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Self-Deception in Neurological Syndromes

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One of the traditional views of self-deception has been in terms of a dynamically-driven defense mechanism which is employed in order to enhance self-esteem by denying contradictory evidence. Denial is evident during stressful events in everyday life, as well as in cases of mental and somatic impairments. A detailed analysis of a specific neurological syndrome, prosopagnosia, where covert recognition of familiar faces may coexist with lack of overt recognition, demonstrates the inapplicability of the dynamic interpretation of self-deception in terms of denial to some neurological syndromes, and the usefulness of a new conceptualization of this process in terms of dissociation between modular and central processes. It is proposed that self-deception be considered a complex process which may be conceived of as a defense mechanism in everyday life, and as a product of functional dissociation in neurological syndromes.

Self-Deception and Denial in Neurological Syndromes

The purpose of the present paper is to show that since an accepted view of self-deception in dynamic terms is inapplicable to specific neurological syndromes, a reconceptualization of the process is required. One of the definitions of self-deception is in terms of a conviction "by oneself (without awareness of the truth) of an entity or event as it is not" (Lockard and Mateer, 1988, p. 24). Self-deception is considered paradoxical, since it implies that two contradictory mental contents or beliefs occur simultaneously in an individual who is not aware of one of them. Furthermore, the

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operation that determines which mental state or belief will access awareness is itself unconscious (Sackeim and Gur, 1997). Self-deception is motivated by the desire to prevent or control anxiety, and to increase subjective well-being or self-esteem, through the biasing of information and reasoning, selection of attention, adoption of unrealistic optimistic approaches, and denial of contradictory evidence (Baumeister, 1993; Erez, Johnson, and Judge, 1995; Greenwald, 1997; Kernberg, 1994; Krebs, Denton, and Higgins, 1988; Sackeim, 1988; Sackeim and Gur, 1978; Taylor, 1989, p. 124; Taylor and Brown, 1988). Endorsement of contradictory beliefs without awareness of their contradictions is made possible by compartmentalization, isolation, or "double-think" (Rokeach, 1963).

The idea that the mind can simultaneously hold two contradictory beliefs without being aware of one of them has also been advanced by Hilgard (1977) regarding specific hypnotic states. However, as Kirsch (1997) pointed out, rather than being unaware of holding two contradictory beliefs, many hypnotized subjects seem to be unaware of the discrepancy between the two beliefs.

Recently, Mele (1997) criticized the notion that self-deception entails an intention to deceive oneself (with or without awareness). He argues that simultaneous possession of contradictory beliefs is an "impossible state of mind." Instead, he suggests that self-deception be defined behaviorally in terms of employment of specific strategies (positive and negative misinterpretations, selective attention, and selective evidence-gathering), regardless of whether or not there is a conscious or unconscious intention to deceive oneself: "When, under the right conditions, such mechanisms are primed by motivation and issue in motivated false beliefs, we have self-deception" (Mele, 1997, p. 98). Empirical evidence suggests, however, that it is possible to simultaneously store apparently paradoxical information (Brown and Kenrick, 1997).

Unlike denial of an impending stressful event (such as death in the family, or progression of a terminal illness) which relates to the future, denial of physical disability has to do with a condition that can be sensorially perceived (Langer, 1994). The latter is conspicuous in neurological diseases with anosognosic symptomatology. Anosognosia refers to unawareness or denial of neurological deficits which may accompany a variety of neurological syndromes, such as hemiplegia (paralysis of one side of the body), amnesia (loss of memory), hemianopia (loss of pattern vision in either the left or the right visual field), aphasia (loss of expression or comprehension of speech), and head injury (Babinski, 1914; Bisiach, Vallar, Perani, Papagano, and Berti, 1986; Bruyer, 1991; Schacter, 1990; Weinstein, Cole, Mitchell, and Lyerly, 1964). As McGlynn and Schacter (1989) point out, ". . . a significant number of patients are entirely unaware of their deficits: some amnesic patients claim that their memory is perfectly normal, aphasic patients fre-

quently do not know that their linguistic productions lack coherence and meaning, and hemiplegic patients often do not realize, and sometime deny, that they have a motor impairment. Yet these same deficits are all too apparent to others and have a profound effect on the afflicted patients' everyday lives" (p. 144). In these cases the mental representations of given objects or events and the mental representation of the self are dissociated from each other (Kihlstrom and Tobias, 1991).

The apparent predominance of anosognosic symptomatology on the left side of the body has been linked by some authors to damage in the right cerebral hemisphere (Brain, 1940; Critchley, 1953; Cutting, 1978; Hécaen and Albert, 1978; McFie, Piercy, and Zangwill, 1950). On this hypothesis, anosognosia appears because the intact left hemisphere, which normally controls linguistic processes, can readily create "denying" interpretations of the stressful events (Weinstein and Kahn, 1955; but see Sackeim, 1983). Yet many believe that anosognosia may be associated with damage in either hemisphere, and that the predominance of the left side of the body is a methodological artifact (Battersby, Bender, Pollack, and Kahn, 1956; Denny–Brown and Banker, 1954; Sandifer, 1946; Weinstein, 1991; Weinstein and Kahn, 1955; Weinstein et al., 1964).

As a process of self-deception, anosognosia has been conceptualized in psychodynamic terms; particularly as denial of illness in order to protect or enhance mood (Guthrie and Grossman, 1952; Nathanson, Bergman, and Gordon, 1952; Sackeim, 1983; Weinstein and Kahn, 1955; Weinstein et al., 1964). Denial (or "defensive denial," as Kihlstrom and Tobias [1991] prefer to call it) was originally conceived by Freud (1923/1961) as an unconscious defense mechanism of ego integrity. As such, it allows one to escape unpleasant realities (such as deficit, illness and trauma) which cause emotional pain by ignoring their existence (Hagopian, 1993; Lewis, 1991).

The more recent analogous concepts of "false self" (the tendency to respond to the demands of others) and "self monitoring" (the way one presents one's "self" in interpersonal situations) are similarly regarded as unconscious defense mechanisms of the "true self" (the tendency to respond to one's own needs) [Bollas, 1987, 1991, 1992; Giovacchini, 1979; Horney, 1950; Kohut, 1971; Laing, 1965; Rogers, 1961; Snyder, 1979]. Similar interpretations of denial of illness as a coping or an adapting mechanism have been offered by others (Beilin, 1981; Beisser, 1979; Connor, 1992; Dansak and Cordes, 1978–1979; Dracup, Mose, Eisenberg, Meischke, Alonzo, and Braslow, 1995; Goldstein, 1939, 1942; Guthrie and Grossman, 1952; Hackett and Cassem, 1970; Lockard and Mateer, 1988; Rosenthal, 1983; for a review of psychoanalytic perspectives on denial, see Lewis, 1991).

Dorpat (1983a, 1983b, 1985, 1987) integrated psychoanalytic and cognitive approaches to the study of denial by taking into account both object

relations and interactional dynamics. Unlike Freud (1923/1961) who regarded denial as a discrete mechanism of defense, Dorpat considers it a mechanism of a preconscious reality repudiation of *all* defensive operations that allay anxiety or other unpleasurable affects. Denial, or self-deception, begins with a cognitive arrest of conscious representation of an aspect of the threatening reality. The resulting gap in the symbolic representation of reality creates a need to make up a cover story or screen (of ideas, memories, affects, fantasies, and overt behavior) in order to hide the discontinuity caused by the cognitive arrest phase. The content of the screen (at the final "positive assertion" phase) is often the exact opposite of the negated reality (at the opening "cognitive arrest" phase), so that for the person involved A *is not* true but B *is* true. The cover story thus created is rendered internally consistent and plausible (Fingarette, 1969). According to Dorpat (1987), both Freud's psychoanlytic theory and his own cognitive-arrest theory share the view that defensive activity entails elements of self-deception in terms of distortion of reality. However, the two theories differ from each other in their formulations as to how this distortion is brought about. According to Freud, the defense blocks the recovery of unconscious, undistorted memories and fantasies, whereas according to Dorpat, it blocks the construction of conscious, disturbing cognitions, so as to enable an attention shift to less disturbing cognitions.

Dorpat (1983a) takes pains to dissociate himself from the "psychoanalytic fallacy" of ascribing to the denier an unconscious knowledge of what he or she has denied, since this paradoxical formulation implies that the denier both knows something and at the same time does not know it. Unlike repression which entails an unconscious knowledge of what has been repressed, cognitive arrest in denial reactions prevents the person from creating verbal representations of whatever has been defensively negated.

The unconscious nature of self-deceptive processes was also questioned by Sartre (1969) who argues that these "bad faith" (*mauvaise foi*) processes cannot be unconscious, since concealing the truth from oneself implies the unity of a single consciousness. One must be conscious of the bad faith in order to know what not to perceive. Accordingly, flight from anguish in order not to know it is in bad faith because one cannot avoid knowing what one is fleeing from (for further discussions of Sartre's ideas, see Keen, 1973; Santoni, 1978).

Regarding illness, self-deception typically begins with a denial that illness exists. Once a diagnosis is established, its clinical significance and implications are denied. As soon as the implications are accepted, denial finally focuses on the prognosis of illness (Connor, 1992; Weisman, 1972). In a further elaboration, Breznitz (1983) proposed a seven stage model of denial of illness. The stages are selectively employed depending on the intensity, probability and imminence of the threat or danger.

A distinction must be made, however, between denial of total information which distorts reality and is therefore primitive and maladaptive, and denial of certain aspects of a threatening reality which serves as a diversional coping response and is therefore normal and adaptive (Breznitz, 1997; Dorpat, 1987; Handron, 1993). The two are distinguished from each other in terms of context, intensity and duration (Dorpat, 1987).

As shown above, as a process of unawareness or denial of neurological deficits, anosognosia was accounted for in psychodynamic terms. Criticizing these accounts, Schacter and his associates (McGlynn and Schacter, 1989; Prigatano and Schacter, 1991) contend that unawareness of neurological disorders is usually present only during the acute stage of the illness, and it disappears when the patients become aware of their conditions (a similar observation from a different point of view led Baumeister [1993] to believe that self-deception tends to vanish whenever objective evidence of deception exists). Patients are fully aware of their neurological disorders if the latter are the consequence of a lesion that does not involve neural structures responsible for higher cognitive functions. Moreover, unawareness may be limited either to the patients' verbal or to their nonverbal responses. McGlynn and Schacter (1989) further refer to a case of a patient with paralysis of the legs (paraplegia) who developed anosognosia following a cerebral hemorrhage after many years of awareness of his disability. As an adaptive mechanism denial should have been employed at an earlier stage. Psychodynamic interpretations also cannot properly account for a number of anosognosic manifestations, such as the selectivity and specificity of anosognosia, the hemispheric differences in anosognosic manifestations, the apparent link between lesion site and unawareness, the lack of correlation between severity of deficit and magnitude of denial, and the phenomenon of remittance of anosognosia for hemiplegia upon ipsilegional vestibular stimulation (McGlynn and Schacter, 1989).

Having rejected the interpretations of self-deception and denial of neurological syndromes in dynamic terms, one may now proceed with the analysis of these phenomena in neurological terms. This analysis seems advantageous over dynamic approaches, since it focuses on accounts of neurological syndromes within their own domain of discourse. The new approach will now be demonstrated by a detailed analysis of a specific neurological syndrome, prosopagnosia. In the following sections it will be shown that a given category of prosopagnosia that entails denial of residual capability, cannot be explained in dynamic terms, but rather in terms of functional dissociation which in turn may account for the patient's self-deception.

Prosopagnosia

Prosopagnosia ("not knowing faces" in Greek) is a rare neurological syndrome which consists of inability to recognize familiar faces in the absence of severe intellectual, sensory, or cognitive impairment; apparently as a result of a failure to extract physiognomic invariants, or associated semantic memories (Damasio, Damasio, and Tranel, 1990; Young, 1988). Usually, prosopagnosic patients know that they are looking at a face, and they can name its parts. Yet they cannot tell whose face it is; that is, they cannot perceive individuality (Damasio, Damasio, and van Hoesen, 1982). This deficit impairs only the normal, configurational (holistic) perception; when perceived componentially (feature-by-feature), the faces can be identified (Davidoff, 1988; Levine and Calvanio, 1989; Nardelli, Coccia, Fiaschi, Terzian, and Rizzuto, 1982; Young, Humphreys, Riddoch, Hellawell, and De Haan, 1994). However, componential perception is inefficient; presumably because it employs inferential processes (Carey, 1981, Carey and Diamond, 1977; Etcoff, Freeman, and Cave, 1991; Frith, Stevens, Johnson, Owens, and Crow, 1983; Rhodes, Brake, and Atkinson, 1993; Sacks, 1987; Sergent, 1988; Sergent and Villemure, 1989).

Cases of patients with face-specific impairments, object agnosia (inability to recognize objects) or dyslexia (reading difficulty) without prosopagnosia (Assal, Favre, and Anderes, 1984; Behrmann, Moscovitch, and Winocour, 1994; Bornstein, 1963; Bornstein, Sroka, and Munitz, 1969; Bruyer and Velge, 1981; Bruyer et al., 1983; De Renzi, 1986; De Renzi, Faglioni, Grossi, and Nichelli, 1991; Ebata, Ogawa, Tanaka, Mizuno, and Yoshida, 1991; Feinberg, Schindler, Ochoa, Kwan, and Farah, 1994; McCarthy and Warrington, 1986; Rumiati, Humphreys, Riddoch, and Bateman, 1994; Tzavaras, Hécaen, and Le Bras, 1970) seem to indicate that prosopagnosia is a specific syndrome, and by implication, that face recognition is a specific perceptual process (see Moscovitch, Winocour, and Behrmann, 1997; Nachson, 1995). Prosopagnosia is usually linked to bilateral lesions in the occipitotemporal areas of the brain (Bauer, 1984; Bruyer et al., 1983; Damasio and Damasio, 1983, 1986; Damasio et al., 1982; Damasio, Damasio, and Tranel, 1986; Damasio, Tranel, and Damasio, 1990; Hécaen, 1981; Nardelli et al., 1982; Sergent, 1995). However, the right hemisphere plays a more important role than the left in producing this syndrome, perhaps because of its greater sensitivity to critical physical characteristics (Benton, 1990; Damasio et al., 1982, 1986; Sergent, 1988; Young, Hay, and McWeeney, 1985).

Covert Recognition of Faces

As will be discussed below, lack of overt (explicit, conscious) recognition of faces does not exclude the possibility of covert (implicit, unconscious) recognition. For some prosopagnosic patients the syndrome is manifested by impairment in explicit retrieval of faces, and not in storage of facial representations. This is evident by the phenomenon of covert face recognition which was first reported by Bauer (1984) who asked a prosopagnosic patient to identify familiar faces of famous personalities and of family members. Each face was presented together with five names, one of which was the correct name. The patient's task was to match the faces with their corresponding names. Skin conductance responsivity was recorded while the patient performed the task. Correct identification of famous faces was obtained for only 20% of the stimuli. However, maximal skin conductance responsivity (an index of covert recognition) was found for 60% of all correct face–name matches. The respective figures for the faces of family members were 25% and 62.5%. Face recognition deficit was specific for familiar faces, as evident by the fact that the patient could readily match unfamiliar faces on Benton and van Allen's (1973) Face Recognition Test. Similar findings have subsequently been reported by others (Bauer, 1986; Bauer and Verfaellie, 1988; Tranel and Damasio, 1985; Tranel, Damasio, and Damasio, 1988).

The physiological findings of covert face recognition are corroborated by behavioral data. Rizzo, Hurtig, and Damasio (1987) demonstrated differential eye movements by prosopagnosic patients while watching familiar and unfamiliar faces. De Haan, Young, and their colleagues (De Haan, Young, and Newcombe, 1987a, 1987b, 1991; Young and De Haan, 1988; Young, Hellawell, and De Haan, 1988) have tested a prosopagnosic patient who showed no overt recognition of faces, but disclosed covert effects of familiarity with the faces on matching and learning tasks. Similar findings have subsequently been reported by others (Greve and Bauer, 1990; Sergent and Poncet, 1990).

Bauer's (1984) account for the phenomenon of covert recognition is based on the assumption that there are two visual pathways from the subcortical limbic system to the visual cortex, ventral and dorsal, which are associated with overt and covert recognition, respectively. When the ventral pathway is impaired and the dorsal pathway is intact, covert recognition without overt recognition appears. Recent findings (Tranel, Damasio, and Damasio, 1995) further show that overt and covert face recognitions are differentially associated with bilateral occipitotemporal and ventromedial brain areas, respectively.

New interactive models suggest, however, that there is a unitary face recognition system. When the system malfunctions, it causes a loss of overt

recognition, even as the residual, low quality functioning of the system can still evoke covert recognition (Burton, Young, Bruce, Johnston and Ellis, 1991; Farah, 1994; Farah, O'Reilly, and Vecera, 1993; van Gulick, 1994; Wallace and Farah, 1992; for a more detailed review of these models, see Nachson, 1997). Whenever a failure to overtly recognize familiar faces is not accompanied by covert face recognition, the deficit is perceptual (Bauer, 1986; Campbell and De Haan, 1994; De Haan, Bauer, and Greve, 1992; De Haan and Campbell, 1991; Humphreys, Troscianko, Riddoch, Boucart, Donnelly, and Harding, 1992; Newcombe, Young, and De Haan, 1989; Sergent and Villemure, 1989; Young and Ellis, 1989; Young et al., 1994).

There are therefore two kinds of prosopagnosia: one which is due to a deficit in perception of facial stimuli, and the other which is due to a disconnection of the visual input from stored representations (Hécaen, 1981). Clearly, whenever prosopagnosic patients recognize familiar faces covertly but not overtly, they demonstrate unawareness of their residual ability. In this respect, covert recognition is distinguishable from similar anosognosic phenomena. Anosognosic patients are totally unaware of, and therefore deny the existence of their respective deficits. By contrast, prosopagnosic patients who profess inability to recognize familiar faces, are unaware of their residual ability to covertly respond to them. The denial is therefore not of the deficit, but of the residual capability (e.g., Bodamer, 1947).[1]

Denial and Self-Deception in Prosopagnosia

All critical comments levelled against the dynamic conceptualizations of anosognosic syndromes are applicable to prosopagnosia. However, an additional point may be made regarding those prosopagnosic patients who do not deny the existence of a deficit, but are rather unaware of their residual functioning capabilities. The well-being of these patients could conceivably be enhanced by acknowledging rather than by denying their ability to recognize stimuli under certain conditions. Denial of the residual capabilities is therefore counterproductive as a defense mechanism.

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[1]As a syndrome characterized by unawareness of the residual capability, prosopagnosia shares some features with another neurological syndrome, blindsight. Blindsight refers to a visual field defect which results from partial destruction or dennervation of the visual area in the cortex (Poppel, Held, and Frost, 1973; Weiskrantz, Warrington, Sanders, and Marshall, 1974). Blindsight patients are consciously blind to visual stimuli presented in parts of the visual field which project to the damaged cortical area. Some patients, however, can nonetheless detect stimulus location, movement direction and velocity, and size (Cowey and Stoerig, 1992; Stoerig, 1987, 1992; Stoerig and Cowey, 1989a, 1989b; Weiskrantz, 1980, 1986, 1987; Weiskrantz et al., 1974). They therefore have covert recognition of particular stimulus attributes. The following analysis is therefore applicable also to blindsight.

Alternatively, Schacter, McAndrews, and Moscovitch's (1988) and De Haan et al.'s (1992) descriptive models of object and face recognition, may account for both anosognosic deficits and prosopagnosic symptoms, despite the differences between the two syndromes. According to this model, when the modular output of facial stimuli does not reach the conscious awareness system due to a specific neurological deficit, there is no overt awareness of the face, but knowledge can still be covertly expressed through verbal, motor and other bodily responses. While both overt and covert face recognitions engage the same neurological functions, they clearly differ from each other functionally; for example, by employing two independent, incompatible (contradictory) memory systems (see Brown and Kenrick, 1997).

According to this model, the phenomenon of implicit (covert) knowledge without explicit (overt) knowledge is accountable in terms of functional dissociation between specific modular processes and the general cognitive system. Since the dissociation occurs irrespective of the content of the modular system, in a given case (e.g., anosognosia) it may be manifested by denial of a defective cognitive function, whereas in another case (e.g., prosopagnosia) it may be manifested by denial of a residual cognitive capability.

Regardless of etiology and dynamics, all the implicit–explicit dissociations in neurological syndromes may be regarded as instances of self-deception in the sense that the afflicted persons consciously believe that they are either capable or incapable of performing a given task, whereas in fact the opposite is true. Self-deception may therefore be conceived of in terms of dissociation between a given modular system and the general cognitive system. Although similar dissociations have been observed across different anosognosic syndromes, experimental tasks, types of information, and perceptual and cognitive processes, all syndromes are domain-specific in the sense that impaired access to consciousness is limited to the affected domain (Bisiach et al., 1986; Schacter, 1989). Thus, prosopagnosic patients who covertly recognize familiar faces without overt awareness may be quite accurate in describing other disabilities that they might have.

The last point is particularly important as it helps distinguish between normal and pathological implicit recognition. Normal implicit recognition without conscious awareness is unlimited in scope and sense modality, and is clearly adaptive. For example, it enables episodic integration by rapid association and recall of sensations and thoughts (Schacter, 1996). Moreover, although one may be unaware of a specific implicit effect (e.g., in priming where prior exposure affects responsivity to stimuli), one is certainly aware of the existence and effects of implicit knowledge in general (e.g., in habitual behavior which is carried out while attention is focused elsewhere). Normally, implicit and explicit memories coexist in a complementary fashion. For example, in perceiving a familiar face, episodic memories may come

to mind automatically (implicitly), but additional information can be retrieved at will (explicitly). Therefore, implicit memory among normals has nothing to do with self-deception.

This is not the case, however, in neurological syndromes where implicit recognition, which is limited to the affected function, substitutes rather than complements explicit recognition. Those prosopagnosic patients who respond to the presentation of familiar faces only implicitly are aware of their cognitive, motor and sensory functions, except for face recognition. Inasmuch as their belief in their face recognition capabilities differs from what they actually are, the patients indulge in self-deception.

However, while covert recognition in prosopagnosia is conceivable in terms of self-deception, it clearly differs from dynamically-driven self-deception in everyday life in that, unlike the latter, the former is not associated with unrealistic dreams and fantasies which help people overcome difficulties or inflate self-esteem, and it does not play a role in purposeful blocking of overt recognition (see Greenwald, 1988; Sackeim, 1988; Solomon, 1993). On the contrary, it involves lack of awareness of some cognitive capabilities which are still functioning.

The case of prosopagnosia therefore suggests a reconceptualization of self-deception as a complex concept which entails a variety of dissociations that differ from each other in terms of etiology and dynamics. In everyday life and in psychopathological cases, self-deception in terms of denial may indeed serve as a defense mechanism which alleviates anxiety and preserves ego integrity. However, in some neurological cases it is rightly considered a functional dissociation, void of dynamic attributes, between a given modular system and the general cognitive system.[2]

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[2]This idea is consistent with recent views of self-deception as a multifaceted phenomenon which may be dichotomized in terms of ignorance-based versus belief-based, private versus public, implicit versus explicit, and motivated versus process-based (see Bornstein, 1997; Krebs, Ward, and Racine, 1997). Similarly to the model herewith proposed, the last dichotomy suggests a distinction between self-deception arising from motivated self-protective mental activities, and self-deception inherent in the human information processing apparatus.

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