

NEGATIVE EMOTIONS AND ANOSOGNOSIA

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ABSTRACT

Patients with anosognosia fail to acknowledge, or feel distressed by, their disability. Given the recent suggestion that right (frontal) systems are selectively involved in negative emotions, it might be claimed that anosognosia results from a disruption in negative emotions. This is not consistent with the finding that some anosognosic patients exhibit substantial fluctuations in emotion, including the experience of negative emotions such as sadness. The present study investigates a patient (IW) with a right convexity lesion and anosognosia. He reported being frequently overcome by powerful emotions, especially sadness. IW was assessed on a self-report emotion questionnaire, where his reports were typically of *higher* levels of emotion than the control group. He was also assessed on the more indirect measure of Affective Story Recall. Here his pattern of emotional experience was similar to that of two control groups, one of which consisted of non-anosognosic patients with hemiparesis. His performance on Story Recall was notable in that he directed his emotions to a different 'object' to that of controls (other vs. self, respectively). These findings are not consistent with any claim that anosognosia results from an absence of negative emotions.

Key words: Anosognosia, positive emotion, negative emotions, affect

INTRODUCTION

Denial of deficit (anosognosia) is a well-described disorder, encompassing a range of unusual phenomena (see Bisiach and Geminiani, 1991; Feinberg, 1997; Gainotti, 1972; McGlynn and Schacter, 1989, Prigatano and Schacter, 1991 for review). However, until recently, the disorder was often regarded as little more than "an enigmatic clinical curiosity" (Ramachandran, 1996, p.124). In its extreme form the disorder presents as a manifest denial of paralysis, despite clear evidence to the contrary, where denial occurs even after explicit demonstration of the deficit by the examiner. In other circumstances, perhaps to be regarded as milder forms (anosodiaphoria) the patient may become aware of their hemiparesis, but deny that it causes them any functional disability (c.f. House and Hodges, 1988).

In some cases the denial of deficit is held with such strong conviction that the false beliefs persist in the face of all contradictory evidence and logical argument, even if this results in strange beliefs or perceptual experiences (Bisiach et al., 1986; Halligan et al., 1995; Ramachandran and Blakeslee, 1998; Weinstein and Kahn, 1955). It is clear that anosognosia can present in a number of ways (Marcel et al., 2004), and this may be reflected in the diversity of explanation for the phenomenon. Some recent accounts stress the way in which predicted or willed movement might be at variance with the visual and somatosensory consequences of the action itself (Blakemore et al., 2002; Daprati et al., 2000; Frith et al., 2000; Heilman et al., 1998; see also Fink et al., 1999; McGlynn and Schacter, 1989; Sirigu et al., 1999). Accounts of this type almost certainly have a role to play in explaining the nature

of the disorder. However, there are several reasons why such accounts are probably inadequate as a sole explanation (Marcel et al., 2004). Thus, while mismatches between intended actions and outcomes "may be necessary for anosognosia they do not seem to be sufficient" (Frith et al., 2000, p.1782).

Another potentially fruitful proposal focuses on issues of motivation and emotion. There are some theoretically-driven grounds for this idea – for example the recent focus on the role of secondary somato-sensory cortex, in the right-hemisphere, in the higher aspects of homeostasis, somato-sensory representation, and emotion regulation (Damasio, 1994 pp. 62-69; 1999 pp. 209-213; 1996). This argument is supported by a well-established literature suggesting a central role for the right hemisphere in the perception and expression of emotion (e.g. Borod, 2000). For example, there are well-described effects of right-sided intra-carotid amobarbital, which produces anosognosia in a high proportion of cases (Brier et al., 1995; Lu et al., 1997; Meador et al., 2000), and has long been known to differ from the more emotionally appropriate outcomes that follow from left-sided amobarbital (e.g. Gainotti, 1972, see Feinberg, 1997 for review).

There is also a long-standing clinical literature on patients with right-sided brain lesions, showing that patients with anosognosia often do not show the negative emotional responses to their paresis that one might expect. Most notably, they have fewer so-called 'catastrophic' reactions (episodes of tearfulness and emotional breakdown) that are more frequently seen in patients with left hemisphere lesions (e.g. Fedoroff et al., 1992; House et al., 1990; Gainotti, 1972; 1997; Jorge and Robinson, 2002). Also, anosognosics are often not merely *unaware* of their deficits, but are sometimes

unnecessarily optimistic about their medical condition (i.e. 'euphoric-maniacal', Gainotti, 1997), and may also over-emphasize their abilities with the paretic limb (e.g. Ramachandran and Blakslee, 1998, pp.138-139).

The absence of a negative attitude towards impairment in patients with right-sided lesions, together with reports of low mood after left-sided lesions (Fedoroff et al., 1992; Gainotti, 1972; Jorge and Robinson, 2002) has led to Davidson et al.'s (e.g. Davidson, 2001; Davidson and Irwin, 1999) suggestion of a right frontal system involved in negative (withdrawal-related) emotional states, with left frontal regions associated with positive (approach-related) emotion¹. Thus, Davidson et al. have suggested that depression might result from disruption of a (left-sided) positive emotion system (e.g. Davidson and Irwin, 1999, p. 13). On this argument anosognosia would result from a disruption of negative emotion systems, leaving the patient with only a (left-sided) positive emotion system.

However, there are several reasons to doubt this possible link between an absence of negative emotions and anosognosia. For example, a disruption of negative emotions would explain only the absence of emotion in relation to paresis, not why the patient might actively deny their paresis and explicit evidence thereof (Marcel et al., 2004). It has also been pointed out (see Gainotti, 1997 for review) that the low mood seen in patients with left-sided lesions is likely to result from an emotionally-appropriate response to their substantial levels of disability – which typically involves hemiparesis and non-fluent aphasia. A further line of argument is that there are some circumstances when right-sided lesions produce an *increase*, rather than a disruption, in negative emotion. For example, the occasional finding of explicit dislike or obsessional hatred of the paretic limb ('misoplegia', Critchley, 1974) seen after right-sided lesions, which is discordant with a loss-of-negative-emotions account. Finally, there have been reports of frank depression after right convexity lesions in patients who were also anosognosic (e.g. Kaplan-Solms and Solms, 2000; Starkstein et al., 1990; see also Turnbull et al., 2002). Thus, many hold the position that the data

are more consistent with "a general dominance of the right hemisphere for emotional behavior than with the alternative hypothesis assuming a different specialization of the right and left hemispheres for opposite aspects of... mood" (Gainotti, 1997, p. 692).

One class of evidence which bears on this question, but is little cited, are the fluctuations in emotion, and awareness of deficit, seen in some anosognosic patients. For example, transient recovery of awareness after caloric irrigation has been reported by various authors (Cappa et al., 1987; Ramachandran, 1994; 1996; Rode et al., 1998; Vallar et al., 1990). It is of interest that, at least in some of these cases, such patients often had a selective failure to recall their, earlier acknowledged, paresis when they had returned to their anosognosic state. A further type of fluctuation was noted by Moss and Turnbull (1996), who described a patient who shifted between a state of anosognosic denial and misoplegia (i.e. from unawareness to obsessive hatred). Finally, Kaplan-Solms and Solms (2000, see also Turnbull et al., 2002) reported patients who experienced transient awareness of their deficit, including "sudden moments of tearfulness and pre-tearfulness" (p. 166) which appeared to be preceded by themes of 'loss' – even if the cause of such loss was apparently unrelated to the hemiparesis. Similar examples have been reported by Ross and Rush (1981), and Starkstein et al. (1988). This suggests that negative emotions may be intact in at least some instances of anosognosia. However, we note also that awareness of hemiplegia may *appear* to vary, depending on the nature of the question asked of the patient (see Marcel et al., 2004), so that it is important to base examples of fluctuations in emotion on the same class of question asked at different times.

The fluctuations in emotion seen in these patients suggests that they might show the full range of emotional experience appropriate to their neurological deficits, including anger and sadness. This runs contrary to any claim (e.g. Davidson, 2001) for a right hemisphere system specialized for negative emotions, and a role for loss of negative emotions in anosognosia. The present study investigates an anosognosic patient (IW) on direct and indirect measures of emotion, especially focusing on three classes of negative emotion.

CASE REPORT

IW was a 70-year-old, right-handed, man, who had long been regarded as an upstanding member of the community, and had spent his life in various forms of public service (Royal Air Force, Police Service, and political office). He was married, with several children. In his younger years he had also been an enthusiastic sportsman.

¹The question of exactly how to define a 'negative' emotion is a complex one. Some authors (e.g. Davidson) classify all experiences that appear to be aversive in a unitary 'negative' category. However, recent work separating independent emotions suggest that there may be several distinct classes of negative emotion, with fear, anger and sadness (and perhaps others) as likely candidates (e.g. Calder et al., 2001; Damasio et al., 2000; Le Doux, 1996; Panksepp, 1998). All such states appear to result from 'aversive' events or potential events; for example, anger with frustration of goal-directed action; fear with risk to bodily harm; and sadness with separation and loss (e.g. Panksepp, 1998, p.52). A second line of evidence that supports the claim that these emotions have a negative valence is that the core anatomy of the purported anger, fear and sadness systems (such as the amygdala, hypothalamus, and dorsal peri-aqueductal gray matter) appear to have their substrate in regions that produce aversive responses when electrically stimulated (Bandler and Shipley, 1994; Depaulis and Bandler, 1991; Panksepp, 1998).

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