the memory defect. This

view was supported by the low correlations between subjective ratings of memory abilities and objective tests in the moderate and poor groups. Neurological factors such as side of lesion or global versus focal damage did not appear to have any consistent relation with degree of awareness in this group of patients.

Rimel, Giordani, Barth, Boll, and Jane (1981) studied 429 patients with mild head trauma 3 months after injury. More than half the patients (59%) reported a change in their memory since the accident but "significant others" (e.g., relatives, close friends) indicated a more serious problem with the patients' memory than the patients recognized or were willing to admit. Therefore, even in cases of mild head injury, some degree of unawareness of a memory deficit may exist.

In marked contrast to other amnesic syndromes that are characterized by unawareness of memory deficit, patients with so-called "pure" amnesias, in which there is neither behavioral nor neurological evidence of frontal-lobe involvement, appear to be aware of their memory defects. The well-known amnesic patient H.M., who developed amnesia after bilateral resection of temporal lobes and hippocampus, is reported to be aware of the existence of his memory deficit (Milner, Corkin, & Teuber, 1968), although no formal assessments of the extent of H.M.'s awareness have been reported. Rose and Symonds (1960) studied four encephalitic patients with disabling memory impairments and found that they had acute awareness of their defects. For example, when questioned about his condition, one patient replied: "There's nothing wrong with me physically but mentally things as they happen don't seem to impress themselves on my mind" (p. 195). Another patient remarked: "It appears to me that my memory is distant and I do not seem to be able to know anything very recent" (P. 200). The encephalitic patients in this study not only demonstrated awareness of their defects, but showed little intellectual impairment and did not confabulate. The authors postulated that restricted temporal-lobe lesions were responsible for amnesia in their four encephalitic patients and this proposition was supported by the occurrence of temporal-lobe seizures in three of the four cases.

Zangwill (1966) also reported that in cases of restricted temporal-lobe damage, a gross memory defect like that of Korsakoff's syndrome may exist without the more florid manifestations of disorientation, confabulation, disorders of judgment, or unawareness of disability. For example, a young soldier who exhibited severe amnesia following a meningitis attack accurately described his memory problem as "desperate" (p. 107), and did not confabulate. Luria (1976) observed excellent awareness of memory deficit in patients with amnesia attributable to pituitary tumor or lesions of the third ventricle. Luria noted that these patients often make remarks such as: "my memory is not as good as it was," or "I forget everything," "things will not stick in my mind," or "I have to write everything down" (p. 342). Two amnesic patients with restricted small tumors in the third ventricle were also reported to have retained an intact "critical attitude" towards their memory impairment. The well-known case of

N.A. (Kaushall, Zetin, & Squire, 1981) provides another example of amnesia, unaccompanied by disorientation, confabulation, or anosognosia. N.A. is amnesic for verbal material because of a lesion in the left dorsomedial nucleus of the thalamus (Squire, 1982). N.A. has clear knowledge of his memory defect. He describes his problem as "not knowing whether I will remember something when I need to remember it" (p. 385). In contrast to Korsakoff patients, N.A shows a normal ability to predict recognition of unrecalled items in the feeling-of-knowing paradigm of Shimamura and Squire (1986).

Studies that have used questionnaire methods to assess subjective memory impairment also suggest that amnesic patients without frontal-lobe damage are aware of their deficits. Bennett-Levy, Polkey, and Powell (1980) administered a self-report questionnaire to a group of patients with memory problems attributable to temporal lobectomy. They found that these patients provided reasonably accurate assessments of their memory function. As part of their training study, Schacter et al. (in press) administered several subjective memory questionnaires to a severely amnesic encephalitic patient who had no signs of frontal-lobe involvement. In contrast to the lack of awareness demonstrated by the ACAA patient on these questionnaires, the encephalitic patient rated herself in a highly realistic manner. In fact, this patient's ratings of her own memory performance were virtually identical to the assessments made by her husband.

Intact awareness has also been reported in the syndrome of transient global amnesia (TGA). This temporary memory loss occurs suddenly and is severe. It is generally attributed to ischemia restricted to the medial temporal lobe region (Jensen & Olivarius, 1981; Pansford & Donnan 1980). Patients experiencing TGA are aware of and often upset by their deficit. For example, Evans (1966) discussed a case of TGA in a 60-year old man who got dressed after a bath, but "looked puzzled and upset. His first words were 'Am I going mad? I can't remember anything' (p. 543). Similarly, Roman-Campor, Poser, and Wood (1980) described a TGA patient who was clearly aware of and concerned about her memory impairment: "The clinical presentation of the memory defect in this patient is characteristic of TGA: an older woman, previously healthy, suddenly confused and repeatedly asks the same questions, realizing that she has a memory defect" (p. 514). Others have reported similar observations (Byer & Crowley, 1980; Fisher, 1982; Haas & Ross, 1986; Landi, Giusti, & Guidotti, 1982; Regard & Landis, 1984).

Critical Assessment

Several studies reviewed in this section have provided quantitative methods for assessing unawareness that go beyond simple statements that patients are "aware" or "unaware" of their deficits. However, most of the quantitative studies have been concerned with a single type of amnesic population, such as head-injured patients or temporal-lobectomy patients, with each group of investigators using a different method for assessing patients' awareness of their deficit. More extensive investigations that compare awareness of memory dis-

turbance in different amnesic poulations (e.g., ACAA, Korsakoff's, encephalitic) using identical assessment techniques for all groups would be extremely useful. The fact that unawareness of memory deficit appears to occur largely in patients with frontal-lobe signs implies that unawareness of memory impairment cannot be attributable solely to patients' forgetting that they have a memory impairment. Since some densely amnesic patients are aware of their impairment, something more than the memory deficit itself would appear to be necessary for unawareness. Memory disorder may contribute to *sustaining* unawareness in those patients who initially exhibit the phenomenon, but the idea that unawareness of amnesia is a function of a patient's "inability to remember that he cannot remember" (Whitlock, 1981, p. 213) can be rejected.

Head Injury

Brain damage resulting from severe head injury may lead to a variety of physical, cognitive, and behavioral deficits (Brooks, 1984, Levin et al., 1982). As discussed in the previous section, unawareness of memory impairment is frequently observed in head-injured patients. In addition, unawareness of personality changes and abnormal behavior, typically associated with frontal-lobe damage (Bond, 1984), are commonly observed sequelae of severe head injury. This section considers the literature concerned with unawareness of personality change and behavioral deficits in head-injured patients.

An early clinical study of 35 cases of severe head injuries was conducted by Schilder (1934), who focussed primarily on the acute posttraumatic phases. Schilder stated that patients were frequently unconcerned about their injuries and unaware of their deficits, although he did not provide any quantitative estimates of the incidence of unawareness. Miller and Stern (1965) commented on the marked absence of patient complaints following severe head injury. Some such patients are euphoric and many minimize their disability. In contrast, patients suffering from the so-called "post-concussional syndrome" after a mild injury frequently complain of their symptoms.

Ota (1969) examined personality changes in a population of head-injured patients. Of the 80 patients examined, 34 were unaware of their psychological disturbances and did not spontaneously complain of physical disabilities. Of the 46 patients considered to be somewhat aware of their deficits, 29 complained of minor physical problems and only 17 commented vaguely of mental difficulties. Ford (1976), in his clinical observations of a large selection of head-injured patients, included "lack of insight" as a primary change resulting from the brain damage. He observed that the patient is usually unaware of his intellectual impairment and will deny any change: "At first he will identify with his own premorbid self-image, and only after many destructive failures comes to see he is not the man he was" (p. 603).

A unique case of unawareness was reported by a head-injured patient who was himself a practicing physician prior to his automobile accident (Labaw, 1969). Shortly after regaining consciousness he insisted that he could soon

return home and he began to make implausible plans for the future. When playing chess with friends he would not acknowledge his errors even when they were pointed out to him. He confabulated about the cause of his illness while in the hospital and exhibited no anxiety about his condition. He repeatedly insisted to his wife that he could return to work, which reflected a totally inaccurate assessment of his condition. His most obvious expressions of unawareness were, surprisingly, in the presence of other medical professionals. For example, when writing of his injury 1 year posttrauma for publication, he referred to his medical condition as "cerebral concussion," which was a striking minimization of his actual state. Labaw indicated that his anosognosia persisted for over 2 years, gradually disappearing to the point where he could recognize his earlier expressions of denial and attempt to understand their presence.

Consistent with Labaw's account, Prigatano (1986) noted that many severely head-injured patients will continue to minimize the severity of residual neuropsychological deficits for several years following trauma. Evidence on this point was provided in a study by Groswasser, Wendelson, Stern, Schechter and Najenson (1977), who found that all patients who exhibited unawareness of behavioral disturbances when evaluated 6 months postinjury continued to do so at a 30-month followup evaluation.

Tyerman and Humphrey (1984) found that severely head-injured patients often lack full insight into their condition, but also exhibit some awareness. They examined self-concept in 25 severely head-injured patients approximately 7 months following injury. Possible changes in self-concept were assessed with scales of anxiety and depression, an attitude questionnaire relating to physical disability, and semantic differential ratings of "Present Self," "Past Self," "Future Self," a "Typical Person," and a "Typical Head-Injured Person." Patients were generally distressed and reported numerous changes in themselves compared to before their injury. However, they expected to recover to their premorbid selves within a year, which was perceived as more positive than a Typical Person. While they viewed themselves as strikingly different from the Past Self, patients rated themselves more positively in some respects than their concept of a Typical Head-Injured Person, which was considered to be dramatically different from that of a Typical Person. In fact, their ratings of Present Self did not differ significantly in most domains from ratings of a Typical Person. These results clearly show that, although patients are aware of some degree of change resulting from head injury, they "cling to hopes of returning to their former, perhaps somewhat glorified, past" (P. 20) This anticipation is clearly unrealistic, because most patients continue to suffer from some degree of impairment.

Newton and Johnson (1985) suggested that lack of awareness may influence the degree of social anxiety experienced by head-injured persons. Eleven severely head-injured patients were assessed with observational and self-report measures, the latter consisting of a Questionnaire of Social and Evaluative Anxiety. Three of the 11 patients reported low social anxiety, and 2 of the 11

indicated high self-esteem, whereas the majority of head-injured patients (72%) reported high social anxiety and low self-esteem. Newton and Johnson noted that the anomalous scores of low social anxiety and high self-esteem may have been manifestations of unawareness in these head-injured patients.

Several studies have examined unawareness of social and behavioral changes in head-injured patients by examining the relation between patients' and relatives' reports. Fahy, Irving, and Millac (1967) investigated the late effects of severe head injury in a 6 year follow-up study. Interviews with 32 patients and their relatives revealed discrepancies between their respective reports of the patient's disability. Patients exhibited some awareness of their intellectual, memory, and speech deficits; however, they rarely acknowledged "temperamental" changes which were reported by relatives. This finding suggests some degree of unawareness on the part of patients with respect to their disturbed behavior. In a study of 50 severely head-injured patients and their families, Thomsen (1974) stated that patients rarely reported posttraumatic disabilities whereas close relatives of 42 patients reported changes in personality of the patient. During clinical interviews, some patients would mention their poor memory but most appeared unaware of any change in their behavior.

Cognitive deficits that frequently follow head injury, such as memory impairment, poor attention span, and defective judgment, may contribute to the observed discrepancies between patients' and relatives' reports by rendering patients incapable of accurately reporting their difficulties. McKinlay and Brooks (1984) investigated this issue. They asked 55 patients and their relatives to assess patients' degree of impairment on 18 items. Results at 6 months posttrauma indicated that, in most cases, disagreement between the patient and relative was restricted to between 0 and 3 of the 18 items. The largest discrepancies occurred in the domain of emotional/behavioral disturbances such as bad temper and anxiety. On those items, relatives reported the problem more frequently than did patients. In contrast, there was close agreement on items related to sensory-motor impairment, memory, and concentration. Correlational analyses revealed no consistent relation between the observed patient-relative discrepancies and patients' performance on several psychometric tests (verbal and nonverbal intelligence, verbal and visual recall, and verbal fluency and comprehension). McKinlay and Brooks concluded that the observed unawareness appears to be unrelated to cognitive deficits as measured by the various psychometric tests used in this study. Furthermore, they suggested that the disagreement between patients' and relatives' reports may be partly attributable to distorted perceptions on the part of relatives rather than patients.

Lezak (1978) noted that only with the passage of time do family members come to appreciate that the patient is not the same person he was prior to injury. Consistent with this idea, Romano (1974) reported that relatives of 13 severely head-injured patients denied various aspects of patients' disabilities. Most of these family members exhibited explicit verbal denial of the fact that patients were different than before their injuries. Some families acknowledged disability

in one domain, usually physical, but continued to deny cognitive and behavioral deficits. Romano noted that this failure of the family to recognize and accept the patient's actual status frequently leads to some form of denial by the patient. The denial of patients' condition and prognosis by these families was regarded as a coping strategy when faced with the unacceptable and tragic reality. Similarly, Bond (1984) commented that the pattern of fluctuation between periods of insight and unawareness often observed in the head-injured patient may also be evident in relatives, reflecting a "mental defence mechanism" employed to eliminate anxiety associated with the patient's actual condition. In a study of relatives' reports of severely head-injured patients, McKinlay, Brooks, Bond, Martinage, and Marshall (1981) indicated that, although relatives reported a variety of symptoms, especially memory disturbances, at 3, 6, and 12 months after injury, emotional problems were more often reported at later follow-ups. It was suggested that these emotional symptoms may not be observed or admitted by the relatives for some time.

Many investigators have commented on the problems posed by unawareness of deficits in head-injured patients for rehabilitation. Unaware patients may lack motivation for treatment (Prigatano & Fordyce, 1986a, b), fail to implement compensating strategies (Brooks & Lincoln, 1984; Cicerone & Tupper, 1986), maintain unrealistic goals for rehabilitation (Ben-Yishay et al., 1985; Diller & Weinberg, 1981), and fail to benefit from therapy (Ford, 1976). Several studies have examined problems of unawareness in the rehabilitation setting.

Hackler and Tobis (1983) reported on anosognosia in young head-injured adults who were participants of a prevocational training program. The participants often fell and hurt themselves as a consequence of their poor balance, spasticity, and lack of coordination. However, when shown the contusions and bruises on their bodies, many would appear completely bewildered by them. Head-injured persons were described as being unaware of their physical and cognitive problems or of the implications of their deficits. For example, when questioned about falls observed by staff members, many participants acknowledged that they fell but did not know why; they apparently did not realize that they had a profound balance problem. In the social realm, they could not understand the effects that their rage or other socially inappropriate behaviors have on people around them because they were incapable of judging when they were behaving in an unacceptable manner. Patients' inability to monitor their own behavior and their failure to remember what they had done were considered by families to be the most common cause of social isolation in the post-trauma years.

Prigatano and Fordyce (1986b) investigated unawareness of deficits in 23 patients who underwent rehabilitation following traumatic brain injury. Patients, relatives, and rehabilitation staff members completed a Patient Competency Rating Scale (PCRS) (Roueche & Fordyce, 1983). This involved rating the patient's ability to perform a variety of everyday activities on a 30-item, 5-point rating scale. In addition, patients were assessed on standard measures of

neuropsychological and emotional functioning. Patients tended to rate themselves as more competent than did family or staff members. Discrepancies between patients and staff members were positively correlated with patients' degree of neuropsychological impairment and negatively correlated with the extent of emotional distress reported on the MMPI. These findings suggest that patients who were unrealistic in appraising their competency relative to staff members tended to have more severe neuropsychological impairment and experienced less emotional distress (see also Prigatano, 1985).

In a study by Fordyce and Roueche (1986), 28 seriously brain-damaged patients, most of whom had suffered traumatic head injuries, participated in a rehabilitation program that attempted to increase patients' awareness of their deficits. Awareness training included strategies such as educating patients on the consequences of brain injury, videotaping patients and providing feedback regarding their behavior, asking patients to generate a list of their individual problems for discussion with other program members, and consistently reinforcing behaviors reflecting patients' increased acceptance of their deficits (Prigatano & Fordyce, 1986a, b). To assess any change in patients' awareness of deficits, patients, staff members, and relatives were asked to judge the patient's competency on the PCRS before and after rehabilitation. Standard measures of patients' neuropsychological, emotional, and psychological functioning pre- and postrehabilitation were also recorded. Three groups of patients were identified on retrospective analysis based on staff-patient differences in perceived impairment, as assessed by the PCRS. Group 1 consisted of 11 patients whose ratings of their abilities were similar to staff members' ratings both before and after rehabilitation. Patients in Group 2 ($n = 9$) and Group 3 ($n = 8$) underestimated their initial level of impairment. By the end of rehabilitation, Group 2 patients had lowered their competency ratings and were, therefore, more consistent with the ratings of staff members. In contrast, an increased discrepancy was observed between ratings of group 3 patients and staff at the end of rehabilitation. These patients rated significantly more improvement in their abilities than did staff, and also showed increased emotional distress on the MMPI. This finding could be interpreted as a defensive reaction to rehabilitation methods. However, the authors noted that patients may have had an organically based awareness disturbance, the manifestations of which were identical to defensive denial. Relatives' ratings before rehabilitation were in the middle range, between patients and staff members, and became closer to staff members' ratings by the end of rehabilitation. Fordyce and Roueche concluded that only some head-injured patients benefit from rehabilitation attempts to increase awareness of deficits.

Ranseen and Bohaska (1987) extended Fordyce and Roueche's (1986) findings by examining the relation between staff-patient rating discrepancies on the PCRS and lesion site following traumatic brain injury. Awareness of disability was studied in 32 patients with focal left, focal right, or diffuse damage before rehabilitation and 1 month into the program. All three groups rated themselves

as significantly more competent than did rehabilitation staff members at both intervals. Although patients' abilities showed improvement after 1 month of rehabilitation, they continued to overestimate their competency. The staff-patient rating discrepancy was significantly greater at both intervals for the group with right-sided damage than for the groups with left-sided or diffuse damage, suggesting that degree of disturbed awareness is, at least partly, a function of lesion site. Ranseen and Bohaska postulated that right brain-damaged patients have a greater awareness disturbance than others because of "the specific nature of their cognitive impairment" which involves perceptual problems and difficulty organizing information in relation to one's self.

Critical Assessment

The development of questionnaires to assess patients' views of their own condition as well as relatives' and staff members' perspectives of the patients' status represents an important methodological development. The discrepancy between patients' and "others" ratings provides a possible measure of degree of patients' unawareness. However, there are problems with using this method as the sole measure of unawareness. First, since family members may engage in some motivated denial, those researchers only considering discrepancies between patients' and relatives' ratings may find less unawareness in patients than is actually present. Some investigators have attempted to overcome this problem by asking rehabilitation staff members (who are presumably more objective than family members) to rate the patient as well (e.g., Fordyce & Roueche, 1986; Prigatano & Fordyce, 1986b). Second, since both psychogenic and neurogenic mechanisms may be contributing to the phenomenon, it is difficult to determine on the basis of questionnaire responses what proportion of the discrepancy between patients and relatives/staff is attributable to a motivated defensive reaction in patients and how much is caused by a neurologically based awareness disorder. This difference may be crucial for understanding why some head-injured patients benefit from awareness training whereas others do not. As Prigatano and Fordyce (1986b) indicated, those patients who fail to benefit may have an organic awareness disturbance which prevents them from acquiring new knowledge about themselves whereas those who succeed in awareness training may have only been expressing defensive denial.

Dementia

Alzheimer's disease (AD) is the most common type of dementia occurring in adult life and has attracted widespread attention in recent years. Lack of awareness of the cognitive deficits associated with this form of dementia has frequently been reported in clinical descriptions of the later stages of the disease. Schneck, Reisberg, and Ferris (1982) characterized the clinical syndrome of AD with three major phases. During the initial "forgetfulness phase," patients notice and become increasingly anxious about their memory problems. As the disease progresses, patients enter the second "confusional phase," at which time

they show clear evidence of impaired cognitive functioning, especially for memory of recent events. Patients at this stage lose insight into their own deficits and earlier anxiety is replaced with unawareness of illness. In the final "dementia phase," the patient becomes extremely disoriented and may exhibit considerable anxiety despite the continuing unawareness. Thus, the general pattern is one of decreasing insight and knowledge with increasing severity of the disease process.

Reisberg, Gordon, McCarthy, and Ferris (1985) conducted a study exploring awareness of deficit in 25 AD patients, 5 subjects with senescent forgetfulness (progressive cognitive decline in normal aging), and 10 control subjects with no memory impairment. Subjects were interviewed and questioned about their own functioning as well as their spouses' functioning. Spouses of subjects were similarly interviewed and questioned about their own functioning and the subject's functioning. Results indicated that subjects with senescent forgetfulness rated their memory problems as somewhat worse than did the controls, and "Early Confusional Phase" AD patients rated their problems as being substantially worse than did the "Forgetfulness Phase" patients. In contrast, following the "Early Confusional Phase" AD patients tended to rate the degree of their memory impairment as progressively less severe, whereas objective measures of memory showed evidence to the contrary, that is, progressive deterioration of memory function. Spouses' reports of patients' memory problems increased consistently as the patients's deficit increased on objective measures. Patients with moderate to severe memory impairment also tended to minimize their emotional difficulties. They rated the extent of their emotional problems as considerably less than did their spouses. The patients did retain insight into two domains unrelated to their own cognitive function: (a) throughout the illness they showed insight into their ability to communicate with the spouse and (b) despite marked unawareness of their own deficits in the final phase, patients continued to display insight into their spouses' cognitive functioning. This latter observation led Reisberg et al. to conclude that defensive denial was operating in AD patients to produce an apparent "lack of insight" with respect to their own deficits.

Other clinical observations of AD have emphasized an early loss of insight as opposed to the late stage symptom described above (Frederiks, 1985b; Joynt & Shoulson, 1985; Mahendra, 1984). Frederiks (1985b) indicated that the patient is generally unaware of the gradual onset of dementia associated with both AD and Pick's disease (PD). This lack of awareness was referred to as "anosognosia for dementia." Similarly, Mahendra (1984) commented on the early loss of insight in both AD and PD. However, Gustafson and Nilsson (1982) reported that early loss of insight is a useful dimension for differential diagnosis of AD and PD. They developed rating scales to identify AD and PD that assessed a number of clinical features. Patients with PD were rated considerably higher than AD patients with respect to early loss of insight on these scales. Thus, there may be a different progression for loss of insight in PD and AD patients. It is

interesting to note that both of these forms of dementia are typically associated with signs of frontal-lobe pathology (e.g., Kaszniak, 1986; Mahendra, 1984), but frontal degeneration is typically more severe in the early stages of PD than AD. Mahendra also noted that, in vascular dementia, insight is relatively well preserved.

There have been few attempts to experimentally investigate unawareness of deficits in dementing patients. Evidence of unawareness of memory dysfunction in AD patients was provided by Schacter, McLachlan, Moscovitch, and Tulving (1986). Alzheimer's patients were given a categorized list and were asked to predict how many items they would be able to recall. Relative to control subjects, AD patients grossly overpredicted their memory performance. Neary et al. (1986) found that different subgroups of AD patients exhibited different levels of insight. One group of patients showed obvious signs of anxiety when required to perform difficult tasks, indicating some degree of awareness of their problems. Others tended to minimize their difficulties but retained some awareness of their disability. Some patients would admit to memory impairment but appeared not to appreciate the severity of the disorder, and exhibited no overt anxiety. These findings suggest that there may be considerable variability in awareness among dementia patients.

Critical Assessment

There is little direct evidence concerning unawareness of deficits in dementing patients. Reisberg et al. (1985) argued that the discrepancy they observed between AD patients' assessment of their own and others' cognitive functioning indicates that patients are engaging in defensive denial. However, such a self-other discrepancy need not imply that denial is motivated. It may simply reflect the fact that patients are basing their judgments on past information concerning themselves and their spouse. Since the spouse's condition has presumably not changed substantially, patients' judgments of them will be accurate. But since their own state has changed, patients's inability to assimilate and monitor new information about themselves will result in defective insight.

Aphasia

Aphasia is a disorder of language production and/or comprehension that is reflected by impairments in processing both semantic information (Wernicke's aphasia) and syntactic information (Broca's aphasia). Aphasic deficits are generally attributable to damage in particular regions of the left hemisphere and may be accompanied by specific forms of abnormal speech such as jargon, stereotypy, or echolalia (e.g., Benson, 1985). A striking feature of these disturbed forms of expression is unawareness of the disordered speech (Lebrun, 1987).

Alajouanine (1956), in his clinical observations of jargon in Wernicke's aphasics, defined jargon as speech that is devoid of any meaning. Jargon aphasics are unaware of the fact that their speech is incomprehensible. They consider their speech to be completely normal, and therefore make no attempt to ascertain whether they are being understood. Alajouanine commented on the

unawareness of the jargon aphasic in the following description:

> It is amazing to see such a patient uttering in a confident and natural way utterly meaningless words or extraordinary sentences. For instance, one of our patients called on by a neighbour who wore splendid new shoes, told her admiringly: 'Oh, what beautiful chemists you have.' This interchange of words which, of course, surprised the neighbour, was the beginning of a paraphasic jargon and the first symptom of a left temporal tumour (p. 23).

The jargon aphasic believes he is communicating satisfactorily and, consequently, does not show halts, hesitations and corrections often seen in other aphasic patients. Brown (1977) noted that jargon aphasia may gradually progress from a stage marked by completely meaningless speech, anosognosia, and euphoria to one of intact word meaning, acute awareness, and frustration.

Interestingly, Alajouanine (1956) observed that patients who exhibit unawareness of their jargon will criticize the physician when he uses expressions from their own meaningless speech. This finding suggests that some degree of speech comprehension is spared in these individuals that enables them to notice errors in others' speech, even though they do not recognize their own disturbed speech. Kinsbourne and Warrington (1963) described two cases of jargon aphasia and anosognosia in patients with preserved intellectual functions following left-hemisphere stroke. The first patient had a right hemiplegia, jargon aphasia, blindness in the left eye, and a right hemianopic field defect. Although the patient was well aware of his hemiplegia and blindness in the left eye, he seemed unaware of his right hemianopia and completely denied his speech deficit. When questioned about his speech the patient maintained that it was "very very good" (p. 29). When a tape recording of the patient's own speech was played back to him, he was content that it was understandable and in good English. Similarly, when he read what he had written, the patient believed it was comprehensible. However, when his own words were played back to him in another's voice and when his own writing was given to him in another's handwriting, he stated that it was incomprehensible and in bad English. The second jargon patient was also unaware of his speech disorder. When a tape recording of his voice was played back and he was asked whether the English was properly used, he responded: "Yes, I would say it was perfectly O.K." (P. 34). He was similarly satisfied with the correctness of his own writing but rejected the adequacy of the same passage in the examiner's writing.

A second language disturbance, verbal stereotypy, is an extremely limited form of verbal expression associated with Broca's aphasia (Alajouanine & Lhermitte, 1964; Brown, 1977). The aphasic patient with this disturbance utters only stereotyped expressions in a completely automatic fashion. An important aspect of verbal stereotypy is the patient's unawareness of the stereotypic content. Alajouanine (1956) noted that: "They are no more conscious of their pattern of speech than are normal subjects who repeatedly interject some ready-

made expressions annoying to their audience, but without noticing it themselves" (p. 6). In contrast, the patient is acutely aware of his difficulty during volitional speech and often expresses frustration with his language production deficit. Verbal stereotypy may gradually disappear as patients become more aware of their abnormal utterances, regain use of volitional speech, and attempt to check their language production. Alajouanine (1956) observed that verbal stereotypy may evolve into another form of disturbed verbal expression called agrammatism. Agrammatism refers to a breakdown of grammatical organization in speech. The patient demonstrating this disturbance appears to have little awareness of his defect. Thus, having been completely unaware of his difficulty at the verbal stereotypy phase, the patient is subsequently unaware of his grammatical impairment.

A form of disturbed verbal expression often observed in patients with focal posterior lesions or diffuse damage is echolalia. This disturbance refers to the meaningless repetition of speech and, similar to stereotypy in aphasia, echolalia is a "brief-latency, well-articulated, often explosive" (Brown, 1977, p. 146) utterance of which patients are unaware. Brown described a continuum of awareness along which patients with echolalia may progress. As awareness of the echoed speech increases, patients make more effort to correct themselves, and may experience increased frustration with full awareness. Brown (1975) reported this transition in a case of conduction aphasia attributable to a tumor in the left parietal region.

Several investigators have attempted to systematically study the relation between anosognosia and aphasia in neurological patients. Weinstein, Cole, Mitchell, and Lyerly (1964) examined 28 patients with right-sided sensorimotor deficits (e.g., hemiparesis, hemiplegia, hemianopia) attributable to various etiologies affecting the left hemisphere. Results showed that most patients with striking anosognosia ($n = 7$) had little or no aphasia, and that 13 cases exhibited aphasia without anosognosia. However, four patients exhibiting jargon or verbal stereotypy were found to have severe anosognosia and aphasia. In three of these cases, the jargon or stereotypy was most striking in response to questions about disability. Four patients with only a mild anosognosia were aphasic but showed no jargon or stereotypic speech.

Weinstein, Lyerly, Cole, and Ozer (1966) subsequently conducted a study comparing 18 jargon aphasics with 26 "standard" aphasics (i.e., those who did not use jargon or verbal stereotypy). Structured interviews and neurological examination were the primary methods of investigation. All jargon aphasics had bilateral damage and onset of jargon was acute in 14 of the 18 cases, following trauma, craniotomy, or ruptured aneurysm. "Standard" aphasics tended to have strictly unilateral brain lesions. All jargon aphasics appeared unaware of their abnormal speech and behaved as if they were being understood. Fourteen of the 18 jargon aphasics persistently denied any speech disturbance or difficulty "putting their thoughts into words" (p. 173) when specifically questioned. In additon, they tended to deny other deficits, such as

motor or visual defects, suggesting that unawareness was not specific. These patients were generally cheerful and placid, in contrast to "standard" aphasics who frequently became upset over their errors. Weinstein et al. concluded that the necessary conditions for jargon aphasia are a left-hemisphere lesion plus some further neurological damage producing disturbed consciousness. This conclusion is consistent with the observations of Gainotti (1972),who found anosognosia in only 5 of 16 Wernicke's aphasics with left-hemisphere damage.

Several investigators have studied self-correction ability in aphasic patients. Based on clinical observations, Wepman (1958) argued that most aphasic patients are impaired, to some degree, in their ability to recognize and correct their own speech errors and this difficulty was considered to reflect the severity of the language disturbance. However, the observed self-correction deficit does not necessarily mean that aphasic patients are entirely unaware of their language disorder. On the contrary, many patients reported to Wepman that difficulty in detecting and correcting their errors was their primary aphasic disturbance. These patients demonstrate a more specific form of unawareness -an inability to monitor their speech production despite their general awareness of the aphasic disorder.

More recently, Marshall and Tompkins (1982) examined self-correction skills in 42 aphasic subjects. Aphasic subjects were classified by speech fluency (fluent vs. nonfluent) and verbal ability (high vs. low), producing four different aphasic groups: High Fluent, High Nonfluent, Low Fluent, and Low Nonfluent. Aphasics were then subclassified by type of aphasia (e.g., Broca's, Wernicke's, anomic, etc.). Subjects' use of self-correction behaviors on 10 verbal tasks was recorded. Aphasic subjects in general attempted to self-correct on more than half of their incorrect responses. The High Fluent and High Nonfluent groups showed significantly more accurate self-correction than did Low Fluent and Low Nonfluent groups, even though the four groups did not differ in the frequency with which they attempted to self-correct. Similarly, self-correction attempts did not differ among the different types of aphasic groups, but the proportion of successful self-corrections was significantly higher for anomic, conduction, and Broca's groups than for the Wernicke's group. Thus, most aphasics were aware, to some degree, of their inaccurate responses. Furthermore, the severity of the aphasic comprehension disorder appears to be a critical factor affecting self-correction competence: Anomic and Broca's aphasics, who tend to have better auditory comprehension than Wernicke's aphasics, achieved the highest self-correction success whereas Wernicke's aphasics, whose auditory comprehension is typically severely impaired, had the lowest self-correction success.

Marshall, Rappaport, and Garcia-Bunuel (1985) subsequently described a patient with severe auditory agnosia and Wernicke's aphasia whose self-correction performance was similar to that of the Wernicke's subjects in Marshall and Tompkins' (1982) study. The patient was aware of her speech-production deficits but had difficulty correcting her errors. She frequently

attempted to correct phonemic errors but generally ignored her semantic errors. The remarkable feature of this patient is that despite her inability to comprehend speech, she was aware of her verbal production errors and showed spontaneous efforts to correct some of them. Marshall et al. (1985) concluded that an auditory comprehension defect may co-occur in aphasics with intact ability to recognize speech production errors. Those aphasic patients who do not acknowledge their errors are likely unaware of them. An early case report of a so-called "pure" auditory agnosia (Cohn, et al., 1947) indicated that auditory agnosia without aphasia may be accompanied by unawareness of meaningless speech. This observation resembles the findings of Marshall et al. (1985) that their patient appeared unaware of her semantic errors. Perhaps the brain lesion producing the agnosia is also involved in disturbed awareness.

Critical Assessment

Experimental findings regarding the relation between anosognosia and aphasia are largely consistent with clinical observations of jargon aphasics, but the experimental studies are methodologically deficient in several respects. A problem discussed in previous sections, and relevant to studies in this section, is the absence of objective measures of anosognosia. Several authors based their evaluation of unawareness simply on whether the patients denied and/or appeared to ignore their deficits. Aside from the obvious subjectivity involved in this assessment technique, it is based on the questionable assumption that patients who deny or ignore their deficits are necessarily unaware of them. By contrast, deficient monitoring of speech output has been assessed by objective techniques, such as measurement of self-correction attempts and successes (Marshall & Tompkins, 1982). Continued development of methods for investigating unawareness in different aphasic groups is clearly necessary.

Miscellaneous Unawareness Phenomena

The studies considered in the foregoing sections constitute the bulk of the clinical and experimental observations concerning unawareness of deficits in neuropsychological syndromes. However, varying levels of anosognosia have been reported in several other neuropsychological disorders, including visual agnosia (Gelb & Goldstein, 1938), auditory sound agnosia (Roth, 1944), movement disorders (Meador, Watson, Bowers, & Heilman, 1986; Roth, 1944), phonemic dyslexia (Patterson, 1978), and perceptual object reversals (Feinberg & Jones, 1985). In addition, anosognosia has been reported in a number of pathological conditions that are not ordinarily considered among the classical neuropsychological syndromes. These include poriomania, where epileptic patients engage in prolonged wandering behavior of which they are unaware (Mayeux, Alexander, Benson, Brandt, & Rosen, 1979); tardive dyskinesia or involuntary movement that occurs without awareness in schizophrenic patients (e.g., Alexopulos, 1979; Myslobodsky, 1986; Myslobodsky, Holden, & Sandler, 1986; Myslobodsky, Tomer, Holden, Kempler, & Sigal, 1985); acute confusio-

nal states resulting from toxic disorders (Chédru & Geschwind, 1972) or right middle cerebral artery infarctions (Mesulam, Waxman, Geschwind, & Sabin, 1976); multiple sclerosis, which affects the white matter of the central nervous system and can produce both euphoria and anosognosia (Harrower & Kraus, 1951; Peyser & Poser, 1986; Sai-Halasz, 1956; Surridge, 1969); and personality disorders in which patients have little or no insight into their problems (Horton, 1976; Ley & Bryden, 1981). Although relatively little work has been done to delineate the nature of unawareness in these conditions, the occurrence of anosognosia in diverse conditions underscores the pervasiveness of the phenomenon.

THEORIES OF ANOSOGNOSIA

We have emphasized that numerous methodological shortcomings are evident in studies of anosognosia. Consequently, the available data base provides a tenuous foundation for theoretical development. The inadequacies of these investigations have not, however, prevented authors from proposing theoretical interpretations and explanations of the phenomenon. This section will review the various theories of anosognosia. Most of these have focussed exclusively on anosognosia for hemiplegia and visual defects.

Neuroanatomically Based Theories

The majority of investigators have subscribed to some form of neuroanatomical theory, attributing anosognosia either to focal brain lesions or to diffuse brain damage which disrupts the functioning of a mechanism or mechanisms necessary for normal awareness of a neuropsychological deficit.

Focal lesion explanations. Proponents of this view generally agree that anosognosia results from lesion sites in the right hemisphere, usually involving the parietal region and its connections (Barkman, 1925; Critchley, 1953; Denny-Brown & Banker, 1954; Denny-Brown, Meyer, & Horenstein, 1952; Gerstmann, 1942; Geschwind, 1965; Hécaen & Albert, 1978; Koehler et al., 1986; Nielsen, 1938; Olsen & Ruby, 1941; Roth, 1949; Spillane, 1942; Von Hagen & Ives, 1937, 1939; Warrington, 1962). However, the idea that anosognosia is exclusively produced by lesions of the right hemisphere has been questioned by several investigators who found that anosognosia for hemiplegia may occasionally be observed in patients with left-hemisphere damage (e.g., Cutting, 1978; Denny-Brown & Banker, 1954; Denny-Brown et al., 1952; Hemphill & Klein, 1948; Nathanson, et al., 1952; Olsen & Ruby, 1941; Paterson & Zangwill, 1944; Weinstein & Cole, 1963; Weinstein & Kahn, 1955). But relative to anosognosia resulting from right-hemisphere lesions, these cases are rare. The reported cases of anosognosia in patients with restricted left-hemisphere lesions are not terribly convincing: Investigators have usually failed to confirm that damage was

restricted to the left hemisphere, and it was frequently unclear whether the left hemisphere was, in fact, dominant in these individuals. Several investigators (Battersby et al., 1956; Brain, 1941; Nielsen, 1938) have attributed the significantly higher frequency of anosognosia following right-hemisphere damage than left-hemisphere damage to the fact that large posterior lesions in the left hemisphere may produce aphasia in addition to anosognosia, precluding assessment of disturbed awareness in many patients. However, even in cases with left parietal damage resulting in mild speech difficulties which do not prevent diagnosis of anosognosia, the predominance of anosognosia in patients with right-hemisphere lesions remains (e.g., Brown, 1972; Cutting, 1978; Gainotti, 1972; Hécaen & Albert, 1978; Nathanson et al., 1952; Weinstein & Kahn, 1955).

Advocates of the focal lesion explanation often view anosognosia as a disorder of cognition arising from a deranged body scheme (Roth, 1949). The ideas of *body scheme, body image,* or *somatognosia* (Hécaen & Albert, 1978) refer to a concept or sense of one's own body and bodily condition which is "outside of central consciousness" (Gerstmann, 1942, p. 901). According to this interpretation, the cerebral lesion (usually in the right parietal lobe) causes the representation of the body scheme or image to be disconnected from awareness. Consequently, the patient fails to appreciate the bodily alteration (Gerstmann, 1942; Schilder, 1935). Schilder (1935) developed the term "organic repression" to describe this process, and asserted that in most cases of anosognosia there is a disturbance "in the special parietal mechanism, the integrity of which secures the tactile postural model of the body" (p. 292). Gerstmann (1942), among others (e.g., Babinski, 1914; Denny-Brown et al., 1952) have stated that anosognosic patients are not usually intellectually impaired and that the disturbed awareness is restricted to the domain of bodily defect. However, noting that some cases of anosognosia are accompanied by a general mental disturbance, Roth (1949) proposed that the intellectual impairment may prevent recovery of the body image, thereby sustaining unawareness of the defect. Critchley (1949, 1953) argued that awareness depends on the integrity of the body image, which is mediated by sensory channels that are integrated in the parietal lobe, and is thus compromised in cases of anosognosia. Consistent with this notion, Denny-Brown et al. (1952) maintained that anosognosia results from ineffective synthesis by the parietal lobe of multiple sensory stimuli coming from one side of the body, and called this disorder "amorphosynthesis." Similarly, Frederiks (1969, 1985a) viewed anosognosia as a disorder of perceptual synthesis, claiming that patients experience "kinesthetic hallucinations" of the paralyzed limbs as a result of reduced impulses from the paralyzed side of the body. Thus, when asked by the examiner to move a paralyzed arm or leg, patients are certain that they are doing so.

The view that anosognosia reflects a disturbed body scheme is consistent with some empirical observations, but has several serious problems. First, it only accounts for unawareness of a physical defect. Unawareness of cognitive or

behavioral deficits, for example, is difficult to understand within this framework. Second, the theory is based on the implicit assumption that the parietal lobe of the right hemisphere differs fundamentally in its functional organization from the parietal region in the left hemisphere. More specifically, the right parietal lobe is regarded as the locus for body scheme in the brain and lesions of the right parietal lobe are alleged to affect only the image of the left side of the body. This raises the question of where and how the scheme for the right side of the body is organized (see Brain, 1941, for discussion). Third, not all hemiplegic patients with right parietal damage exhibit anosognosia, and some patients with no known parietal damage are unaware of their deficits. Finally, anosognosia for hemiplegia may occur without sensory impairment, which argues against Denny-Brown's notion of amorphosynthesis.

Other ideas related to the disturbed body scheme view of anosognosia have been put forward. Several investigators have proposed that disorders of sensitivity and position sense resulting from focal lesions in the right hemisphere are involved in the production of anosognosia (e.g., Babinski, 1914; Barré et al., 1923). Waldenstrom (1939) suggested that anosognosia results from a severe disturbance in the sense of position in space together with a paralysis of central origin. Nielsen (1938) argued that the lesion site causing anosognosia (isolation of the thalamus from the cerebral cortex) would prevent abnormal sensation in the limbs from reaching the cortex. Consequently the message of paralysis would not be conveyed to consciousness. These ideas are subject to many of the same criticisms as those described for the body scheme disturbance interpretation.

In contrast to the foregoing, Geschwind (1965) proposed a disconnection theory of anosognosia that emphasized isolation of cortical speech areas. He argued that lesions of the right-hemisphere association cortex are more likely to produce disturbed awareness than comparable left-hemisphere lesions because "the normally poorer linkage of the right side to the speech area, and possibly to other 'dominant' areas of the left hemisphere, is further weakened by the lesions" (p. 398). According to this view, patients are unable to "introspect" about the activities of a brain region which has no connection to the speech area. The confabulation observed in patients who deny their blindness (or other defects) may reflect attempts by the left hemisphere to explain what the patient cannot comprehend (see also, Galin, 1974). If disconnection from the speech center were the critical determinant of anosognosia, however, patients who verbally deny their defects should be able to express awareness of deficits through nonverbal means, for example, by pointing to an impaired limb or by providing nonverbal responses to questions that probe awareness. There is little evidence to suggest that patients are in fact aware of their deficits but simply lack the means to express this awareness verbally. Moreover, if the source of unawareness were solely the verbal (left) hemisphere's inability to gain access to information from the nonverbal (right) hemisphere, the patient ought to show awareness when information about the deficit is communicated directly

to the verbal hemisphere, either by visual demonstrations or repeated discussion about the deficit with an examiner. Yet these phenomena are typically not observed (Friedland & Bodis-Wollner, 1977). Therefore, unawareness of deficits must involve more than simply a disconnection of information from the language center.

A variety of neuroanatomical theories emphasizing focal lesions have been proposed to explain anosognosia for visual defects. Anton (1899, cited in Redlich & Dorsey, 1945) proposed that, when association tracts between the occipital lobes and other cortical areas are interrupted, the lack of sensory stimulation due to blindness is no longer perceived. Magitot and Hartmann (1927, cited in Hécaen & Albert, 1978) maintained that any lesion of the visual cortex produces both a visual field defect and unawareness of the defect. They regarded the unawareness as a "reduction of the internal perceptual field." Some authors contend that the bilateral lesions and substantial tissue destruction resulting in cortical blindness produces additional neural dysfunction that is responsible for the anosognosia. Cobb (1943) suggested that a disconnection of reverberating circuits between the thalamus and visual cortex produces Anton's syndrome. Hécaen and Albert (1978) proposed that a combination of factors lead to the development of anosognosia for visual defects. These factors include sudden sensory deprivation resulting from the occipital lesion, disinhibition of visual imagery, and confusion. The memory defect and confabulation often observed in these patients have also been regarded as contributing factors to the occurrence of anosognosia. Redlich and Dorsey (1945) suggested that memory impairment may prevent patients from learning about their blindness. Gloning, Gloning, and Hoff (1968, cited in Brown, 1972) postulated that unawareness of blindness may occur as a consequence of some residual vision, but there is no evidence to support this contention. Overall the foregoing theories are all deficient in one or more of the following ways: (1) the theory is limited to explaining unawareness of visual defects and therefore cannot be applied generally to account for all types of anosognosia; (2) the theory does not explain unawareness of visual defect in patients with no intellectual or memory impairment; and (3) the theory does not account for unawareness in patients whose blindness results from peripheral lesions.

Bisiach, Meregalli, and Berti (1985; see also Bisiach et al., 1986) focussed on the domain-specificity of anosognosia and its relation to particular lesion sites in developing a cognitive model of the awareness disorder. They viewed anosognosia and related phenomena as "modality-specific disorders of thought" resulting from disruption of specific mechanisms that normally monitor the output of individual perceptual and cognitive modules. The model was introduced by applying it to anosognosia for visual defects. Bisiach and colleagues initially stated that patients who become blind as a consequence of peripheral lesions acknowledge their visual defect and behave in a realistic manner. However, patients with more central lesions of the visual system resulting in blindness have an associated "visuo-specific cognitive dysfunction manifesting itself in a

disordered monitoring of the disability" (p. 7). These patients deny their blindness and/or act as if they can see. The model defines the first type of blindness as failure of the "sensory transducer," which transmits impulses from the retina to the brain (e.g., optic nerve), and the second as a breakdown of a "sensory processor" that mediates between the transducer and the neural regions where visual input is cognitively processed. Bisiach and colleagues proposed that messages flowing from modality-specific sensory processors travel along relatively independent paths in the direction of various "response-systems." The authors thus argue for a modular structure of central processing to account for the domain-specific nature of anosognosia and reject the notion of a central, higher order monitoring system on the grounds of its inability to explain the specificity of anosognosia - that is, if impairment of a single, monolithic monitoring system were responsible for unawareness, one would not expect patients to be aware of one deficit and unaware of another (we present an alternative view on this point in the next section). Unawareness of multiple deficits could be accounted for by postulating disruption of multiple monitoring mechanisms. This model is consistent with many of the clinical and empirical findings on anosognosia and appears to be well worth developing further. However, some unawareness phenomena are not readily described in terms of disruptions of individual monitors associated with specific modular functions. For example, head-injured patients may be unaware of personality changes and behavioral deficits. It is not clear how a model such as that of Bisiach and colleagues would account for unawareness of such global functions, which are not readily identified with individual modules.

Stuss and Benson (1986) discussed the possible contribution of frontal-lobe damage to the pathogenesis of anosognosia. They argued on several grounds that regions of the frontal lobe are involved in self-awareness and monitoring of one's own cognitive function, and that anosognosia could be viewed as a deficit in self-monitoring (cf. Anderson, 1986). The converging clinical and empirical evidence that unawareness of memory deficits is generally observed in cases of amnesia attributable to various etiologies involving the frontal lobes strongly suggests that frontal malfunction contributes to unawareness of deficits. Frontal involvement in unawareness of deficits is further supported by the substantial literature on head-injured patients who often exhibit symptoms of frontal-lobe damage in addition to unawareness of deficits. Stuss and Benson postulated that anosognosia probably results from simultaneous lesions of several cerebral areas, with varying degrees and combinations producing different forms of the disorder. The particular type of anosognosia (e.g., Anton's syndrome) may be dependent on a specific combination of brain deficits.

Nauta (1971) provided a detailed analysis of the frontal lobe and its relation to other regions of the brain which may be relevant for thinking about the neuroanatomical substrates of anosognosia. Most importantly, he described two cortical association areas where visual, somesthetic, and auditory pathways converge. The first convergence of these modalities occurs in the inferior

parietal lobule and, at a later stage of processing, these systems converge in the frontal lobe. There are reciprocating afferent connections between the frontal lobe and inferior parietal lobule. As we suggest in the next section of the article, this relation may provide a unifying link between different forms of unawareness phenomena. For example, unawareness of hemiplegia may result from lesions in the inferior parietal lobule that disrupt processing at an early stage of integration, whereas anosognosia for memory deficits or behavioral changes may require frontal lesions where processing has reached a higher level of integration. A different view has been put forth by Hier et al. (1983b), who conceptualized anosognosia as a disturbed function which is served by a diffusely organized structure. This notion was based on the observation that recovery from anosognosia occurs more rapidly than for motor and visual functions which were thought to be mediated by tightly organized structures. It would be difficult to explain the specificity of anosognosia with this general view of structural organization.

Several investigators have related anosognosia to an affective disturbance resulting from lesions in particular neural regions. Bear (1982) described anosognosia as a failure in "emotional surveillance." The patient does not detect a severe threat and, consequently, exhibits no emotional concern. Bear noted the critical involvement of right-hemisphere damage in anosognosia, especially right parietal and dorsal frontal lesions. He recommended that rehabilitation strategies include telling the "verbal (left) hemisphere" repeatedly about the tendency to neglect, minimize, and misperceive deficits. Gainotti (1969) reported the prevalence of indifference, independent of intellectual impairment, in stroke patients during the acute phase of extensive right-hemisphere damage. He observed that some patients became aware of the severity of disability during the early phase and develop a "catastrophic reaction" to this knowledge. Gainotti also commented on the similarity between the indifference behavior seen in anosognosics and mental disturbances characteristic of frontal lesions. Heilman, Schwartz, and Watson (1978) reported hypoarousal in patients with the neglect syndrome and emotional indifference. They suggested that, since many neglect patients have anosognosia, a defective arousal system arising from a lesion in the corticolimbic reticular loop may also cause the awareness disturbance.

The view that anosognosia is secondary to a neurologically based affective disturbance presents several problems. First, it does not account for the frequent domain specificity of anosognosia: If unawareness reflects only lack of concern or indifference, such indifference should be expressed with respect to all deficits. Second, this account would have serious difficulty explaining the persistence of anosognosia in the face of repeated demonstrations of a deficit; patients who are simply "unconcerned" about their deficit should acknowledge it upon confrontation. Although indifference and anosognosia often co-occur, it seems likely that indifference may be secondary to the awareness disorder or may be attributable to the proximity of neuroanatomical substrates underlying

the affective and awareness disturbances. Consistent with the latter account, evidence suggests that frontal and parietal regions may be involved in both disorders.

Diffuse damage explanations. A second group of neuroanatomically oriented theorists consists of those who view anosognosia as a manifestation of a general mental disorder that can be associated with diffuse brain pathology (Battersby et al., 1956; Cole et al., 1968; Goldstein, 1939, 1942; Redlich & Bonvicini, 1907; Sandifer, 1946; Schilder, 1935; Stengel & Steele, 1946; Ullman, 1962; Weinstein & Kahn, 1955). Sandifer (1946) argued that the severe forms of anosognosia only occur in association with intellectual impairment from diffuse damage. Schilder (1935) noted that "organic repression," a concept discussed earlier with respect to focal lesions, can also operate in cases of diffuse impairment. Ullman (1962) emphasized the necessity of diffuse cerebral dysfunction for the development of anosognosia in stroke patients and suggested that unawareness of a deficit may reflect an impairment in "abstract function" (p. 93). Weinstein and Kahn (1955) also considered a general alteration in brain function important in sustaining anosognosia, although, as discussed below, they asserted that the brain pathology does not cause anosognosia. They stated that: "The effect of the brain lesion is to provide a milieu of function in which *any* incapacity or defect *may* be denied whether it is hemiplegia, the fact of an operation or an unfortunate life situation" (p. 96).

There are several limitations to accounts which rely on general intellectual impairment to explain anosognosia. First, anosognosia has been observed in some patients who were allegedly free of any general intellectual impairment (e.g., Babinski, 1914; Barkman, 1925; Cutting, 1978; Gerstmann, 1942; Gilliatt & Pratt, 1952; Joltrain, 1924). However, it must be noted that this claim is based largely on clinical observation, and detailed assessment of intellectual functioning was not provided in these cases. Second, the specificity of anosognosia for a particular deficit with good awareness of other co-occurring defects argues against the general mental impairment view. If global intellectual deterioration produces anosognosia, patients would be expected to deny all their disabilities. Although such a pattern has been observed, the repeated observations of specificity of anosognosia represent a serious problem. Third, there may be a lack of correspondence between the onset of anosognosia for different deficits (Brown, 1972; Roth, 1944). If a generalized intellectual disturbance is responsible for producing anosognosia, it would presumably cause anosognosia for multiple defects simultaneously.

Motivational Theories

In contrast to neurophysiologically based theories, several investigators have proposed that anosognosia reflects primarily motivated use of the psychological defense mechanism of denial. The major proponents of this view are Weinstein and Kahn (1955), who interpreted anosognosia within a psychodynamic frame-

work. Based on their clinical observations of patients, Weinstein and Kahn hypothesized that premorbid personality factors are critically involved in the development of anosognosia. This idea was supported by their finding that anosognosia occurred only in patients with particular premorbid personality characteristics. Such patients had always regarded illness as an imperfection, had a history of denying their perceived inadequacies, had compulsive drives, and had a great need for prestige and the esteem of others. Anosognosia was therefore considered a manifestation of the patient's "drive to be well," a means of protection against the recognition of disease or defect. This psychological defence mechanism was always associated with other changes in behavior such as disorientation, reduplication, and paraphasia. In contrast to many authors who stressed the domain-specific nature of anosognosia, Weinstein and Kahn argued that anosognosia usually occurs for multiple defects simultaneously, for example, hemiplegia, a craniotomy, and a sense of inadequacy. Goldstein (1939, 1942), another motivational theorist, viewed anosognosia as a "quite normal biological reaction to a very grave defect" (p. 39). He regarded unawareness of deficits as a coping mechanism - a way of avoiding severe anxiety. Rosenthal (1983) considered denial in head-injured patients to be a secondary behavioral disturbance employed as an adaptive mechanism. He noted that patients rarely deny the injury itself or the physical defects resulting from the injury. Rather, they tend to minimize or completely deny the cognitive deficits. Furthermore, patients frequently cooperate with rehabilitation efforts to remediate mental deficits even though they persistently deny the problems. Similarly, Guthrie and Grossman (1952) conceptualized anosognosia as an adaptation to stress observed in patients who are facing "internal disorganization" and an inability to cope.

Psychodynamic and motivational acounts of anosognosia are subject to a variety of criticisms. First, a case reported by Stengel and Steele (1946) of a paraplegic patient who developed anosognosia following a cerebral hemorrhage despite having been well aware of his paralysis for many years argues against the notion that personality factors determine anosognosia. If personality factors are responsible for denial of deficits, this patient would have been expected to deny the paraplegia from its onset, not years later after a hemorrhage. Second, the specificity of anosognosia also poses serious problems for a psychodynamic interpretation: One would expect that patients who use the defense mechanism of denial would apply it to all serious deficits (Bisiach, in press). Third, Weinstein and Kahn's (1955) explanation does not account for the frequently observed relation between lesion site and unawareness. Thus, this theory cannot explain why damage to parietal and frontal lobes seems to be associated with the appearance of anosognosia. If unawareness and denial were purely a function of defensive mechanisms and premorbid personality, site of brain damage should be uncorrelated with incidence of anosognosia. Similarly, this theory has difficulty accounting for differences in the frequency of anosognosia between the two hemispheres (Bisiach, in press), although Weinstein and

Kahn did attempt to deal with this point (see Weinstein et al., 1964). Fourth, anosognosia generally occurs immediately following neural insult when patients are still confused and disoriented and frequently disappears in the following few hours or days during which patients begin to appreciate the implications of the brain damage. The time course for motivated denial would be expected to mirror this pattern of development rather than parallel it - those employing denial as a psychological defense mechanism would presumably exhibit increased denial as they become more aware of the severity of their deficits (Bisiach, in press). Fifth, as Cappa et al. (in press) noted, anosognosia for hemiplegia may remit upon vestibular stimulation ipsilateral to the side of lesion. Although the reasons for this effect are not understood, it is not clear how a motivational interpretation of anosognosia would account for the finding. Sixth, Critchley (1949) rejected a "repression" explanation for anosognosia in hemianopic patients on the grounds that these patients do not suffer from severe disability in everyday life nor do they experience significant embarrassment as a function of their defect. Yet, despite the relative absence of reasons for motivated denial, they remain completely unaware of and deny any visual loss. Finally, Stuss and Benson (1986) have criticized the view that psychodynamic factors are the primary determinants of anosognosia, arguing that "similar factors are so prevalent in the general population that it becomes difficult to accept that they cause a striking syndrome such as anosognosia in one individual but not in another. That psychogenic factors contribute to unilateral inattention or denial in some individuals can be hypothesized; that they are the major factor in most appears dubious" (p. 120; see also Frederiks, 1985a; Grimm & Bleiberg, 1986).

Toward a Theoretical Integration

As indicated by the foregoing discussion, no single theory provides an entirely satisfactory account of unawareness of deficits in neuropsychological syndromes. We now sketch the outlines of a theoretical framework that we believe can accommodate a number of important phenomena. This framework does not attempt to account for defensive denial; though a clinically significant phenomenon, it lies outside the purview of our theoretical discussion.

Our approach is based on a descriptive model outlined recently by Schacter (in press), referred to as Dissociable Interactions and Conscious Experience (DICE). For the present purposes, a key idea in this model is that a conscious experience of remembering, knowing, perceiving, or comprehending - phenomenal awareness of a particular kind of information - requires the activation of a specific system that is distinct from, but interacts with, modular systems concerned with language, memory, perception, and so forth. It is further postulated that this *conscious awareness system* (CAS) takes as its input certain kinds of output from perceptual, memory, and knowledge modules - highly activiated information that reflects a significant change from the resting or "baseline" state of the module. Sufficiently low levels of activation in a particu-

lar module, by contrast, would not constitute input to CAS. According to the model, CAS can be selectively disconnected from specific modules in different neuropsychological syndromes, thereby resulting in domain-specific deficits of consciousness (for pertinent evidence, see Schacter, in press; Schacter, McAndrews, and Moscovitch, 1988). DICE further postulates that, at the neuroanatomical level, CAS is a posterior system involving the inferior parietal lobes and structures connecting them, most importantly the cingulate area in the splenium of the corpus callosum. This idea and the evidence supporting it had been discussed originally by Dimond (1976), who argued for the existence of a "consciousness circuit" extending across the posterior cortex, with inferior parietal regions constituting the lateral endpoints of the circuit.In DICE, CAS has an output link to an *executive system* that is involved in the initiation, organization, and monitoring of complex sequences of ideas and actions. Consistent with previous suggestions made by others, the model postulates that frontal regions, which are known to subserve complex integrative functions (for review, see Stuss & Benson, 1986), constitute the neural basis of the executive system.

Let us now consider these ideas in relation to anosognosia. According to the model, unawareness of deficits could result from disruptions at the level of either the posterior CAS or the anterior executive system. This idea is broadly consistent with the bulk of the literature which, as stated earlier, implicates both parietal and frontal regions in anosognosia. Consider first anosognosia that is attributable to disruption at the level of CAS. In DICE, there are two ways in which such disruption could occur: damage to CAS itself, or selective disconnection of CAS from a particular input module that is damaged as a consequence of neural insult. The former type of disruption would be expected to result in unawareness of all neuropsychological deficits that occur in a particular patient, because CAS would not respond appropriately to inputs from any damaged modules. In contrast, the latter type of disruption would produce specific anosognosia. If CAS no longer received the highly activated information that normally "alerts" it to a change in the state of a module, it would behave as if the module were in a "baseline" state of low activation - that is, CAS would have no information about the module's damaged condition. However, CAS would continue to receive distorted input from all other damaged modules (and normal input from intact modules), thus producing normal awareness of their activity. Accordingly, the model can handle the observations of both selective and generalized unawareness of deficits that have been reported in the literature.

Consider next instances of anosognosia that are attributable to disruption of the frontal executive system. This kind of unawareness could result from either damage to the executive system itself or disconnection of the executive from CAS. The consequences in either case, however, would be similar: disrupted awareness of complex functions that normally require the executive system for integration and monitoring. Thus, the model leads us to suggest that different

types of anosognosia will be observed as a consequence of parietal and frontal damage. Unawareness of specific perceptual and motor deficits can occur with parietal damage, as observed in the classical anosognosias for hemiplegia, hemianopia, and so forth. In contrast, we suggest that frontal damage is associated primarily with unawareness of more complex deficits, such as difficulties in problem-solving, retrieving and integrating information, as well as social, behavioral, and personality changes. Empirical observations discussed earlier in the article are generally consistent with this notion.

The foregoing constitutes no more than a preliminary and rather general framework for theorizing about unawareness of deficits, one that is similar in some respects to ideas put forward by Bisiach et al. (1986), Stuss and Benson (1986), and others. It does not attempt to account for all of the individual observations of unawareness reported in the literature, does not speak directly to those cases of anosognosia that do not appear to involve either parietal or frontal regions, and is mute concerning various important problems, including the predominance of right-sided damage in anosognosia, the role of defensive denial, and so forth. Nonetheless, the model is broadly consistent with major trends in the literature, and has heuristic value insofar as it focusses attention on such important issues as the differences between parietal and frontal anosognosia, and the kinds of unawareness observed following selective disconnection of CAS from particular modules versus damage to CAS itself. More generally, we wish to emphasize that anosognosia can be viewed profitably within the context of other neuropsychological deficits in which disruptions of awareness are a prominent feature (for discussion, see Dimond, 1976; Schacter, in press; Schacter et al., 1988; Stuss & Benson, 1986).

UNAWARENESS OF DEFICITS: FUTURE DIRECTIONS

It is clear from the numerous observations discussed in this review that there has been a good deal of interest in unawareness of neuropsychological deficits during the past 100 years. Nevertheless, it seems equally clear that systematic investigation of the problem has begun only recently and will likely increase in the future. We now consider possibly fruitful directions for future research.

One of the points that we have stressed repeatedly concerns the dearth of appropriate methods for assessing unawareness objectively and quantitatively. Although some inroads have been made through the use of questionnaires, experimental paradigms, and rating scales, these developments are not widespread and are themselves characterized by various methodological problems discussed earlier. What is needed now are assessment tools that provide reliable information concerning the *degree* and *quality* of unawareness in various patient groups so that it will be possible to answer questions such as the following: Are there different levels of unawareness and if so, how can they be characterized? Since anosognosia is often a transient phenomenon, what can be said about the nature

of the progression from unawareness to awareness of a deficit? When individual patients are unaware of multiple deficits, is the nature of the unawareness identical for all deficits, and if not, how can any differences be characterized? What role, if any, do confabulation and delusion play in the development and/or manifestation of anosognosia? Answers to such questions will be needed if our understanding of unawareness phenomena is to progress beyond its current modest level, and satisfactory answers will require new investigative techniques.

Some helpful clues concerning the development of research tools are provided by the literature on metacognition. Metacognition refers to the processes whereby people monitor and reflect upon their own cognitive processes (e.g., Flavell, 1981). Such processes appear to be deficient in anosognosic patients. Metacognition has been investigated in some studies of normal adults, particularly in the area of memory (e.g., Hart, 1965; Herrmann, 1982; Nelson & Narens, 1980; Schacter, 1983) and has been examined extensively in developmental research. Here, paradigms and techniques have been developed for studying metacognitive awareness of memory (e.g., Brown, 1978; Cavanaugh & Borkowski, 1980), comprehension (e.g., Garner, 1980; Markman, 1985), language production (e.g., Clark, 1978; Van Kleeck, 1982), reading (e.g., Brown, 1980), and attention (e.g., Yussen & Bird, 1979). Neuropsychological investigators of anosognosic phenomena would do well to attempt to adapt some of these paradigms for investigation of unawareness in various patient groups. Indeed, use of experimental techniques for investigating metacognitive processes has already proved helpful in characterizing unawareness phenomena in amnesics (Shimamura & Squire, 1986), aphasics (Marshall & Tompkins, 1982), and dyslexics (Patterson, 1978), and the extensive developmental literature on metacognition could contribute substantially to further progress.

A second, related issue that clearly requires intensive investigation concerns the relations between neurologically based unawareness and defensive or motivated denial. As stated earlier, the notion that all anosognosic phenomena are entirely or partly attributable to defensive denial can be rejected. Nonetheless, it is quite likely that motivated denial plays a role, perhaps a very large role, in some cases of anosognosia. Consistent with this possibility, denial has been observed in various patients with injuries and illnesses that do no not involve brain damage, including severe burns (Hamburg, Hamburg & deGoza, 1953), spinal cord injury (Trieschmann, 1980), heart disease (Hackett & Weisman, 1969), and cancer (Levine & Zigler, 1975; for review, see Breznitz, 1983; Caplan & Schechter, 1987). These observations suggest that the use of denial as a defense mechanism can operate in the absence of brain damage. In order to make appropriate inferences regarding the nature of unawareness following brain damage, it is thus crucial to distinguish the defensive component of denial, which can be observed even after nonneurological disease, from the neurogenic component, which is directly produced by a specific kind of brain damage. No firm criteria have yet been developed to differentiate the two. One possibility is

that patients with neurogenic unawareness are characterized by neuropsychological signs not observed in patients with defensive denial. Elucidation of such signs would be an important task for future research.

A third important area concerns the neuroanatomical basis of anosognosia. One key issue that requires further investigation concerns whether and to what extent diffuse brain damage and consequent generalized intellectual impairment contribute to anosognosia. This problem has surfaced repeatedly in different areas of investigation and has not yet been resolved satisfactorily. Although there is evidence that anosognosia occurs without diffuse brain damage and global intellectual deficits (e.g., Babinski, 1914; Bychowski, 1920; Gerstmann, 1942; Gilliatt & Pratt, 1952; Roth, 1949), there is also evidence indicating that unawareness can diminish or disappear when conditions such as confusion and disorientation resolve (e.g., Bender et al., 1949; Nathanson et al., 1952; Ullman, 1962). Another important problem concerns the critical lesion site or sites that produce anosognosia. Though it has long been acknowledged that there is a greater incidence of unawareness following right-than left-hemisphere damage, this phenomenon is still poorly understood. In addition, there has been no attempt to compare directly and systematically the unawareness phenomena attributable to parietal and frontal lesions, even though such comparisons are critical for the kind of model that we outlined in the previous section. Moreover, some cases of anosognosia have been reported that do not appear to involve either parietal or frontal damage. For example, anosognosia has been reported in several cases where damage was restricted to subcortical structures possibly affecting a cortico-limbic reticular loop (e.g., Healton, Navarro, Bressman, & Brust, 1982; Jacome, 1986). This finding raises the question of how subcortical-cortical pathways are organized such that damage at either level can produce anosognosia. It also poses a challenge for the view of anosognosia put forward in the previous section. Clearly, considerable research will be needed in order to clarify the neuroanatomical basis of anosognosia.

A fourth area that needs to be explored further concerns training patients who are unaware of their deficits to become aware of them. As noted earlier, several investigators have pointed out that unawareness represents a major obstacle for rehabilitation efforts and causes numerous problems in patients' social adjustment (Ben-Yishay et al., 1985; Fordyce & Roueche, 1986; Prigatano, 1986). Yet, with the exception of Prigatano and Fordyce's (1986a,b) research program and a few scattered studies, there is a virtual absence of literature on awareness training. One possible reason for this is that in severe forms of anosognosia, patients do not become aware of their deficit even after repeated demonstrations of it. The seeming intractability of some anosognosic patients would appear to militate against any attempts at awareness training. Nevertheless, not all patients are characterized by such intractability, and it is not clear whether sufficiently powerful techniques have been used to induce awareness in those who are. For example, recent research has indicated that successful training of brain-damaged patients with memory disorders requires

massive repetition (e.g., Glisky & Schacter, 1987; Schacter & Glisky, 1986), and the same may be true of anosognosic patients. Attempts at awareness training must also carefully distinguish between psychogenic and neurogenic contributions to denial, because different intervention strategies may be called for in the two cases (Prigatano, 1986).

Finally, it is clear that a great deal more effort needs to be devoted to the development of theoretical models and conceptualizations of unawareness phenomena. A notable feature of the literature is that there has been little attempt to make use of relevant concepts from other domains of psychological inquiry. One possibly useful source of new ideas and theoretical constructs is the aforementioned literature on metacognition; another is research concerning the phenomenon of self-monitoring. Recent work in social cognition has explored situations in which people persist in holding inaccurate beliefs about the self (e.g., Lepper, Ross, & Lau, 1986) and provided models of how information is related to and monitored by the self (e.g., Greenwald & Pratkanis, 1984; Kihlstrom & Cantor, 1984). Theorizing about unawareness phenomena could profit by making use of current ideas on self-monitoring. Equally important, findings concerning anosognosia might provide new insights into the mechanisms underlying self monitoring in normal subjects. Although work on anosognosia has not yet influenced psychological theories of self-monitoring or metacognition, it represents a potentially rich source of insight into these phenomena (Bisiach et al., 1985).

One further point regarding theoretical interpretation of anosognosic phenomena merits consideration. Unawareness of a neuropsychological deficit can be conceptualized as a failure to gain *conscious* or *explicit* access to information regarding the state of a perceptual, cognitive, or motor function: The patient is not consciously aware that a once-intact function is currently impaired. But is it possible that at an implicit or nonconscious level, the patient possesses "knowledge" that a function is impaired, knowledge that may be expressed in behavior? Some clinical observations suggest that anosognosic patients possess implicit knowledge of deficits that they deny explicitly, as pointed out by Weinstein et al. (1964):

> ... the term 'anosognosia', meaning 'lack of knowledge', is not wholly accurate. The patient indicates knowledge of the neglected extremities by referring to them in such expressions as a 'dummy' and a 'rusty piece of machinery'. Patients who deny that they are ill subscribe to hospital routine and express no surprise when they are told, for example, that they are to have a craniotomy. The very fact of the selectiveness indicates some knowledge of the deficit ... (p. 384).

These suggestive observations fit nicely with recent evidence indicating that patients with a variety of neuropsychological impairments can show implicit knowledge of information that is not accessible to conscious awareness (for

review and discussion, see Schacter et al., 1988). Further exploration of the kinds of implicit knowledge that anosognosic patients possess about impairments that they deny explicitly could increase our understanding of what it means to be "unaware" of a neuropsychological deficit.

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