

RECONCILING PSYCHODYNAMIC AND NEUROLOGICAL
PERSPECTIVES ON DENIAL

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Turnbull and Solms (2007, this issue) say that their review should not be construed as arguing that “Freud was right”. However, they argue for a restoration of Freudian psychology to the scientific pantheon because it turns out that a number of his specific hypotheses about the global architecture of the mind have a fruitful application to intriguing results in modern neuropsychology. We would argue that it is useful in this context to separate two quite different aspects of Freudian theory. The first is the framework that Freudian theory provides for integrating motivation and emotion into otherwise desiccated cognitive theories. In this sense we can agree that modern neuropsychology will benefit from a reconsideration of Freud. But the second is the specific content of Freudian theory. There seems no reason to believe that this is any more acceptable to scientific psychology today than it was fifty years ago and for Freud this would represent a failure that the former success could not come close to assuaging.

It is our view that Freud was in fact the first cognitive psychologist. We mean this in the sense that the burden of explanation in Freudian theory was laid upon mental mechanisms. Of course Freud took this framework further than most contemporary psychologists have tended to do, by attempting to build motivation and emotion into the key mechanisms of the mind from the beginning. Piaget, in contrast, had largely left motivation and emotion out of his account of child mental development, and the subsequent school of cognitive psychology pretty much stuck to that path. For us it is little wonder that the general framework of Freudian psychology will undergo something of a renaissance because it provides a model of how cognitive psychology can address issues surrounding motivation and emotion. We will now illustrate the need for this move with respect to a consideration of denial in neuropsychology.

Turnbull and Solms (2007, this issue) suggest that anosognosia involves an “impaired ability to manage powerful negative emotions”, an impairment stemming from damage to right-lateralized emotion-regulation systems. On this

view, anosognosic patients fail to acknowledge their deficits because to do so would entail levels of aversive emotion that – because of their brain damage – they could not tolerate. We find this type of explanation very congenial, but would like to offer a slightly different gloss on it, via consideration of Ramachandran’s ideas about hemispheric specialization (e.g., Ramachandran and Blakeslee, 1998; see McKay et al., 2005, for a critical review). Ramachandran has suggested that the neurological locus of psychological defence is in the brain’s left hemisphere, and has contended that the left-hemispheric mechanisms that underpin defensive processes are opposed by a complementary right hemispheric mechanism, a “discrepancy detector”. Operating normally, this mechanism prevents everyday defences from escalating into delusion. If this mechanism is damaged, however, patients will be left without constraints on their capacities for psychological defence – permitting the flagrant denials observed in anosognosia. Like Turnbull and Solms’ account, Ramachandran’s approach incorporates the deficit and motivational perspectives (see McKay et al., 2005) as different explanatory levels of a single system. Both explanations are at once neurological and motivational, and posit that anosognosia is simultaneously a low-level neurological impairment and a high-level psychological palliative. The difference is that whereas Turnbull and Solms (2007, this issue) characterise anosognosia as involving an impaired ability to manage powerful negative emotions, Ramachandran’s suggestion seems to indicate the opposite – that anosognosic patients are, as a consequence of organic insult, over-endowed with this ability.

The beauty of neuropsychodynamic accounts is that they neatly defuse an obvious objection to psychodynamic conceptions, namely, that there is a striking left-biased asymmetry in anosognosic expression. If anosognosia results from an abnormality in the brain’s management of negative emotions, an abnormality stemming from damage to right-hemispheric mechanisms, then damage to the left hemisphere should not lead to anosognosia. But what of other criticisms that have been levelled at psychodynamic accounts of anosognosia? For

example, Stone and Young (1997) observed that anosognosia for left-hemiplegia is often seen in association with left-unilateral neglect, and that temporary remission of both symptoms can be achieved, remarkably enough, via caloric vestibular stimulation (e.g., Cappa et al., 1987). However, Ramachandran and Blakeslee (1998) argue that anosognosia is not merely a consequence of neglect, as neglect and anosognosia are double-dissociated, and anosognosic denial typically persists even when the patient's attention is drawn to their paralysis (thus overriding their neglect). Moreover, as Ramachandran and Blakeslee (1998) point out, connections from the vestibular nerve project to the vestibular cortex in the right parietal lobe as well as to other areas of the right hemisphere. Given Ramachandran's suggestion that the discrepancy detector mechanism is located in a region of the right hemisphere innervated by the right parietal lobe, it seems conceivable that caloric vestibular stimulation may arouse and re-activate the very right-hemispheric mechanisms whose putative disruption enables anosognosia.

While these ideas are speculative, their importance lies in the fact that they are formulated within a framework that is familiar to cognitive neuroscience and that would, we submit, have been congenial to Freud, in his early days at least. Moreover, these ideas facilitate testable predictions. If it is true that everyday self-deception and anosognosic delusion are underpinned by a unitary, defensive, "left-hemisphere-implemented" process, a process ordinarily held in check by a right-hemispheric discrepancy detector, then caloric activation of the right hemisphere should occasion

a particularly sober, realistic appraisal of facts about the self and the world and might therefore attenuate non-hemiplegic forms of anosognosia (e.g., Anton's syndrome), as well as non-anosognosic forms of delusion (e.g., persecutory delusions) and ordinary, "garden-variety" self-deception. These are all testable claims.

The empirical investigations reported in the Turnbull and Solms paper (2007, this issue) support the psychodynamic notion that motives are important causal forces where confabulations and delusions are concerned. Any theoretical attempt to explain such claims must therefore incorporate both motivational and neuropsychological factors. The type of theoretical framework envisaged by Freud, with its accent on mental mechanisms, seems just the kind of framework conducive to modern cognitive neuroscience.

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