

AWARENESS, DESIRE, AND FALSE BELIEFS: FREUD IN THE LIGHT OF MODERN NEUROPSYCHOLOGY

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INTRODUCTION

Psychoanalysis has had a turbulent and complex relationship with neuropsychology for the century in which the two fields have existed – largely side by side. Some within the neuroscientific community have found much of value in Freudian ideas – Paul Schilder springs to mind as an early example, with Eric Kandel as the most prominent recent advocate (Kandel, 1999, 2005). However, for most neuropsychologists, indeed for most *scientists*, the obvious response to the mention of psychoanalysis has been one of blanket rejection. The central objection has been the issue of empirical evidence. While many of Freud's ideas appear to have derived from his pre-analytic work in neuroscience (e.g., Freud, 1891, for more on this topic see Sulloway, 1979; Solms and Saling, 1986), the clinicians who dominated the *practice* of psychoanalysis for a century have been, by-and-large, remiss in even attempting to design and conduct well-formulated scientific experiments. Where such research has taken place, it has often been aimed at the methodologically-fuzzy domain of treatment outcome (Galatzer-Levy et al., 2000; Anderson and Lambert, 1995; Crits-Christoph et al., 1988; Fonagy and Moran, 1991; Fonagy and Target, 1996) rather than addressing core theoretical concerns. As Kandel incisively put it: “although psychoanalysis has historically been scientific in its aim, it has rarely been scientific in its methods” (Kandel, 1999, p. 506).

However, failure to adequately test a theory does not imply that it is without foundation, and it has long been clear that there is much merit in Freud's broad-brushstroke sketch of the functional organization of the mental apparatus. Thus, the same Kandel who feels skeptical about the *methods* of psychoanalysis, also describes it as “still the most coherent and intellectually satisfying view of the mind” (Kandel, 1999, p. 505). Kandel's optimism about Freudian ideas is based, at least in part, on the fact that modern neuropsychology is rediscovering some of Freud's most fundamental insights into the workings of the human mental apparatus.

In this brief review we survey a range of findings support some of the central claims of

classical psychoanalysis. Some of the findings are well-known – others less so, and have not yet received general attention in mainstream neuropsychology. This survey should certainly not be read as endorsing a “Freud was right” view – though this appears to be the central message distilled by journalists (Guterl, 2002; Lakotta, 2005). Rather, it seems that Freudian *hypotheses* about the global architecture of the mind now seem ripe for the more detailed, and more sophisticated, treatment that modern neuropsychology can offer.

COGNITION OUTSIDE AWARENESS

The best known aspect of psychoanalytic theory is the claim advanced by Freud – with several scientific precursors – that most mental activity occurs outside of conscious awareness. Famously, his scientific contemporaries strongly opposed this notion (Freud, 1915), such that subjective awareness and mental activity were generally considered synonymous. Thereafter, for much of the 20th century, interest in subjective experience was regarded as a topic inappropriate for a scientific psychology – much less the idea that conscious awareness could characterise but a small fraction of mental life, and that our experience of volition might be illusory. However, the last several decades have seen a wide range of remarkable scientific findings bearing on these questions, and today one would be hard pressed to find a cognitive neuroscientist who did not consider mental activity outside of conscious awareness to be a well-established fact (see Libet et al., 1967, 1983; Schacter, 1992, 1994).

How else are we to understand phenomena such as blindsight (Weiskrantz, 1986), implicit awareness in neglect (Marshall and Halligan, 1988), non-declarative learning even in dense amnesia (e.g., Knowlton et al., 1994, 1996; Turnbull and Evans, 2006a), or the remarkable “split-brain” phenomena (Gazzaniga, 1995)? Examples abound, though they differ somewhat in their exact bearing on the question of unconscious mental life. Nevertheless, in each instance it is clear that mental processes, often of a sophisticated nature, can occur without

any subjective experience of the task being accomplished. Indeed, it has been claimed that the vast majority of motivated mental acts occur outside of conscious awareness (Bargh and Chartrand, 1999 cite a figure of some 95%; see Solms, 1997a for a review of Freud's position on this topic).

However, a closer look at these findings reveals that, while they clearly support Freud's *general* claim of ubiquitous unconscious mental activity, they do not constitute support for some *specific* aspects of his model. The principal distinction is that the findings cited above confirm the existence of *cognitive* processing outside of conscious awareness, whereas Freud claimed specifically that *unconscious motivational* and *emotional* factors shape conscious mental life.

During the second half of the 20th century, when almost all the great advances in neuropsychology were in the cognitive domain, it should have come as no surprise that "pro-Freudian" findings – of the sort cited above – were primarily cognitive in nature (Turnbull, 2001). However, during the last dozen years or so there has been a remarkable increase in interest in the biological basis of emotion – and the associated rise of an "affective" neuroscience to rival its cognitive cousin (Panksepp, 1998; LeDoux, 1998; Rolls, 1995; Damasio, 1994, 1999). We are therefore now better placed to evaluate whether there is more directly pertinent neuropsychological support for specifically-Freudian claims (Kandel, 1999).

BASIC EMOTION SYSTEMS

Freud argued that the unconscious mind was dominated by unconstrained instinctual mechanisms – which in turn revealed the fundamentally *biological* nature of human mental life. Thus humans, no less than other living creatures, are animals: driven by evolutionarily conserved drives. To Freud's Victorian contemporaries the prominent role he gave to base biological urges appeared downright scandalous, especially when the ideas had such an explicit Darwinian link to reproductive fitness. The moral outrage, of course, waned during the decades of the 20th century. Freudian ideas concerning the importance of biological drives were, however, also challenged by others, not least the behaviorist tradition with its opposition to the very notion of innate dispositions, and its denial of emotion [Skinner (1953) famously described emotions as a 'fiction']. Now that so much of the ideological baggage of behaviorism has been discarded, and with the growing awareness that its notions of 'reward' and 'punishment' are simply emotion repackaged (Berridge, 2003; Hobel, 1997; Schultz, 1998, 2001), Freud's view that human behaviour is implicitly driven by hard-wired motivation systems is finally receiving the scientific attention it deserves.

There is now overwhelming evidence for the importance of instinctual drives and basic emotions in mental life (see below), and we have unprecedented access to the neurobiological base of these phenomena – to a level of accuracy that Freud could only have imagined, though he was clear that such a day would come:

"Biology is truly a land of unlimited possibilities. We may expect it to give us the most surprising information and we cannot guess what answers it will return in a few dozen years ... They may be of a kind which will blow away the whole of our artificial structure of hypotheses." (Freud, 1920, p. 60)

Recent findings unequivocally *confirm* Freud's claim that mental activity is fundamentally grounded in a set of motivation and emotion systems, which are both phylogenetically ancient and dominant in early mental development (Panksepp, 1998; LeDoux, 1994; Pfaff, 1999). Of course, with greater knowledge has come a vast increase in the level of detail. Where Freud could only speculate about broad systems associated with sexuality and aggression, we now have a growing understanding of an architecture for multiple independent emotion systems, running to perhaps half a dozen in number (e.g., Berridge, 2003; Calder et al., 2001; Davidson, 2001; Panksepp, 1998; Phan et al., 2002): We also now know that these systems are largely anatomically and chemically independent, widely distributed across several areas of archicortex, deeply embedded in distinct subcortical structures broadly linked to the classical "limbic" system (again, see Calder et al., 2001; Panksepp, 1998; Phan et al., 2002), and tightly linked to highly specific key regions of the upper brain-stem (Panksepp, 1998; see Watt, 2000 for review).

Indeed, the instinctual mechanisms that govern human motivation may be even more primitive than Freud imagined. We appear to share the basic emotion systems that determine our core values not only with our nearest *primate* relatives, but also with *all mammals*, and to a lesser extent even with more "primitive" species such as birds (Panksepp, 1998). These primitive systems are also clearly of great functional importance and complexity. Indeed, we might well regard the basic emotional landscape as being of roughly the same "scale" and "scope" as cortical cognitive systems – though, thus far, emotion systems have been the focus of far less neuropsychological investigation.

Yet we should at this stage still retain a coolness of judgement about the importance of these findings for psychoanalysis. Certainly, there is powerful support for the claim that basic instinctual emotion systems represent an important component of the human mind. But these are not the most *fundamental* of Freud's original

conceptions. Rather, the central psychoanalytic claim is that emotion systems (and the drives that govern them) might *distort* cognitive representations of reality, by hijacking executive resources (through so-called defences). Thus, Freud argued, humans are often “irrational” (holding patently false beliefs) because the consequences of such beliefs are subjectively advantageous.

There are several examples of contemporary neuropsychological evidence that appears to support this claim in modern neuropsychology. We provide a brief review of some instances.

CONFABULATION AND ANOSOGNOSIA

One striking example of apparently irrational beliefs is frank confabulation after deep frontal lesions (Schnider, 2003). As one might expect after several decades of a neuropsychology focused primarily on cognition, most current accounts of confabulation are purely cognitive in nature, focussing especially on the importance of executive deficits (e.g., Kopelman, 1987, 1995; Kapur and Coughlan, 1980; Luria, 1976; Papagno and Baddeley, 1997; Johnson et al., 1997; Moscovitch and Melo, 1997). However, the dysexecutive account as a sole explanation of the disorder is open to criticism (e.g., Fotopoulou et al., 2004; Turnbull et al., 2004a, 2004b) for a range of reasons. Notably, most patients with dysexecutive disorders do not confabulate, and the executive impairment in confabulators is often relatively minor. Also of note is the highly selective nature of many confabulations, with small “islands” of strongly-held delusional beliefs, in otherwise entirely rational patients (e.g., Burgess and McNeil, 1999; Conway and Tacchi, 1996; Villiers et al., 1996). We contend that, while there is much merit in a dysexecutive account, it is almost certainly a necessary but not sufficient condition for confabulation (e.g., Fotopoulou et al., 2004; Turnbull et al., 2004a, 2004b).

There have long been reports that the content of confabulations might be influenced by their affective valence (e.g., Burgess and McNeil, 1999; Mercer et al., 1977; Talland, 1961), including the suggestion that confabulations might modify the patient’s subjective state – altering the patient’s self-representation in an improved or even grandiose way (Conway and Tacchi, 1996; Downes and Mayes, 1995; Paterson and Zangwill, 1944; Prigatano et al., 1996; Villiers et al., 1996). This proposal was however woefully under-investigated – even though it is also consistent with what is now known about the anatomical basis of the disorder, notably its association with damage to key emotion-related structures in the medial frontal and anterior limbic areas (e.g., De Luca and Cicerone, 1991; Fisher et al., 1995; Schnider, 2003; Schnider et al., 1996).

In our initial attempts to systematically investigate this issue, we have recently demonstrated that the false belief scenarios of confabulatory patients have a clear *positive affective bias*, tending to misrepresent the patient’s circumstances as more pleasant than their actual situation (Fotopoulou et al., 2004; Turnbull et al., 2004a, 2004b), with the patients apparently believing that they are engaged in important business activities, or leisure pursuits, or that their hospital ward has become their home (Fotopoulou et al., 2004; Turnbull et al., 2004b). An especially interesting aspect of this research is the finding that confabulations tend to occur when the patients are in a low mood state (Turnbull et al., 2004b); where one plausible interpretation is that confabulations occur *in order* to improve the patient’s mood, serving a role in emotion-regulation, and acting as a form of “defence”.

A similar shift away from purely cognitive, to more emotion-oriented explanations can also be demonstrated by attempts to understand unawareness of deficit, or anosognosia: with traditional explanations (see McGlynn and Schacter, 1989 for review) linked to hemi-spatial neglect (Bisiach et al., 1986; Feinberg, 1997) or inconsistencies between willed action and the sensory consequences of movement (Blakemore et al., 2002; Daprati et al., 2000). However, after a long history of anedoctal antecedents (Feinberg, 2001; Weinstein and Kahn, 1955), a flurry of new experimental findings have been reported that suggest a central role for emotion in generating this class of false belief.

It is now evident that the emotional experience of patients with anosognosia has the same range as that of controls (Turnbull et al., 2002, 2005), but that they have impaired ability to manage powerful negative emotions. Strikingly, the manipulation of psychological variables strongly modifies the extent to which the disability is acknowledged (Aglioti et al., 1996; Bottini et al., 2002; Marcel et al., 2004; Ramachandran, 1994, 1996). Most notably, when the patient is offered a world-view in which awareness of the left limb does *not* imply that they are permanently disabled (e.g. that the paralysis is a temporary consequence of a neurological investigation), they are willing to acknowledge that the limb cannot move. On this argument, anosognosic patients have no impairment in primary emotion systems, but a disruption in the regulation or acknowledgement of emotions (or at least one aspect of this process).

Those who have long opposed “psychodynamic” accounts of anosognosia have quite naturally asked why anosognosia should be so clearly lateralized – given that patients with left-sided lesions (and right-sided paralysis) seldom show denial of deficit (e.g., Bisiach and Geminani, 1991, pp. 24-28). A plausible answer would be that the emotion-regulation systems are right-lateralised (Turnbull et

al., 2002, 2005), a claim that is consistent with many findings on the lateral organization of emotion systems (e.g. see Borod, 2000 for review). Thus, these patients may deny their deficit because they have difficulty tolerating aversive emotional states, of which awareness of paralysis would be an extremely salient instance. On this account, however, the deficit should extend (albeit to a lesser extent) to other classes of negative emotion. There is some data to support this claim – most notably that such patients are often overwhelmed by sadness when discussing separation and loss, even in discussions unrelated to their bodies or neurological disability (e.g., Kaplan-Solms and Solms, 2000; Turnbull et al., 2002, 2005).

Viewed from this perspective, confabulatory and anosognosic patients may suffer no more than an extreme version of the tendency we *all* have to down-play our limitations, as we protect ourselves from events with aversive consequences. The effects are, of course, greatly magnified as a result of their brain lesions – which may well be disrupting the cortical control of a range of anatomically distinct emotion regulation systems. This proposal makes a series of very clear claims – all yet to be adequately tested. For example, if the changes in the emotional lives of anosognosic patients are restricted to the domain of hemiparesis, or are narrowly related to disturbances of body-schema, then it is clear that grand accounts of emotion-regulation in relation to anosognosia are an inadequate hypothesis. Similarly, an emotional bias should be demonstrable (and consistent) in several aspects of the thinking of confabulatory patients, not merely in the areas which produce their most florid false beliefs (e.g., Burgess and McNeil, 1999).

PSYCHIATRIC DISORDERS

We suggested above that there might be examples *other* than those from neurological patients that would support the claim that emotion systems play a key role in unverified belief states. Perhaps the clearest instances are the many varieties of false belief that form the basis of psychotic disorders. It is interesting to observe that the pharmacologies by which organic psychiatry exerts its therapeutic influence target precisely the same chemical systems that were mentioned earlier as core emotion systems. Let us take a well-known example:

Dopamine pathways, which travel from the upper brain-stem to a range of ventral and mesial frontal forebrain sites, are the substrate for a key emotion system (Panksepp, 1985; Robbins and Everitt, 1992). Various referred to over the years as a “reward” (Schultz, 2001), a “preparation” (Hobel, 1997), or a “seeking” system (Ikemoto and Panksepp, 1999), this chemistry appears to

consistently activate ventromesial frontal structures during tasks involving reward and punishment (Dias et al., 1996; O’Doherty et al., 2001; Schultz, 2001); it appears especially apt to motivate us to investigate the environment, search for rewards (e.g., Robbins and Everitt, 1992), and to construct causal relationships between events in the perceived world (Schultz, 2001).

What then of a quite different set of findings relating to dopamine? It was known since Arvid Carlsson’s work in the 1960’s, and especially since the 1970’s (see Snyder, 1976), that pharmacological management of the positive symptoms of schizophrenia (the delusions and hallucinations) occurs by targeting one or more of these dopamine systems in the brain. This “dopamine” theory of schizophrenia remains (through a range of modifications) by far the most robust account of the neurochemical basis of the disorder: originally emphasizing the simple over-activation of the dopamine system, but more recently uncovering roles for other members of the D₂ dopamine family, and revealing the extent to which cortical mechanisms regulate these forebrain dopamine systems (see Carlsson and Carlsson, 1990; Egan and Weinberger, 1997; Moore et al., 1999 for review).

It certainly appears that changes to this emotion system (whether by over-activation or poor regulation) lies at the heart of the irrational beliefs held by psychotic patients. Of course, the precise details of *how* these modified emotions produce delusions remain poorly specified – though there have been findings which suggest that emotion-related variables are important in the disorder (e.g., Birchwood, 2003; Brown et al., 1972; Bentall et al., 1991; Vaughn and Leff, 1976; Kapur, 2003). It seems likely that any complete account of psychosis will include a central role for emotion (e.g., Kapur, 2003; Evans et al., 2005; Turnbull et al., 2006b) – not merely as a consequence of the disorder, but in a *causal* role.

DREAMS

Another striking example of the re-discovered role of emotion and unverified beliefs is in the field of sleep and dream research. When REM sleep and its near-perfect correlation with dreaming were discovered in the 1950s (Aserinsky and Kleitman, 1953; Dement and Kleitman 1957a, 1957b), and when REM sleep’s cholinergic brainstem mechanism was laid bare in the 1970s (Hobson and McCarley, 1977; Hobson et al., 1975), Freud’s (1900) dream theory appeared to lose all scientific credibility. However, more recent research has revealed that dreaming and REM sleep are doubly dissociable states, controlled by distinct (but interactive) brain mechanisms (Solms, 1997b). Dreaming turns out to be generated by a

network of forebrain structures centered principally around the same ascending dopamine systems discussed above in relation to psychosis (Braun et al., 1997, Maquet et al., 1996, Nofzinger et al., 1997; Solms, 1997b, 2000).

Most intriguing is the observation that dreaming stops completely when fibers in the ventromesial frontal lobes are severed; a symptom that coincides with a general reduction in motivated behavior (Solms, 1997b). The lesion producing this syndrome is the same as that which was deliberately produced in pre-frontal leucotomy, which was of course replaced in the 1960s by drugs that dampened activity in the same dopaminergic pathways discussed above in relation to schizophrenia.

There is therefore robust support for the claim that the system that mediates powerful positive emotions (Berridge, 2003; Panksepp, 1998; Schultz, 1998, 2001), and is *also* centrally implicated in hallucinations and delusions (Silbersweig and Stern, 1996; Silbersweig et al., 1995), is at the heart of the “false belief” states that generate the dream process (Solms, 1997b, 2000, 2002). If this hypothesis is confirmed, then the ‘wish-fulfillment’ theory of dreams could yet again set the agenda for mainstream dream science (Shevrin and Eiser, 2000). Either way, few neuroscientists today would still claim, as they once did, that dreams are motivationally neutral (Hobson, 1988), and still fewer that they have “no primary ideational, volitional, or emotional content” (Hobson and McCarley 1977, p. 1347).

This opens the fascinating possibility that emotion may play a generative role in *all* classes of false belief: from dreaming in the neurologically-healthy, to changes seen after focal brain lesions, and also encompassing the pharmacologically-sensitive disorders that are central to the several psychiatric conditions with delusional features. The empirical foundations for such a grand theory remain limited, but it offers the prospect of a unification of findings across wide domains.

CLAIM AND COUNTER-CLAIM

Alongside the suggestion that several recent findings appear to be consistent with Freud’s claims, there are two other “big picture” issues worth noting. Firstly, it has always been clear that neuropsychological research would be useful in testing core psychoanalytic ideas. However, it is also increasingly clear that there are ways in which analytic ideas can inform *neuropsychology* (Kandel, 1999, 2005) – of which the emotion-related accounts of false belief states discussed above would be prime examples. The neuropsychological community has traditionally defaulted to cognitive-based accounts for these states (Fotopoulou et al.,

2004; Turnbull et al., 2002, 2004b, 2005), at least in part because the training of the scientists concerned has been primarily cognitive. However, there are some striking, and laudable, examples of change in this approach (e.g., Coltheart, 2000; Conway and Tacchi, 1996; McKay et al., 2005; Ramachandran, 1994, 1996), and we anticipate that the next decade will see growing numbers of emotion-related accounts of psychological disorders, as neuropsychologists come to recognize the role of the newly-acknowledged emotion systems in explaining such phenomena.

The second important issue relates to the empirical status of psychoanalysis in relation to these new findings. Psychoanalysis has a long history of generating sophisticated hypotheses, which are often difficult to understand, largely by virtue of their obscure language – hypotheses which at times appear frankly untestable. However, it is clear that the new generation of emotion-related hypotheses are not of this type. This is not to say that each of these ideas will be *easy* to test. Many of the hypotheses are not easy to address directly, at least in part because the specifics of the emotion systems that underpin them remain poorly understood. The central point, however, is that there is a clear desire to frame questions in a language that can be readily comprehended by the broader psychological community, to tackle problems that are ‘bite-sized’ and amenable to empirical investigation in relatively modest experiments. The goal is to plainly frame these questions in ways designed to stimulate further investigation of the core issues – rather than the more vague goal of testing grand Freudian ideas.

Any science worth its salt should be able to generate hypotheses that are falsifiable. In this spirit, we propose a series of claims in relation to emotion and false beliefs which – if disproved – would seriously undermine core psychoanalytic assumptions. Firstly, we propose that emotion systems play a central, causal role in false beliefs. Thus, the “magnitude” of the false belief should be somehow “proportional” to the magnitude of the emotion required to generate and sustain it, and systematic modification of emotion systems (by psychological or pharmacological means) should demonstrably induce, titrate, or extinguish the unverified beliefs. In addition, it should be possible to show that the unverified beliefs observed in “pathological” states (in psychiatric and neurological disease) are not different in kind (only in magnitude) to distortions in beliefs seen in the neurologically and psychiatrically normal. At present there appears to be wide support for these claims in a range of domains – from confabulation and anosognosia, through dreams, to psychosis.

A key point is that, as we investigate the role of emotion more closely in each of these disorders, the more solid the evidence appears to be: a principle that has correctly been regarded as a touchstone in

separating science from pseudoscience. As Richard Feynman was fond of pointing out, a common property of pseudoscience (he typically used extra-sensory perception as an example) was the fact that the statistical effects *reduced* in size as the methods of investigation improved (Feynman, 1974/1985). In contrast, the investigation of *genuine* causal mechanisms should show increased effect sizes as they are studied with improving tools. We therefore predict that the growing interest in emotion will be accompanied by increasing awareness of the importance of emotion in *all* classes of delusional thinking. Indeed, to turn this argument full circle, we note that the “skeptical” (anti-pseudoscience) movement itself has always recognized that emotions lie at the heart of unverified beliefs, even in the neurologically and psychiatrically normal: such that “Pseudoscience speaks to powerful emotional needs that science often leaves unfulfilled” (Sagan, 1997, p.18). Thus, with some intellectual relish, we suggest not merely that Freudian claims regarding the importance of emotion in false beliefs are *not* pseudoscience, but also that they might be useful in explaining the origins of belief *in* pseudoscience.

CONCLUSION

A century after Freud introduced his many radical and counter-intuitive ideas into psychology, several of his most basic claims appear to stand on surprisingly firm neuropsychological ground. Of course, much of what Freud proposed, on the basis of purely clinical observations, has been refined, enhanced, and surpassed by subsequent knowledge. However, this should come as no surprise. The early intuitions of every fledgling discipline require substantial modification with the passage of time: just as modern molecular biology and genetics have superseded Darwin's insights concerning the broad mechanisms of natural selection. Thus, as we better understand the detail, so we come to appreciate that vague conceptions such as “the unconscious”, “libido”, and “defence”, cannot possibly do justice to the vast complexity of the neuropsychological processes actually entailed by these terms.

But this is not the essential issue. As modern neuropsychology finally begins to tackle the centrally important questions of human psychology that so preoccupied Freud, even as we discover that he was wrong in this respect or that, so we are finding that his first-pass theories still provide us with “the most coherent and intellectually satisfying view” we have of the global structure and functions of the human mind: suggesting novel ways in which we might design experiments and offering fruitful explanations of the outcomes of these studies. It is, in short, “not a matter of proving Freud wrong or right, but of finishing the job” (Guterl, 2002, p.63).

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FREUD AND NEUROPSYCHOLOGY:
COMMENTS RELATED TO ANOSOGNOSIA

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There are many issues discussed in this article about which I would like to comment, but I will focus on the authors' discussion anosognosia. One of this review paper's major hypotheses is that Freud claimed that motivational and emotional factors shape mental life. The authors of this article attempt to explain unawareness of hemiplegia, or what Babinski (1914) called anosognosia (without knowledge of disease), based on Freud's emotional postulate. These authors suggest that patients with anosognosia have "an impaired ability to manage powerful negative emotions." Weinstein and Kahn (1955), in their book *Denial of Illness*, were one of the first groups of investigators to suggest that anosognosia was a psychological means of dealing with the devastating consequences of brain damage. As a physician who developed crushing chest pains with diaphoresis while playing singles tennis and then treated my pain with antacids, I am aware that people deny illness to reduce emotions such as fear and moods such as depression. Although, as Weinstein and Kahn postulated (1955), people deny their hemiplegia for psychological reasons, we will provide evidence, gathered in our laboratory, that partially discredits this emotion hypothesis of anosognosia.

Weinstein and Kahn (1955) noted that many of the reports of patients who demonstrate an anosognosia of hemiplegia were more likely to have right than left hemisphere injury. Weinstein and Kahn's psychological denial hypothesis cannot account for this hemispheric asymmetry and they attempted to explain this asymmetry as a sampling artifact, suggesting that many patients with large left hemisphere lesions have aphasia, and with this aphasia they might not be able to understand questions or "explicitly" deny weakness.

To test Weinstein and Kahn's explanation (1955) of the reported hemispheric asymmetries, we assessed anosognosia in epileptic patients undergoing Wada (selective hemispheric barbiturate anesthesia) testing. Since we did not want aphasia to obfuscate our results, we waited until these patients recovered from their hemispheric anesthesia before we asked them if they had limb weakness. Using this procedure we found that there were hemispheric asymmetries, such that patients

were more likely to be totally unaware of their hemiplegia after their right hemisphere anesthesia than after left hemisphere anesthesia (Gilmore et al., 1992). These results suggest that the hemispheric asymmetry in anosognosia, seen clinically, cannot be related to language dominance and that there might be other factors that might account for this hemispheric asymmetry.

The authors of this paper, about Freud and neuropsychology, acknowledge that there is a hemispheric asymmetry of anosognosia, but claim that these patients might deny their deficit because emotion regulation systems are right lateralized and these patients have difficulty tolerating aversive emotional states. Although there is evidence for the postulate that the right hemisphere might be critical for the mediation of emotions, and especially negative emotions (Please see Heilman et al., 2003, for a review), it is unclear why these authors conclude that damage to the right hemisphere would enhance the experience of negative emotions rather than induce apathy or euphoria. Starting with the work of Goldstein (1949), it has been repeatedly (but not always) reported that it is more likely that patients with left *versus* right hemisphere disease, that includes the frontal lobes, are more likely to have negative emotions, including depression and anxiety (Narushima et al., 2003).

Further evidence against this emotional-denial hypothesis comes from the study we mentioned above (Gilmore et al., 1992). In this Wada study the patients were asked about their hemiparesis after they had recovered their hemispheric function and thus, it would be unclear why they would have to use psychological denial.

In addition to this evidence against the emotion-denial hypothesis, there are also other studies that provide evidence for alternative hypotheses. Because of the word limit imposed by the editors of this journal, we cannot discuss all these hypotheses in detail, as well as the evidence that supports these alternative hypotheses. However, there is evidence that patients might be unaware of their hemiparesis because they have other deficits. For example, it has been shown that some patients have disorders of sensory feedback and this can be related to either sensory denervation (hemianopia,

and/or loss of tactile and proprioceptive feedback (Levine et al., 1991) as well as hemispacial neglect. In addition, asomatognosia or personal neglect might induce anosognosia (Adair et al., 1995). A failure of the patients to attempt to use the contralesional arm (self-test the arm for weakness), an intentional deficit that is often associated with motor neglect, might also be responsible for unawareness of hemiplegia (Gold et al., 1994). Anosognosia is a form of confabulation (Feinberg et al., 1994). Geschwind demonstrated the right hemisphere lesions can not only injure cognitive networks stored in the right hemisphere, but can also induce an interhemispheric disconnection (Geschwind, 1965). A hemispheric disconnection or dissociation where information from the right hemisphere cannot access the language-speech areas of the left hemisphere might induce a verbal confabulation form of anosognosia (Adair et al., 1997).

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FALSE BELIEFS AND MOTIVATED ABNORMAL EMOTIONAL BEHAVIOUR
IN RIGHT BRAIN-DAMAGED PATIENTS

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In their discussion of the contribution that contemporary neuropsychology could give to test the Freud's claim that the emotional system unconsciously distorts the cognitive representations of the reality, Turnbull and Solms (2007, this issue) take shortly into account the role that the right hemisphere could have in this distortion. In particular, they mention the well established prevalence of anosognosia/denial of hemiplegia in patients with right-sided lesions and explain this asymmetry as a consequence of a right hemisphere prevalence in emotion regulation processes. From this point of view, anosognosia/denial of hemiplegia should be viewed as a motivated distortion of reality and its prevalence in right brain-damaged patients (RBDP) could be due to the disruption of right hemisphere structures playing a crucial role in emotion regulation. Since I have recently reviewed (Gainotti, 2005) the clinical and experimental evidence documenting the crucial role of the right hemisphere in emotional processing, I will not return here upon this general problem. I will, rather, focus attention on some specific clinical aspects of RBDP, which could be relevant to the debated issue of how reliably distinguishing between motivated (defensive) and non-defensive forms of unawareness. Most of these clinical phenomena have been mentioned by the authors in various parts of their paper and can be introduced by a short description of the criteria suggested to distinguish defensive from non-defensive forms of unawareness. These criteria include: (a) the resistance of patients to the examiner's claims or acts stressing their impairments and disabilities; (b) the temporal evolution of anosognia/denial of illness; (c) the presence of concomitant unexplained negative, anxious, hostile feelings; (d) the presence of metaphorical or symbolic references to the disease, which are also observed in other threatening life conditions, in patients without brain damage. The patterns of behaviour taken into account in the present commentary on the basis of these criteria had already been considered as typical of RBDP in an old personal article (Gainotti, 1972) and concern in particular: (1) some forms of anosognosia of left-sided

hemiplegia; (2) some abnormal negative attitudes toward the paralysed limbs; (3) other paradoxical attitudes towards the affected limbs, such as their "personification"; (4) some forms of confabulations that could be considered as defences protecting the patient from unacceptable aspects of the reality.

ANOSOGNOSIA OF LEFT-SIDED HEMIPLEGIA

The distinction between forms of anosognosia due to defensive and non-defensive forms of unawareness can be based not only on the obstinate, surprising resistance of some patient to the doctor's claims or acts showing that their limbs are paralysed, but also on the temporal evolution of this unawareness. In these instances, few days after the disease onset, patients begin to forcedly admit the existence of some problems with their paralysed limbs, but attribute them to trivial and unpalatable reasons, such as weariness, injections, arthrosis, etc. During this transitional period, their emotional attitude is characterized by indifference toward disabilities, which are treated with cheerful acceptance, sometimes even jokingly. Only at a later time, the acknowledgment of hemiplegia becomes more full and realistic and is accompanied by a more or less stable depressive reaction.

ABNORMAL NEGATIVE ATTITUDES
OF "ANOSOGNOSIC" PATIENTS

If the patient's apparent unawareness is of defensive nature, we should expect that the unexpressed anxious-depressive feelings of the patient emerge through other patterns of behaviour. Some of these patterns have been reported by Kaplan-Solms and Solms (2000) and by Turnbull et al. (2005). The former have noted that anosognosic patients show frequent breakthroughs of emotions related to themes of separation and loss, whereas the latter reported that their anosognosic patient was frequently overcome by fluctuating unexplained experiences of negative emotions. An other pattern, previously described as "misoplegia" by Critchley (1955) and consisting of melodramatic

or sarcastic expressions of hatred towards the paralysed limbs had been observed only in RBDP in my previous paper (Gainotti, 1972). These expressions often referred to the paralysed limbs in a metaphorical manner, labelling them as a “serpent” or as a “dried branch”, which should be “cut with a scythe and cast away”.

“PERSONIFICATION” OF PARALYSED LIMBS
(AND OF OTHER UNBEARABLE SOURCES
OF ANXIETY)

A further paradoxical attitude towards the paralysed limbs consists of the fact that patients not only focus their attention on these limbs, but also treat them as non belonging to their bodies, labelling them with proper names (‘little John’, ‘Peter’, etc.). What is interesting here is that this pattern of behaviour, which had been described by Critchley (1955) under the name of “personification” of the paralysed limbs, has been observed not only in right brain-damaged patients, but also in patients affected by other life threatening conditions such as cancer.

They are considered as instances of defensive behaviour, allowing the patient to split the source of anxiety from the “self” and to take the distance with respect to it.

CONFABULATIONS OF DENIAL

This behavioural pattern can be defined as the tendency, shown by some patients, either to report actions inconsistent with the limitations imposed by their disease or to deny being in a hospital (a place necessarily associated with a disease condition). The resistance of these confabulations to the arguments raised by the examiner can be exemplified by one of our patients with an extensive right hemisphere lesion, who claimed: -that she was at home and not in a hospital, -that she was in a bed because she was tired and -that the other patients were friends who were visiting her. After a long negotiation, she concluded that

she was on a train which was carrying her at home from the hospital. Confabulations such as the one described in this patient are particularly interesting with respect to the problem of the motivated false beliefs of right brain-damaged patients, because they have been observed by not only in stroke patients, but also in patients with degenerative brain disease (Gainotti, 1975). These kinds of confabulations have been described under the term of “Delusional misidentification and reduplication syndromes” by Feinberg and Keenan (2005), who have stressed, on one hand, the personal significance of these confabulations and, on the other hand, their strong association with a lesion of the right hemisphere. These facts argue against the hypothesis that the right hemisphere prevalence of the abnormal patterns of behaviour observed in stroke patients may be considered as an artefact, resulting from the language disorders of patients with left-sided stroke. On the contrary, these observations suggest that denial of left-sided hemiplegia, abnormal negative attitudes toward the paralysed limbs, personification of the affected limbs, and confabulations of denial may be considered as defensive behavioural patterns typical of the right hemisphere and aiming to protect the patient from unacceptable aspects of the reality.

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FROM NEUROSCIENCES TO PHILOSOPHY OF MIND

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Turnbull and Solms' (2007, this issue) overview of recent developments in neuropsychology (and similar studies on other neurosciences) suggests that the project Freud dreamed of is being carried out. Freud was torn all his life by an unresolvable dilemma: always hankering after the scientific discoveries (neuroanatomical, neuropsychological, biochemical...) that would provide a basis for his theories; always ambivalent when it came to indicating how far his theoretical thinking was set apart from scientific advances. On the one hand he claimed that psychoanalysis was independent from biological research (Freud, 1905/1972), that it did not have to explain itself in terms of anatomy, biochemistry or neurophysiology and need only employ purely psychological knowledge (Freud, 1917/1969). On the other, he was sure that neuroscience, from which he had drawn inspiration for his theoretical concepts (Guttmann and Scholz-Strasser, 1998), would one day produce the empirical discoveries that would provide foundation and content for the ideas put forward by psychoanalysis (ideas formed after listening to patients' stories, interpreting and examining them in the light of his own theory). Until the laboratories came up with these findings no science had, he believed, the right to censure him.

Freud's ambivalence towards neuroscience (including neuropsychology, as reviewed in Turnbull and Solms' paper, 2007, this issue) can be taken as one particular aspect of the general nature of his work and the place he occupies as a figure in the history of western culture. He wanted to be an experimental scientist but became a decipherer of human mind. He wanted to win the Nobel Prize for Medicine but won the Goethe Prize for Literature. He wanted to become a new Harvey or a new Claude Bernard but was acclaimed as a new Shakespeare or a new Nietzsche.

Turnbull and Solms' article states that we have nowadays an increasingly objective view of the neuropsychological mechanisms underlying the psychic phenomena Freud described through clinical observation and interpreted through his own idiosyncratic analysis – target, it must be said,

for much justifiable criticism (Lázaro, 2003). But the admirable researchers turning out such a fantastic number of neuropsychological and neurophysiological discoveries sometimes give the impression that they are as confused as Freud over the gap between what they believe they are doing and what they are actually doing. Neuroscientific research gives us extremely valuable information about *how* psychic phenomena occur (taking these phenomena as events in consciousness of which the subject experiencing them is aware, or which are identified and described by an observer). Neuroscience has little to say, however, about *why* these phenomena occur. *Why* is the question that could lead to the “final cause” (in the Aristotelian sense) of psychic phenomena and to their meaning in the life of the individual experiencing or producing them, since human life is a dialectical relationship with a world moulded by, and moulder of, what is known as “the identity of the human individual”.

Freud only raised this question *despite* his scientific mentality. He had been brought up in, and clung to, a tradition of positivist, naturalistic thinking. The concept of energy, which he took from nineteenth century physics and physiology, was always a fundamental reference point for him (Lázaro, 1997). He contributed many valuable findings (including those looked at in Turnbull and Solms' article) to explain the *how* of psychic phenomena, but when he tried to understand the *why* he found himself burdened by a model of the human being that reduced him to his natural state but failed to embrace his personal dimension.

Present day neuroscience should not fall into the same trap as Freud. The idea that a narrow, materialist reductionism can explain all that human beings are and do (from their most negative and terrible attributes to their most sublime creations) is too naïve. The model of emergentist and unlimitative evolutionism seems more promising, among other reasons because human history has a stubborn way of showing that explanations along the lines of “this is no more than...” are only ever temporary and soon outdated.

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“[H]E IS NO MORE A PERSON NOW BUT A WHOLE
CLIMATE OF OPINION”
(AUDEN, 1940)

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A man leaving the theater after seeing Hamlet for the first time says, “*I don’t see why everyone thinks it’s such a good play. It’s full of clichés*” – Traditional joke.

Freud was a prolific, and often prescient, theorist. A criticism of his theories is that they have not been tested scientifically and they may in fact be untestable with current scientific methods. The article in this issue of *Cortex* by Turnbull and Solms (p. 1083), compellingly refutes this criticism. They assert that aspects of Freud’s theories are testable, and that some of his theories, for example the theory of mental activity outside of conscious awareness, have been tested and shown to be correct. We agree with Turnbull and Solms that Freud’s theories should be empirically tested. Turnbull and Solms also argue that some of Freud’s theories have been unnecessarily rejected by the scientific community. We agree and in this commentary we argue that the converse is true as well; we assert that some of Freud’s theories have been accepted by the scientific and clinical communities without sufficient proof. While Turnbull and Solms (2007, this issue) argue not to throw out the Freudian baby with the bathwater, we claim that some babies are being kept unnecessarily and unhealthily in dirty bathwater.

In the article by Turnbull and Solms (2007, this issue), they use Freud’s claim that “mental activity is fundamentally grounded in a set of motivation and emotion systems” as an example of a confirmed Freudian theory, which we do not contest. However, in his “instinct” or “drive” theory (the term used depends on the variable translation of the German word “Trieb”), Freud proposed that libidinal and emotional drives (the id) are modified and inhibited by opposing forces (represented as the ego and superego) forming a balance of drive and inhibition of drive. Emotional drive has since been associated with the limbic system and inhibition of these drives with the prefrontal cortex (PFC). We argue that while this model has face validity and a pleasing symmetry – after all it is a law of physics that a force must be countered by an opposing and equal force to

achieve a stable system – widely accepted aspects of this theory are unproven and likely incorrect.

The drive theory can appear to be supported by human lesion data. Patients with damage to their PFC who subsequently demonstrate socially inappropriate behaviors (for example a patient walking up to strangers and asking if they will have sex with him or her) are said to suffer from “disinhibition”. According to drive theory, the opposing force to this patient’s libidinal drive has been reduced or removed and thus the drive, unfettered, becomes excessive (the patient would also usually be clinically described as ‘hypersexual’). Such a patient would receive a DSM-IV-TR (the psychiatric manual) diagnosis of “Personality Change Due to a General Medical Condition, Disinhibited Subtype” which is described as “the predominant feature is poor impulse control (e.g., as evidenced by sexual indiscretions)” (APA, 2000).

This clinical and scientific formulation is widely accepted, evidenced by the fact that it has been placed in a clinical manual. However, in our laboratory we have proposed an alternative theory. We have asserted that the PFC, rather than containing counterbalancing inhibitory drives, contains memories of how to perform complex behaviors, including the societal rules delineating acceptable behavior. In this view, the patient described above has lost, or at least degraded, aspects of the memory that it is considered inappropriate in our society to ask strangers to have sex with you and thus no longer understands that this is a socially prohibited behavior. In support of our theory, patients with frontotemporal dementia have a normal to decreased sexual drive since the onset of their illness (Miller et al., 1995). Please see Grafman (1995) and Huey et al. (2006) for further discussion and testable hypotheses of this model.

We have argued that, with some exceptions, these memories are activated without conscious awareness (Huey et al., 2006), similar to other areas of the brain (e.g., the motor memory of how to ride a bicycle). We believe this theory explains some inconsistencies within Freudian theory. For

example, in Turnbull and Solms' article they state that a Freudian theory that has been proven is that "conscious awareness could characterize but a small fraction of mental life, and that our experience of volition might be illusory". Sexual and emotional drives, by definition, are associated with palpable physical changes and can usually be subjectively described. How then can these drives, as Freud has suggested, reside in the unconscious without conscious detection? Our model based on memories in the PFC resolves this inconsistency; much of memory operates unconsciously and is not associated with consciously detectable changes (e.g., one is not consciously aware of the memory of how to ride a bicycle or how to swim).

Turnbull and Solms demonstrate that some Freudian theories have been proven and others are scientifically testable. We have argued that parts of Freudian theories have been accepted beyond their supporting evidence. These arguments are compatible. Freud's theories are sufficiently numerous and complex that some aspects are likely to be dismissed, and others accepted, unnecessarily. Similar to Darwin, Freud's ideas have become enmeshed into our intellectual culture to the point where, like the man in the joke at the start of this

commentary, we may not be aware which ideas have been integrated into our scientific assumptions. We agree with Turnbull and Solms that further thought and experimentation is necessary to explore the "climate of opinion" created by one of the most influential theorists of the last 150 years.

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THE “HARD PROBLEM” OF ANOSOGNOSIA: DELUSIONAL
CONFABULATION AND ANOSOGNOSIA

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In this commentary I will focus my remarks to the sections addressing confabulation and anosognosia. It is particularly gratifying to see this work progress, as the experimental findings fit nicely with the work and hypotheses we have promoted for over a decade (Feinberg et al., 1994, 2000; Feinberg and Roane, 1997; Feinberg, 2001).

ANOSOGNOSIA AND CONFABULATION

It may seem natural now, as the authors do in the target article, to assume that confabulation plays a role in the syndrome of anosognosia for illness, but this was not always the case. Although the two syndromes had been mentioned together in the literature, their relationship remained largely unexplored until relatively recently. In our first study to explore this association (Feinberg et al., 1994) we compared patients with right hemisphere lesions and anosognosia for hemiplegia (AHP) with a control group of patients with right hemisphere lesions without AHP. Patients were shown objects in the neglected hemispace and instructed to either identify the object or tell the examiner that they can’t see it. Responses were recorded as correct, incorrect or admission of failure to perceive. When compared with patients without AHP, patients with AHP had higher error rates (confabulations) and lower admission of failure to perceive rates than non-anosognosic patients in all conditions of exposure. The two groups did not differ, however, in the degree of neglect, lesion size or location, atrophy, sensory loss, or disorientation. This study provided early experimental evidence that confabulation is an important determinant in anosognosia.

In a second experiment designed to further clarify the specific relationship between anosognosia for hemiplegia (AHP) and confabulation, 11 patients with acute right cerebral infarctions and left upper limb hemiparesis were assessed for anosognosia for hemiplegia and illusory or confabulatory limb movements (ILMs) in which the patient claims that the arm is actually moving normally. In order to assess the presence of confabulatory limb movements, patients were first instructed to raise their unaffected right arm and were asked “Is your

right arm on the bed or in the air?” Then they were told to raise their plegic left limb and asked if the left arm was on the bed or in the air. Lastly they were told to raise both arms and were asked about the location of each arm. Five of 11 patients had unequivocal confabulation as evidenced by ILMs. The presence of ILMs was associated with the degree of anosognosia ($p = 0.002$) and with hemispatial neglect ($p < 0.05$). From this we concluded that a strong relation exists between anosognosia for hemiplegia and confabulations concerning the movement of the plegic limb. An interesting and somewhat unexpected finding was that all patients with unequivocal ILMs had asomatognosia (5/5), and all patients without ILMs lacked asomatognosia (4/4), and confabulation and asomatognosia were highly associated ($p < 0.01$). Since asomatognosia is often a form of delusional misidentification, this latter finding suggested a further link between confabulation, anosognosia, and misidentification (see below).

CONFABULATION AND POSITIVE BIAS

Once the association between anosognosia and confabulation was established, we wondered if confabulation fully “explained” the phenomenon of anosognosia. We concluded that it does not. Our reasons for this conclusion were simple. Confabulation can be the result of many different and interacting factors including confusion, memory loss, executive and self monitoring defects, reality monitoring defects, retrieval defects, and other causes (for review, see DeLuca, 2000). Confabulation that results from these defects could also account for a simple *unawareness* of a neurological problem. For present purposes, I would argue that this is part of the “*easy problem*” of confabulation and anosognosia, since these negative features do not fully explain the most interesting and perplexing positive aspects of some varieties of confabulation that may occur in association with anosognosia. These latter features include the delusional nature of some confabulations, the delusional nature of some anosognosic syndromes, and the metaphorical and symbolic aspects of some

confabulations. In other words, the first set of explanations does not explain the most *Freudian aspects* of these conditions. This latter problem I consider the “hard problem” of confabulation and anosognosia.

The findings that confabulations sometimes have a positive bias, as noted in the Turnbull and Solms target article, runs into similar difficulties, as has been pointed out in prior commentaries (Feinberg, 2004; DeLuca, 2004) All of the aforementioned *negative* neuropsychological defects could actually result in an artifactually *positive* bias in confabulation. For instance, if a disoriented patient cannot remember where he is, and mistakenly thinks he’s at home or work, as opposed to being in a hospital, or thinks she’s perfectly well in actually is very sick, what exactly does that demonstrate? Is that a simple failure of memory or self-monitoring, or something more interesting from a dynamic standpoint, such as psychological denial or a motivated and possibly delusional belief?

DELUSIONAL CONFABULATION, MISIDENTIFICATION, AND PSYCHOLOGICAL DEFENSE

But not all anosognosic-confabulatory patients are created equal. Coming from an analytical-dynamic perspective, we have been most interested in those cases in which the anosognosia and confabulation could not be fully explained on the basis of these negative factors. We therefore have focused our studies on those cases in which the patient seemed deluded regarding their confabulations and denials, in which the patient seemed to have implicit knowledge of the deficits, and this implicit knowledge emerged in a metaphorical fashion in the patient’s confabulations. In other words, we were interested in the most Freudian aspects of these syndrome. Based upon this point of view, we posited that a variety of confabulation that we called *personal confabulation* held the most promise for revealing these self-related, motivationally and defensively driven varieties of confabulation (Feinberg and Roane, 1997).

One domain of confabulatory delusional belief that the authors do not address in the target article

are the delusional misidentification and reduplication syndromes that commonly occur in association with confabulation and anosognosia, as noted above. In our most recent study (Feinberg et al., 2005), we analyzed a series of previously published cases of these conditions. The criteria for inclusion in our series were patients who displayed stable misidentification(s) or reduplication(s) of the Capgras or Frégoli type in the presence of focal brain pathology. Case descriptions also had to provide sufficient clinical detail to determine the nature and stability of the misidentification and adequate neuroanatomical detail to determine to the anatomical focus of the brain lesion. Cases were categorized according to the predominant type of misidentification into six classes: 1) Capgras syndrome for person(s); 2) Capgras syndrome for environment; 3) Capgras for the arm (asomatognosia); 4) Frégoli syndrome for person(s); 5) Frégoli syndrome for environment; 6) Delusional reduplication (without misidentification) for the self or other persons. There were a total of 27 cases and 29 observations reviewed. Two cases who had prominent misidentification for both persons and the environment appeared in both categories.

One of the most striking finding of this investigation was the extremely high incidence of right frontal damage. In over 96.6% of the observations (N = 28 of 29), there was right frontal damage. This is compared to left frontal damage which was present in only 48.3% of the observations (N = 14). A likelihood ratio test revealed that the occurrence of right frontal damage was significantly above chance ($p < .001$).

CONCLUSIONS

As the authors point out in the target article, one of the most fruitful areas for exploring neuropsychanalytic concepts is within the intersecting domains of the syndromes of confabulation and anosognosia. Within this group, our researches indicate that it is valuable to segregate the negative neuropsychological deficits from the positive adaptive, defensive, and symbolic aspects (Table I). It is within this domain that I

TABLE I
Negative and positive features of delusional misidentification, reduplication, and confabulation

Negative features	Positive features
Spatial disorientation	Paranoia
Visuoperceptual disturbance	Delusional nature
Hemispacial neglect	Wish-fulfillment
Executive dysfunction	Defensive operations: projection and denial
Retrograde and anterograde amnesia	
Autobiographical memory loss	
Temporal context confusion	
Reality monitoring failure	
Anatomical disconnection	
Disturbance of self-representation/ego functions	

From Feinberg et al. (2005), with permission.

suggest the most Freudian aspects of these syndromes are revealed. It is of greatest interest that right hemisphere, and especially right frontal pathology seems so prevalent when patients display these dynamic features. We have previously suggested that classical Freudian ego functions and ego boundaries are particularly disturbed in the presence of right frontal dysfunction. I would also argue that the same damage facilitates the release of certain defensive functions that may be within the purview of the verbal left hemisphere.

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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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MOTIVATIONS AND EMOTIONS CONTRIBUTE TO A-RATIONAL UNCONSCIOUS DYNAMICS: EVIDENCE AND CONCEPTUAL CLARIFICATION

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Turnbull and Solms (2007, this issue) call attention in their target article to several ways in which neuropsychological and psychoanalytic concepts throw light on each other. They conclude that emotion and motivation so central to psychoanalysis have been underinvestigated in neuroscience. We agree with this position, but believe there is more to psychoanalytic theory and its implications for neuroscience than the authors have discussed.

AWARENESS

After a brief review, the authors conclude that “The principle distinction is that the findings... confirm the existence of *cognitive* processing outside of conscious awareness, whereas Freud claimed specifically that *motivational* and *emotional* factors shape conscious mental life” (p. 5). Fortunately, there have been quite a few studies of unconscious processes dealing with emotional and motivational factors. Employing a time-frequency analysis of ERPs, Shevrin et al. (1992) have identified neurophysiological markers for unconscious conflict in social phobics which correlate with personality measures related to repression. Unconscious conflict is a central concept in Freud’s theory of psychopathology, and involves powerful emotional and motivational factors. Shevrin et al. (2002) have shown that the same measure of repression correlates significantly with Libet’s measure of time-to-consciousness of a stimulus, repressive subjects having a greater time to consciousness. In a series of two subliminal aversive conditioning studies, Wong et al. (1994, 1997) have demonstrated that, 1) a frowning face conditioned to a shock consciously when presented subliminally subsequently will elicit a greater P300 than a pleasant face, 2) the same frowning face can be aversively conditioned unconsciously with the same difference in P300 present in subsequent supraliminal presentations. Bernat et al. (2001) have shown that negative valence words presented

subliminally will elicit greater event-related potential amplitudes for components across the brain (N100, P200, P300, LP1, LP2) than positive valence words.

The investigation of unconscious emotional and motivational factors is alive and well in neuroscience and speaks to a greater convergence of interests between neuroscience and psychoanalysis in the study of unconscious processes than identified by Turnbull and Solms.

EMOTIONS AND MOTIVATIONS

The authors appear to be of two minds about the role of emotional and motivational factors in false beliefs. At one point they appear to consider them to be independent factors: “...the central psychoanalytic claim is that emotion systems (*and the drives that govern them* (italics ours) might distort cognitive representations of reality...” (p. 8). Elsewhere the authors confound the two: “...there is powerful support for the claim that *basic instinctual emotion systems* (italics ours) represent an important component...” (p. 8). Furthermore, while they emphasize how emotions generate false beliefs in anosognosia, they also describe how patients need to maintain a state of positive feeling through denial while dealing with loss, thus implicating motivation. Following their own formulation these motivations would presumably constitute one set of causes governing the emotional dysregulation, along with the presumed direct effect of right hemisphere lesions on emotional systems.

Finally, the authors are clear in identifying an independent motivational system when they refer to findings that “...dreaming stops completely when fibers in the ventromedial frontal lobes are severed; a symptom that coincides with a general reduction in *motivated behavior* (italics ours)” (p. 15), which they then relate to similar effects produced by prefrontal leucotomy involving destruction of the same pathways. However, almost immediately after

making these points, the authors revert to placing primary importance on the role of “powerful positive emotions... at the heart of the ‘false belief’ states that generate the dream process” (p. 16).

The authors need to clarify to what extent they mean emotion and to what extent motivation. This distinction is of prime importance in Freud’s theory of mind, as well as mobilizing different physiological systems. Drives and motivational systems induce action by mobilizing motor systems from motor areas to striate muscles; by contrast, emotions primarily mobilize the inner body smooth muscle and gland systems, the effects of which are sensed as affects. In our view Freud did not understand delusions, confabulations and dreams as primarily a dysregulation of emotion systems, but as a different organization of action systems. Indeed, Freud (1895/1966) would not hold that individuals experiencing false beliefs, delusions, or dreams simply behave irrationally, but that they are employing different rules, namely those of the primary process that Brakel has suggested should be referred to as a-rational (Brakel, 2002). For Freud (1895, p. 325-326), the primary and the secondary process are both differently organized action systems. Only the secondary pathway, referring to the rational has the means, thanks to the ‘indications of reality’ (1895, Freud, p. 309), to engage in a reality check. Freud’s take on the role of primary processes in a-rational behavior is very much consistent with modern sensorimotor theories: indeed different authors (Frith, 1992; Blakemore et al., 2000) have underscored the role of absent or dysfunctional efference copies leading to a disturbed reality check in psychosis and there is good argument for a functional equivalence between the efference copies and Freud’s secondary process ‘indications of reality’ (Bazan, in press). Moreover, the suggestion that a-rationality is due to an action rather than to an emotion dysregulation, also makes it easier to understand why all delusions are not mostly positively biased. While the authors propose that positive affective bias of false beliefs serves as a defense against an unbearable reality it should be stressed that neither psychotic delusions, nor dreams are mostly positively biased. In delusions, paranoid imagery of

poisoning, weapons etc is very common; when positive imagery is present, it is always together with negative (paranoid) ideas. For all these reasons, we doubt the author’s proposition that “...the growing interest in emotion will be accompanied by increasing awareness of the importance of emotion in *all* classes of delusional thinking.” (p. 18).

In summary, although we agree on the need of neuroscience to take unconscious emotion and motivation into account, we stress that (1) significant experimental results have already been obtained in these areas, and (2) dysregulation at the level of the organization of action, and not primarily at the emotional level, is a more likely explanation for “a-rational” behavior in accord both with Freud and modern sensorimotor theories.

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NEURO-PSYCHOANALYSIS MAY ENLIVEN THE MINDBRAIN SCIENCES

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Early in the 20th century, radical behaviorism served as antithesis for a conceptually enriched but scientifically impoverished psychoanalysis. That extreme “never-mind” perspective has now bred enormous neuroscientific problems of its own (Panksepp, 2005), needing a strong antidote along the lines outlined by Turnbull and Solms (2007, this issue). The banishment of the affective mind from behavioristic psychology continues to be problematic for our understanding of animal behavior as well as cognitive processes. To make sense of many brain functions, the neuroscientific community must invest more vigorously in a scientific analysis of affective experiences. Turnbull and Solms make a good case for a new synthesis, where all relevant approaches to the brain synergize as they jointly seek a deep empirical understanding of the real complexities of the human mental apparatus. Turnbull and Solms provide several cogent examples from neuropsychology. Psychiatry and academic psychology provide many others.

By seeking a workable synthesis of MindBrain studies, we may avoid mental “sciences” without adequate brain perspectives, a characteristic of psychiatry during the first-half of the 20th century. The second half, when biological psychiatry prevailed, saw abundant brain science depleted of mentality. Hopefully, more balanced psychiatric traditions will emerge as experiential and neural aspects of the BrainMind equation are brought together (Panksepp, 2004). As Turnbull and Solms clarify, affective perspectives are as relevant for understanding neuropsychological issues as cognitive abilities, and there are cogent reasons to bring psychoanalytic ideas and tools back to the scientific table.

Obviously, no single experiment can evaluate global theories of the mind such as Freud generated. No experiment can illuminate the whole mental apparatus. Each question has to be broken down into empirically workable chunks. Experimental science always clarifies *parts* of phenomena, never the *wholes*. Modern cognitive neuroscience has also not been resistant to the allure of part-whole confluences (Bennett and Hacker, 2003). The big picture can only be constructed with imagination, leading to

contentious opinions, as with Freudian theory, which remain empirically unresolved. Perhaps, through many highly replicable experiments, rather than the weight of argumentation, we can construct a realistic theory of the whole neuromental apparatus that most psychoanalysts, and some neuroscientists, would like to have. Turnbull and Solms (2007, this issue) encourage the arduous accumulation of “peppercorns of evidence” that may convince the scientific community that certain psychoanalytic ideas can yield new predictions that will illuminate various perplexing psychological phenomena.

As neuro-psychoanalytic research matures, hopefully practitioners will avoid the allure of saying too much on the basis of too little data, a temptation that is becoming rampant in the popularization of many modern neuroscientific findings. We hear headlines about “oxytocin, the love molecule” and such. Yes, we can be confident that brain oxytocin networks contribute something to broad concepts such love and social attachments, but this does not mean that we are close to any thorough understanding what “love” and “social attachment” really are within the brain. Many other neurochemistries participate, including brain opioid and prolactin systems. However, a substantive start has been made. The same goes for all the basic emotional systems of the brain (Panksepp, 1998), which may be yielding a scientifically resolved description of “the id”.

A dilemma for a neuro-psychoanalytic synthesis is that we remain in the era of ruthless neural reductionism – a radical materialism that leaves little causal room for mentality in the brain-body-environment equation. Such mindless materialism will prevail until investigators can demonstrate that a study of subjective experience provides novel, robust and useful scientific predictions. Turnbull and Solms (2007, this issue) provide some and highlight that cognitive neuroscience came to accept the Freudian dictum that the staging ground for our experienced lives is controlled by innumerable unconscious processes. However, we should not fall into the trap of reifying reported “subjective awareness” as the *sine qua non* of consciousness. We must remain open to more radical views, for instance that *raw experience* is

the primordial variant of consciousness – that affective feelings can emerge from brain dynamics, even when there is no cognitive *awareness* of those feelings, as in children born with essentially no neocortex (Shewmon et al., 1999). We must remain open to the possibility that consciousness is constructed from evolutionary layers of experiential capacities, and that earlier subcortical forms may be actively suppressed by higher brain functions, which can be released by brain damage or other compromised mental states.

We must fully consider that the non-speaking right hemisphere is as consciously aware as the left; the lack of access to linguistic output channels in split-brain people does not diminish its experiences. We must develop experimental tools to get at non-propositional, non-speaking dispositional aspects of mental life that are surely pervasive in other mammals (Panksepp, 2005). When we do, we may be surprised to learn that much of what has been placed in the unconscious category by the more self-centered and oft delusional and arrogant left hemispheric linguistic apparatus, is actually experienced somewhere or at some time in the brain.

When neuroscience opens itself up again to the high probability that one of the functions of complex neural networks is the creation of psychological experiences that control overt behavior, we may resolve many paradoxes. For instance, it is rarely considered that Benjamin Libet's elegant experiments, positively summarized by Turnbull and Solms (2007, this issue), may *not* have demonstrated that intentional behaviors emerge in the brain unconsciously (i.e., those experiments may only indicate how synaptic delays in the self-directed visual system have difficulty evaluating exactly when one did have the intention to move). Libet's striking findings may indicate that deep intentionality is blind, and it requires some time for the visual apparatus to register when the intentional act occurred. Maybe deep intentionality occurs essentially at the same time one sees electroencephalographic indices of forthcoming motor acts on the cortical surface, and that *same* intentionality, after a brief attentional

“blink,” redirects visual processing to the second hand of the clock. Thus, an important point to keep in mind is that every empirical finding has multiple interpretations, and rational sifting of alternatives always requires more research. It is good to see concrete negations proposed for pet ideas.

The methodological problems associated with a MindBrain science that aspires to deal with a fuller evolutionary understanding of the human mental apparatus will remain a challenge for generations of interested scholars (Northoff et al., 2006). However, we will make little progress on issues of utmost importance – the biological nature of “soul” (id-ego?) and its intrinsic values (i.e., affective experiences – from anger and grief to joy and euphoria) – until we probe, in detail, comparable mental processes in other animals (Panksepp, 2005). Scientifically enlivened psychoanalytic approaches to the MindBrain, tethered to rigorous methodologies where conclusions are constrained by the weight of evidence, will help reveal the true complexities of the mental apparatus that Freud only envisioned in broad and, at times, exceedingly creative brush strokes.

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FREUD THROUGH THE CENTURIES

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The 19th century saw a total revamping of our understanding of brain function and its relation to behavior and experience. Sigmund Freud partook of this revamping and contributed substantially to it. In this brief essay, I want to highlight some of the less publicized transactions between Freud and his intellectual milieu.

From the time of Hippocrates the essential relationship of the brain to the normal functioning of all aspects of the psyche had been accepted, as had been the disturbances of psychological experience and of behavior as a result of brain injury. During the nineteenth century these correlations were refined and regions of the brain were identified as serving a variety of “faculties” of mind. In his classical book *“On Aphasia”* Freud took exception to these restricted observations of correlations and proposed a more integrated view of brain processing of language that involved the interactions among several brain regions.

Freud’s next accomplishment was the solution of the problem of what was, during the latter part of the nineteenth century, called the “Act of Thinking.” The philosopher Franz Brentano had taken up this issue from the Würtzberg School of Experimental Psychology led by Oswald Külpe. The Würtzbergers had shown that when people thoroughly grasped the meaning of a question posed to them that everyone would come up with the same answer. However, the way in which they solved the problem posed by the question was not uniform. Some people worked step by step; others went to sleep and woke up in the morning with the answer. There were many variations of problem solving that fell in between these extremes. Brentano, and also William James, concluded that the thought process is active, an activity that is somewhat like a flowing stream that was bounded by its banks – the problem set by the experimenters.

Freud, a pupil of Brentano’s, saw in the “act of thinking” an opportunity to get to understand how people had become neurotic. A neurosis, Freud defined as an overriding wish. Normally when we want something we subject that want to reality testing. In his “Project for a Scientific Psychology”, also called a “Neurology for Psychologists”, Freud proposed that ordinarily we use a double attentional process to satisfy our

wants. The double attentional process consists of noting something in the environment and comparing it to one’s stored experience and then “taking a second look” to ascertain whether the “object” satisfies that “experience”. Wishing fails to reality test – and chronic wishing is neurotic.

Freud wondered what blocked reality testing in neurotic people. These people had problems and those problems were readily identified and agreed upon. Freud proposed that the patient’s thought processes held the key to the blockage and therefore had them “free associate” – within the setting set by the problem. Psychotherapy was invented.

With regard to the brain, Freud pointed out that in daily life we need two very different sorts of processes. One process allows us to fleetingly notice objects in our environment, the other stores what we have experienced. What we need, Freud noted is two brains, one for perception and one for memory. And, on the basis of the then current knowledge he suggested that the brain cortex serves perception and that the deeper structures of the brain (the basal ganglia?) serve memory.

During the 18th century the brain was viewed, as it had been for centuries, as an organ that functioned by way of “spirits”, thought of in terms of respiration – as expressed by such terms as being inspired. By the end of the 19th century the contemporary view of brain as an electrochemically functioning organ had been attained. Freud, in his “Project for a Scientific Psychology”, proposed that the cortex and deeper structures were made up of component cells separated by contact barriers (the term synapse had not been applied to these contacts). The way in which the cellular components operated was threefold: action currents, local graded potential changes, and resistances at the contact barriers. (‘Action currents’ was awkwardly translated into English as currents in flow; ‘local graded potential changes’ as cathexis.) Freud had proposed an Ohm’s law of neural activity spelling out in detail the nineteenth century view of the electrochemical nature of brain activity.

Freud’s view of the operation of the “local graded potential changes” which today we call “field potentials” and Freud’s translators called “cathexis” has led to many misinterpretations of

psychoanalytic doctrine. Potential changes are changes in energy (potential energy) so when psychoanalysts claim that a person invests energy, that is “cathexis” in another person, the brain correlate to this is that dominant field potentials move from one brain location to another. We now call these shifts in “cathexis” – “temporary dominant foci”.

On a personal note, I recall the vehemence expressed during the mid-20th century by behavioral and neuro-scientists against Freud and psychoanalysis for using energy concepts – and psychoanalysts who wanted to give up energy concepts in favor of the now popular “information” processing. Nineteenth century Freud carefully defined his terms, knew his neurology first hand as well as its relation to the behavior he was observing and the stories (free associations) that his articulate female patients told him.

In addition to his discernment of the double loop of attention involved in reality testing, Freud had another important insight: the prospective aspect of memory is motivation. Our behavior is motivated, guided by, the structure of our memory. The term motivation had hardly been used before Freud had this insight.

But motivation as memory-based got into trouble during the early part of the 20th century. Some of Freud’s patients realized that their memories might not be veridical to what actually occurred – that their memories had become distorted. Freud has recently been accused of changing his tune on early traumatic experiences to satisfy the parents of his patients and, in this way, to help himself become established. To my mind this is poppycock for two reasons: one is that Freud was shaken by the revelation that perhaps memories are not veridical: his therapy and ideational frame were based on the “memory-motive” relationship. Second, Freud as a Jew had already become alienated from the Viennese establishment – no recantation would in itself heal that breach.

Freud’s articulate patients suggested that the endocrine surges that occur at puberty were the origins of the changes in memory. Drives influence motivation. During the twentieth century, the memory-motive relationship was forgotten and drive based motivation became a dominant theme (as in Clark Hull’s drive reduction theory of learning). As is the case with many twentieth century ideas, they somewhat distort fundamental insights by overemphasizing a flashier modification. Hormones are important for setting dispositions, levels of behavior and experience, but they do not by themselves guide, motivate, the detailed patterns of behavior and experience.

Another twentieth century casualty of Freud’s 19th century insights is the concept of the superego. Initially the superego was the memory network formed from the intervention by a baby’s caretaking person to relieve the pressures of drive

stimuli. The baby becomes hungry, his ego functions have learned to cry in a certain way, and the caretaking person comes to feed the baby. The superego is conceived as a helping process, not as a means to block a person’s needs. During the 20th century, perhaps because psychoanalysis deals with ailing patients, the superego processes become repressive instead of facilitating.

Freud frequented the coffee houses of Vienna. One of the topics of conversation during the last decades of the 19th century was the discovery of the laws of thermodynamics. The heat engine powered the burgeoning economies of the century: railroads, steam boats, factories were all powered by the heat engine. It was economically important to know how much heat was being used effectively and how much was wasted. Ludwig Boltzman was formulating the laws of thermodynamics in Vienna and there was much discussion as to their validity. Freud was formulating the laws of behavioral dynamics and, most likely, could not help but become influenced by what he was hearing in the coffee houses. Boltzman established the first and second laws of thermodynamics; Freud came up with primary and secondary psychological processes. Boltzman’s first law describing equilibration in thermodynamics, that every action begets an equal and opposite reaction. Freud’s primary process concerns the equilibration of energy among neurons and among people. The second law of thermodynamics states that in any closed system patterns of energy tend to dissipate into heat. Freud’s secondary process states that, contrary to non-living systems, in living systems raw energy (heat) becomes more and more organized into patterns, that is, into cognitions (Freud coined the term agnosia, for loss of cognition as a consequence of brain injury).

There are many more such insights recorded in Freud’s “Project”. One of them deals with dreaming which he calls a compromise process. Sleep is due to a lowering of energy throughout the brain, a suggestion that has been confirmed with direct current recordings a few years ago. As energy becomes restored on awakening, the beginnings of cognitive patterns are formed. These cognitions are piecemeal, joining segments of brain processes that had become distributed during sleep.

But not only is memory basically distributed in the brain: so is the basic brain process operating in perception. Sensory input reaches the brain cortex, the part of the brain that deals with perception, via direct pathways that have very few contact barriers. Therefore there are few resistances to distort the patterns of energy sensed by receptors. Freud puts it: The patterns of periodicity that characterize sensed energy are transmitted practically without change to the cortex. Today we speak of frequency, the inverse of periodicity. Field potentials are characterized by patterns formed by the frequencies of their oscillations.

Freud distinguished his clinical theory from his metapsychology, one branch of which I've detailed above. Another branch, his cultural metapsychology he developed during the twentieth century. Also, during the twentieth century, he developed his clinical theory. One facet of this theory is worth noting here: the stage theory of emotional development. This theory suggests that an oral stage is followed by an anal stage and finally a sexual stage. Freud observed these stages in his own children and they have been seen by most of us who have children.

I paid little heed to Freud's stages until I heard Jean Piaget, who is noted for his theory of stages of cognitive development, present a lecture in New York describing the parallels between his stages and those of Freud. In dinner together afterwards Piaget and I discussed the commonalities that characterized the stages, looking for words that would apply to both theories. I had already noted a similarity between Piaget's stages and those developed by Harry Stack Sullivan in his interpersonal theory of psychiatry. Roughly speaking we appear to go through stages in all of our development: from a touchy-feely, through a control phase, and finally a mastery stage.

During Freud's oral stage (Piaget's sensory-motor stage and Sullivan's protaxic stage), the baby smears the food all over his face; during the anal stage (Piaget's operational stage(s) and Sullivan's parataxic stage), the child needs to get control over his sphincters and also attain control over his

caretaker to get him to the toilet. Graduating from these stages, mastery is achieved; ideally for Freud in a satisfying sexual (in Piaget post-operational, and in Sullivan, a communicative) relationship.

I have suggested that these stages apply not only to children's development, but also to all really new endeavors we undertake. For example, when a student comes into my laboratory I tell him to get the feel of what we are doing: fiddle with knobs on the instruments; try taking a few of the behavioral tests, etc. Next, when the student wants to do some research he or she must learn to control the instruments and the testing situations involved.

In interpersonal relations such as falling in love, during this initial phase we literally can't keep our hands off each other. The lovers, when they come to live together must decide who does the dishes, who is to take out the garbage, and so on. Graduating from these stages, mastery is achieved.

These are a few examples of the roots of Freud's thinking and the influence his insights can have outside the sphere of psychoanalysis and psychotherapy. Freud, in this way, is very much alive, in this, the 21st century.

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ANOSOGNOSIA, CONFABULATION, AND DREAMING:
A NEUROPSYCHOLOGICAL PERSPECTIVE

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Undoubtedly, psychoanalysis is one of the most well-known theories that attempt to explicate the workings of the human mind. Its enormous cultural legacy contrasts greatly with the paucity of studies that have tried to test its ideas. In the paper by Turnbull and Solms (2007, this issue), the authors argue that such tests are feasible and review four domains of neuropsychological evidence (anosognosia, confabulation, psychotic delusions, and dreaming) that appear to support Freudian postulates. We agree that at least some of these ideas can be tested and believe that future methodological advances can increase the number of such testable claims. We do not believe, however, that at present, empirical findings in these four areas of neuropsychological inquiry convincingly support or disprove psychoanalytic ideas. As discussed below, one reason for these inconclusive results is the omission of important features from the design of previous studies. Another reason is the lack of studies aiming to test hypotheses that are specific to psychoanalysis and that, therefore, cannot be easily explained by other, very different theories. This state of affairs is not surprising considering how young the field of neuropsychanalysis is. Thus, the following discussion is not meant to imply that the studies discussed by Turnbull and Solms (2007, this issue) have been fruitless, but to show that the time is ripe for more complex investigations. Most of this discussion focuses on our reading of the literature that investigates anosognosia and confabulation. We conclude our commentary with brief and speculative remarks on the psychoanalytic theory of dreaming.

Though it is plausible, even likely, that emotional and motivational factors contribute to anosognosia, there is currently no adequate evidence showing that these factors are similar to Freudian defense mechanisms (e.g., repression of the information that evokes powerful negative emotions). Two studies (Turnbull et al., 2002, 2005) cited by Turnbull and Solms (2007, this issue) seem to suggest that anosognosics have impaired ability to tolerate strong negative emotions as a result of their brain lesion. These studies have shown that whereas anosognosics display adequate levels of emotion in general, they

experience emotional breakdowns more often than non-anosognosics. The vast majority (75%) of these breakdowns were preceded by discussions involving emotions of separation and distress (Turnbull et al., 2002). However, because the neurological control groups in both studies consisted almost entirely of patients with left-sided brain lesions, differences in lesion location or size between the anosognosic and control groups may have accounted for the group differences reported. The results of a study testing the same participants at various stages of recovery from anosognosia would be more conclusive. Such a design does not rely on complete resolution of anosognosia as even partial recovery would be expected to reflect increased ability to tolerate negative emotions. Similarly, the findings (e.g., Turnbull et al., 2004) that seem to suggest that the content of confabulations shows a positive affective bias are difficult to interpret since, with one exception (Fotopoulou et al., 2004), they were obtained in studies that did not utilize a control group.

Another methodological concern is the absence of differentiation between the subgroups of anosognosic and confabulating patients in many studies examining these populations. Such differentiation is important given the evidence that patients within each of these populations have heterogeneous symptoms and etiology (Marcel et al., 2004; Schnider et al., 1996). For example, important distinctions between the etiologies of provoked versus spontaneous confabulation have been demonstrated (Schnider et al., 1996). It is possible that inconsistent patterns obtained across participants in some studies (e.g., Fotopoulou et al., 2004) are a result of this heterogeneity.

The increased methodological rigor of future investigations will be valuable only insofar as it is used to test psychoanalytic ideas as directly as possible. Considering that the psychoanalytically driven questions posed by Turnbull and Solms (2007, this issue) are quite novel, the relevant findings that have been obtained so far are encouraging. Many of these findings, however, may, at present, be explained by theories that are not related to psychoanalysis. Such is the finding

that confabulations show positive affective bias (Fotopoulou et al., 2004), a result that, in and of itself, can be explained by several other theories. Similarly, the findings of some of the case studies (e.g., Bottini et al., 2002) cited by Turnbull and Solms (2007, this issue) in support of the idea that anosognosics have impaired ability to tolerate strong negative emotions are more consistent with the alternative interpretation that these patients are unable to tolerate the enormous change to their body schema caused by hemiplegia.

Given the possibility of alternative explanations for these phenomena, the next logical step would be to test theoretical constructs that are unique to the Freudian theory. Although these constructs are discussed in the introduction of Turnbull and Solms's paper, they have not been empirically examined in the context of confabulation and anosognosia. Among them is the idea that anosognosics are implicitly aware of their hemiplegia (Turnbull et al., 2002). Research methods used to manipulate and detect subconscious cognitive processes (e.g., presenting subliminal information and/or measuring physiological indices that reflect emotional processing) are suitable for this line of research. Defense mechanisms are another mental process that is central to the psychoanalytic account of anosognosia. Although the influence of defense mechanisms has been inferred from patients' emotional reactions (Turnbull et al., 2002), observer-based measures (Perry and Ianni, 1998) may more directly assess the presence of defense mechanisms. It may be worthwhile to apply the same approach of testing specifically Freudian notions in investigations of Turnbull and Solms's hypothesis that "the 'magnitude' of the false belief should be somehow 'proportional' to the magnitude of the emotion required to generate and sustain it." For example, after additional evidence for this claim is accumulated, it would be interesting to study whether the emotional factors fueling false beliefs are related to the Freudian drives of sex and aggression, and whether most of the time, such fueling is subjectively advantageous to the individual.

Much more than confabulation and anosognosia,

dreaming has long been a subject of psychoanalytic interpretation. The psychoanalytic perspective argues that the function of dreams is to help repress unconscious thoughts and wishes that may be threatening for the individual to cope with while being awake/conscious. It is possible that dreams can serve as a conduit to these wishes since roughly 75% of dreams carry emotional content (Fosse, 2001). Further, Turnbull and Solms have provided evidence from brain imaging studies that emotion systems (i.e., amygdala and nucleus accumbens) are activated and that reality monitoring areas (i.e., prefrontal cortex) are deactivated in individuals during REM sleep. Therefore, we agree with the authors that further study may elucidate Freud's wish-fulfillment theory of dreaming.

Based on the preceding arguments, we conclude that the findings to date do not allow us to accept or rule out the psychoanalytic view of anosognosia, confabulation, and dreaming. It is our feeling that the studies done by Turnbull, Solms, and their colleagues are a laudable prelude to more rigorous and direct tests of this view in the future.

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WAKE UP OR DREAM ON?
SIX QUESTIONS FOR TURNBULL AND SOLMS

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With respect to dreams, Sigmund Freud was not only not right. He was dead wrong. And so are Turnbull and Solms (2007, this issue) in their desperate effort to save Freudian psychoanalysis from the junk heap of speculative philosophy. Neuroscientists, psychologists, and psychiatrists beware: you are being led down the garden path by this pair of misguided neo-Freudians.

In this commentary, I will show that Freudian psychoanalysis, à la Turnbull and Solms, is not only pseudoscientific but will suggest that it is actively antiscientific. Turnbull and Solms' failure to acknowledge the errors of psychoanalytic dream theory warrants the rejection of their suit for the hand of neuroscience. My comments will be organized as a set of six questions to which Turnbull and Solms should give clear cut yes or no answers. Press conference equivocation is not welcome!

QUESTION 1:

ARE REPRESSED INFANTILE WISHES
THE INSTIGATORS OF DREAMING?

Sigmund Freud was not aware of the periodic brain activation of REM sleep that is now a well accepted factor in the instigation of the long, bizarre, hallucinatory REM sleep dreams that he sought to understand. Freud therefore hypothesized that unconscious infantile wishes were the instigators of dreaming. While Turnbull and Solms do accept "the near perfect fit between dreaming and REM", they argue that because REM sleep and dreaming are sometimes doubly dissociable, the instigation of REM sleep dreaming is not the motivationally neutral brain activation in sleep as McCarley and I have suggested (Hobson, 1988; Hobson et al., 2000; Hobson and Pace-Schott, 2002).

I now challenge them to explicitly deny the Freudian hypothesis of dream instigation by repressed unconscious infantile wishes and to explicitly accept the simpler, more plausible hypothesis that dreaming is instigated by motivationally neutral brain activation in sleep. Brain activation is very significant even in NREM sleep and we have never argued for an exclusive relationship of dreaming to REM. This does not mean that dreaming reveals no motivation. It

certainly does and dreaming is thus of interest to psychologically oriented people (Hobson, 2004). But that repressed infantile wishes (if they exist at all) are instigators of dreaming is not longer a tenable hypothesis. Do you agree? Yes or No?

QUESTION 2:

IS DREAM BIZARRENES CAUSED BY DEFENSIVE
TRANSFORMATION OF REPRESSED INFANTILE
WISHES?

Sigmund Freud hypothesized that defensive transformation was the mechanism of dream bizarreness. By means of disguise and censorship, the repressed, unconscious, infantile wishes that Freud supposed instigated dreaming were rendered incapable of invading consciousness and causing awakening. Thus dreaming was seen, by Freud, as the "guardian of sleep." This defensive transformation hypothesis is the kernel of Freud's erroneous view of mental life in general. He posited a universal neurotic compromise between the dynamically repressed unconscious and consciousness.

Because this disguise-censorship concept is so pivotal, it is important to the scientific evaluation of Freud's theories. The simpler, more robust, alternative hypothesis that dream bizarreness is the undisguised read out of inchoate disparate orientational data by the autoactivated but demodulated brain in sleep was advanced by us 30 years ago. But to date, there has been no explicit negation by Turnbull and Solms of the now obsolete disguise-censorship hypothesis that is at the very center of Freud's dream theory. I therefore challenge Turnbull and Solms to state, publicly, whether or not they have rejected disguise-censorship as the explanation for dream bizarreness. Please vote yes or no on question 2.

QUESTION 3:

HOW DO YOU (OR FREUD) ACCOUNT
FOR NEGATIVE DREAM EMOTION?

Freud was aware of the inability of his dream theory to account for the negative emotion of many dreams. We now know that at least half of our

dreams are fraught with anxiety or aggressiveness. (The other half may be ecstatic and thus reflect undisguised pleasure). We also now know that a major forebrain target of the cholinergic brainstem to forebrain activation circuit of REM sleep is the amygdala. It is probably this intrinsic brain activation process that causes such intense and often unpleasant dream emotions. If Turnbull and Solms agree with this theory, let them say so. They will thereby let Freud off the hook and buy into a renewed interest in dreams as the synthesis of the undisguised emotions with cognition. This is question 3. Yes or No?

QUESTION 4:
WHY ARE DREAMS SO HARD
TO REMEMBER?

This question is particularly relevant if, as Freud erroneously surmised, dreaming was the result of the successful obfuscation of latent content. Why should the already bowdlerized manifest content be so difficult to recall?

Instead of re-repression or some other equally farfetched Freudian explanation, it is likely that recent memory mechanisms are disabled in sleep. Neurobiology offers two possible mechanisms: one is the unavailability of serotonin and norepinephrine and the other is the persistent deactivation of the dorsolateral prefrontal cortex that disable recent memory both within the dream and upon awakening from sleep.

Simple organic amnesia is the culprit, not the attempt to sweep incompletely disguised unconscious infantile wishes under the carpet. I invite Turnbull and Solms to answer this question clearly. Do you agree? Yes or no?

QUESTION 5:
IS DREAM INTERPRETATION
SCIENTIFICALLY VALID?

The brain based hypothesis that Robert McCarley and I put forth in 1977 always has insisted that the forebrain "made the best of a bad job" in synthesizing an emotionally salient story out of the motivationally neutral signals sent up to it from the brainstem in sleep. In other words, we never suggested that dreams were meaningless as Turnbull and Solms and other psychoanalysts-on-the-run have argued. Far from it. We have steadfastly maintained that dreams were possibly of personal relevance. Their emotional salience could well be coupled to cognitive structures in ways that might be useful to examine clinically.

We only insisted that this approach could as well be undertaken by studying one's own dream journal, or in discussion with a friend, family member or open-minded therapist. By open

minded, we meant even those psychoanalysts who had already pitched overboard such quaint Freudian notions as repressed infantile wishes as dream instigators, and disguise censorship as the mechanism of dream bizarreness.

If this is what Turnbull and Solms really mean, let them say so. Is the Freudian dream theory of dream interpretation scientifically valid? Yes or no? But more specifically, can and should such dream features as bizarreness be used as starting points for the search for repressed infantile wishes in the hope of reducing symptoms? (Please note carefully that this question does not concern the possible clinical utility of free association.)

CONCLUSION

Carl Popper said that psychoanalysis was not a science because its hypotheses were untestable. In other words, psychoanalysis could not develop its own experimentalism to weed out error and to revise its theories. Turnbull and Solms seem to acknowledge that problem and now argue that neuropsychology offers the solution to psychoanalysis' experimental shortcomings. So far, so good.

But what Turnbull and Solms do not say is that since 1953, psychoanalysis has turned its back upon the experimental opportunity offered by laboratory sleep and dream research. Psychoanalysis has ignored sleep and dream research because it did not believe it was relevant and/or because it did not like having its shibboleths challenged. Sad to say, that is still true. Turnbull and Solms refuse to acknowledge the error of Freud's basic assumptions about dreams.

To summarize my commentary, let me repeat the following six questions to which the answers are either yes or no. Their reply could thus be limited to six words.

1. Do you believe that dreams are instigated by unconscious infantile wishes which are released from repression and threaten to invade consciousness. Yes or no? I say no.

2. Do you believe that dream bizarreness results from the defensive transformation, by disguise and censorship, of the unrepressed unconscious wishes that are released in sleep? Yes or no. I say no.

3. Do you believe, as Freud himself and as we do, that the preponderance of negative emotion in dreams is incompatible with wish fulfillment/disguise-censorship ideas? Yes or No? I say no.

4. Do you believe that the poor memory within dreams and following awakening from them is due to re-repression? Yes or No? I say no.

5. Do you believe that dream interpretation according to Freud is scientifically valid? Yes or No? I say no.

6. If you agree with me that the answer to any of the questions is no, do you also agree that psychoanalytic dream theory is erroneous and that its central tenets cannot be rescued by any conceivable scientific observation. Yes or no? I say yes.

The avowed breadth of psychoanalytic theory is hardly a virtue if it is wrong in its basic assumptions.

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BIG ISSUES, LITTLE ISSUES... AND NON-ISSUES

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THE BIG PICTURE

It was gratifying to see such clear support for the core ideas in our article, from virtually all of the commentators – with the major exception of Hobson (2007, this issue), about whom more below. Of course, there was diversity in the range of comments, and also in the degree of support for our ideas. However, we will begin by summarising their general thrust (leaving Hobson aside for the moment).

Firstly, there was widespread support for the idea that Freud's theoretical work may fruitfully be seen as presaging much of modern mental science. This support does not apply to all the *details* of his theoretical conclusions (see below). Rather, it seems to apply to Freud's overall conception of the nature and structure of the mind, and especially to his realisation that cognition cannot be understood without reference to emotion and motivation, and to non-conscious mental processes. Freud's approach is thus variously described by commentators as "prescient" (Huey et al., 2007), "very much alive in the twenty-first century" (Pribram, 2007), and likely to "undergo something of a renaissance" (McKay and Anderson, 2007). Every scientific field has intellectual debts to pay. Almost all our commentators seem to agree that modern psychology has now accumulated a large debt to Freud. If this represents acknowledgement of the debt, we have achieved one central goal of our Forum article.

A second strand in the commentaries was general agreement that psychoanalytic theories have not been adequately investigated. However, there also seems to be acceptance that those theories are now receiving the empirical attention they deserve, largely due to the advent of an affective neuroscience to complement its cognitive cousin. Moreover, these investigations are eliciting a range of surprisingly supportive findings. We are grateful to the commentators who supplemented our examples with additional findings (often from their own research) that are congruent with this general position. This applies to, for example, the vast body of Panksepp's work (2007, in this issue) on basic emotions, Feinberg's investigations of

delusional beliefs after brain lesion, the related work by MacKay and Anderson (2007), and the work on unconscious cognition by Bazan et al., 2007. The general opinion of the commentators seems to be one of cautious optimism: core psychoanalytic concepts are now enjoying converging lines of empirical support – though the evidential basis for definitive claims in this regard is still far from secure. The qualification that the existing empirical support is "a laudable prelude to more rigorous and direct tests in the future" (Dumer et al., 2007) is a position that we are happy to endorse.

It is important to emphasise, however, that the goal of our article was not merely to draw attention to ways in which neuropsychology is rediscovering, supporting and advancing classical psychoanalytic theories. We also argued that these same theories might be useful for a better understanding and investigation of traditional *neuropsychological* topics. Several commentators endorsed this view, especially in relation to anosognosia and confabulation (e.g. Feinberg, 2007; McKay and Anderson, 2007) and affect in general (Panksepp, 2007). In particular, there seems to be general support for our view that psychodynamic concepts greatly facilitate understanding of the role that motivational and emotional factors play in many complex neurocognitive disorders. Thus, the delusional beliefs of some neurological patients are readily conceptualised as "defensive behavioural patterns...aiming to protect the patient from unacceptable aspects of reality" (Gainotti, 2007). The commentators generally seem to "find this type of explanation very congenial" (McKay and Anderson, 2007), offering as it does many "fruitful areas for exploring neuropsychanalytic concepts" (Feinberg, 2007). It is pleasing to see how the role we envisage for emotion and motivation in these disorders is becoming central to the thinking of many leading neuropsychologists and behavioural neurologists. Needless to say, this must ultimately have important implications for our broader understanding of how the brain works.

In sum, there is widespread support for a rapprochement between psychoanalysis and neuroscience. This remains in the early phases of its

scientific development, though there already seems to be a great deal to be positive about. On the other hand, the general air of optimism does not extend to a number of *specific* psychoanalytic theories.

SPECIFICS

There was a tendency for some commentators to focus on highly specific theoretical proposals by Freud, and to ask (implicitly or explicitly) whether we wish to defend them. Some commentators even cast doubt on *every* specific theoretical proposal by Freud, while simultaneously endorsing his general approach to the mind (!): “the specific content of Freudian theory is no more acceptable to scientific psychology today than it was fifty years ago” (McKay and Anderson, 2007). In our view, Freud’s overall theoretical approach matters more than any specific conclusion, especially in view of the dearth of appropriate experimental investigations of those conclusions. The baby would be thrown out with the bathwater if we were to abandon a massively promising model of the mind simply because it has not been adequately tested. What we are arguing for, instead, is a fresh look at the specifics, using the new empirical and conceptual tools now available.

Heilman provides a case in point, focussing on our claims about the potential importance of emotion in understanding anosognosia. His commentary is a good example of precisely the sort of dialogue that we would like to engage with. It tackles a ‘bite-sized’ scientific issue, built on a growing empirical literature, where contrasting yet testable predictions flow from the competing theoretical perspectives. We are pleased that Heilman acknowledges that our account of the role of emotion in anosognosia, based on the idea of a right-lateralised emotion-regulation system, is more plausible than earlier accounts (e.g. Weinstein and Kahn, 1955). Critically, our damaged-emotion-regulation-system model addresses many concerns suggested over the years in relation to psychodynamic accounts of anosognosia (see Bisiach and Geminani, 1991 for a summary). We are also pleased that Prof. Heilman should provide an example of emotion-regulation from his personal experience, showing that he is “aware that people deny illness to reduce emotions such as fear, and moods such as depression”. However, he then offers a summary of evidence from neurological patients that he feels is *inconsistent* with our claim: principally cases in which low-level perceptual or cognitive impairments (grouped together as disorders of sensory feedback) have been shown to be important factors in anosognosia. He then suggests that these findings “partially discredit this emotion hypothesis of anosognosia”. We take no issue with the studies that he cites. We suggest only that the studies that *we* cite can also be accommodated in a more all-encompassing account of anosognosia.

We do not claim that our emotion-related proposal explains *all* aspects of anosognosia. So-called sensory feedback factors may be important in many instances. Indeed, the diversity of ways in which unawareness of deficit can manifest have become increasingly clear as this issue has been surveyed in larger samples (e.g. Bisiach et al., 1986; Marcel et al., 2004). However, we are of the opinion that emotional factors are over-riding in the majority of instances. The principal basis for our holding this position is the growing body of evidence that the manipulation of “emotional” factors (while holding “cognitive” factors constant) produces modifications in anosognosia, sometimes to the extent of temporary yet striking remission. These occur when, for example, a saline injection offers the patient a temporarily plausible explanation for their left-sided weakness (Ramachandran and Blakeslee, 1998, pp. 151-152; Ramachandran, 1996, pp. 130), when rings are placed on or off the patient’s fingers (Aglioti et al., 1996), when hands are labelled as being the patient’s own or that of a relative (Bottini et al., 2002), or when deficits are referred to in the first or third person (Marcel et al., 2004). These manipulations alter few, if any, of the low-level aspects of the patient’s sensory capabilities, but they strikingly alter the affective consequences of the world that the patient faces, and hence strikingly affect the magnitude of denial of deficit.

A second issue which leads us to conclude that emotion-related factors are central in anosognosia is that in some cases the denial of deficit is so *frankly* delusional, involving such clear repudiation and distortion of clearly acknowledged information, that one cannot explain the full clinical picture in terms of a lack of low-level knowledge. Many of the cases cited above fall into this category (Aglioti et al., 1996; Bottini et al., 2002; Ramachandran, 1994). To take another example, the experiment where Ramachandran et al. (1997) places an obliquely aligned mirror on the right of the patient, so that objects placed on their left side are reflected to appear in their ‘good’ visual field: were low-level sensory deficits the only factors at play in such cases, one might expect the patient to say: “Ah... *there* the object is. I couldn’t see it before, because I can’t look towards the left – thanks for reflecting it across for me” (see Turnbull, 1997; p. 710). Instead, these patients *continue* to behave irrationally towards the object, and even attempt to reach into or behind the mirror – apparently jettisoning a life-time of experience with the physics of mirrors. As is the case with manipulations involving rings, the hands of relatives, and saline injections (Aglioti et al., 1996; Bottini et al., 2002; Ramachandran, 1994), these patients do have access to sensory information, but do not use the available information in a sensible and rational way – often selecting piecemeal from the evidence, with a striking tendency to ignore things that might remind them of the inconvenient reality of their deficit.

We therefore suggest that these emotional factors are under-investigated, but potentially pivotal, determinants of anosognosia – in conjunction with other factors. Heilman's questions therefore provide the setting for an ordinary scientific debate, based exclusively on empirical evidence, about the importance of emotion in explaining a specific neuropsychological deficit. This historical transformation in the debate from speculative generalities ('Was Freud right?') to empirical specifics ('How important is emotion in explaining denial of deficit?') is surely a step in the right direction. For this reason, we were rather taken aback by Hobson's remarkable claims that our work is "not only pseudoscientific... but actively antiscientific".

Continuing with the theme of "specifics", we might also briefly consider Huey et al.'s (2007, this issue) arguments about the regulation of drives after frontal lesions, which they use as an example of "parts of Freudian theories that have been accepted beyond their supporting evidence". Their arguments to the effect that frontal cortex plays a role in the drive state that is different from the classical "inhibition" account are interesting. The simplest explanation for the relevant data might be that the regulation of drive states is multi-componential in nature, as we have argued elsewhere for several cortical emotion-regulation systems (Kaplan-Solms and Solms, 2000; Turnbull et al., 2002, 2005). However, we have no axe to grind about particular notions of inhibition. We are happy to accept the more general point that Huey et al. make about Freudian theory, namely that it is likely to be wrong in several specific points of detail. But this must surely also apply to *every* general theoretical approach, in any scientific field.

To be clear, we do not wish to defend Freudian theory in any specific respects, except where it appears to have specific applicability to contemporary neuropsychological problems, and then only to the extent that this leads to direct empirical investigations. However, by the same token, we should not *reject* any specific claim *before* it has been pertinently tested. In our article, we drew attention to some specific, central claims that *are* now receiving empirical support. If further research reveals that these or other claims are incorrect in matters of detail, or even if they turn out to be entirely unfounded, then so be it. Also, we want to encourage our colleagues to make use of these claims in their investigations into more traditional neuropsychological topics which seem likely to benefit from them, especially as regards the role of emotion/motivation. We suspect that all neuropsychologists would agree that the neurocognitive basis of anosognosia and confabulation (for example) is currently poorly-understood, and would welcome a systematic investigation of novel proposals which offer a cogent framework for explaining them.

Happily, we do not need to defer *all* discussion regarding the accuracy of specific Freudian theories to future research. In addition to the above-mentioned, limited examples, there is a further topic where the body of evidence is sufficiently well-developed to speak directly to several detailed empirical questions. This is the topic of dreams.

HOBSON'S QUESTIONS

Hobson (2007, this issue) asks us to defend six specific theoretical claims about dreams. We note that each of his questions begin with the phrase "Do you believe...?". Science is, of course, not a matter of *belief*, but of evidence, so not all of his questions can be answered in the simple yes/no manner that he requests. We also note that Hobson sometimes wants us to swear allegiance to a parodied version of Freud's dream theory, when our goal has only been to re-investigate some of his more general claims. Nevertheless, we will do our best to tackle each of Hobson's questions (2007, this issue):

1. "Do you believe that dreams are instigated by unconscious infantile wishes which are released from repression and threaten to invade consciousness? Yes or no? I say no."

The main problem with this question is that we do not really know what the neural correlate of a "repressed infantile wish" might be. The question therefore needs some speculative translation into modern neuropsychological terms. Let's try: if "*repression*" in this context refers to the inhibitory and regulatory influences on cognition that are normally contributed (during waking life) by the pre-frontal cortical structures that are deactivated during dreaming sleep, then we disagree with Hobson and say YES to this part of the question. The available evidence unequivocally supports the view that dorsolateral prefrontal cortex is deactivated during sleep (e.g. Braun et al., 1997).

Similarly, if "*wishes*" are equivalent to the motivational mesocortical-mesolimbic dopaminergic influences on cognition that are unusually active during dreaming sleep, in contrast to all the other midbrain monoamine influences; then, especially in view of the deactivated executive control just mentioned, we again disagree and say YES to this second part of the question. The available evidence strongly supports the claim that this motivation/emotion system is central to initiating the dream process (Gottesman, 2005; Nofzinger et al., 1997; Solms, 1997b, 2000).

Lastly, if "*infantile*" refers to the fact that such influences are derived from systems that are fully active from birth, which in turn *might* imply that early experiences provide the basic representational schemata they activate (e.g., Solms and Turnbull,

2002), then we say to this part of the question: a tentative YES, if we have to choose, though one cannot really answer the question as framed on the available neurodevelopmental evidence.

Hobson's second question (2007, this issue) again refers to repressed wishes, but it goes further:

2. "Do you believe that dream bizarreness results from the defensive transformation, by disguise and censorship, of the unrepressed unconscious wishes that are released in sleep? Yes or no. I say no."

As Yu (2000, 2003) has shown, Hobson misunderstands this aspect of Freudian dream theory. Freud's view was that dream cognition is bizarre for a *variety* of reasons, *one* of which is that the defensive functions of the ego are weakened during sleep. Thus, to answer Hobson's second question (2007, this issue), we need to both translate and correct it: Do we believe that dream bizarreness results in part from the degradation during sleep of those executive functions that normally transform crude instinctual strivings into reality-constrained action programs? As discussed above, it is well-established that executive abilities are impaired during dreaming (see Braun et al., 1997; Solms, 1997b, 2000). Of course, it is not clear to what extent, and how, an absence of executive functions, combined with powerful emotions, might produce what Freud called 'disguise and censorship', but we are confident that the contents of consciousness would be greatly changed by this combination of psychological factors. Whether these changes would coincide exactly with the process that Freud called "censorship" is another matter. Thus, we would like to leave our answer to this question open, with the additional caveat that, like Freud, we suspect that factors other than disguise and censorship might also contribute to dream bizarreness (see Yu, 2000).

3. "Do you believe, as Freud himself and as we do, that the preponderance of negative emotion in dreams is incompatible with wish fulfillment/disguise-censorship ideas? Yes or No? I say no."

Freud initially believed that the preponderance of negative emotions in dreams was not only compatible with disguise-censorship, but actually *due* to it, in the sense that any failure to defensively transform crude instinctual impulses caused negative emotion (Freud, 1900). At that stage, Freud believed that the primary instinctual influences in question were always appetitive ('sexual') and that the negative emotions were secondary, in that they were signals of failure of executive control. Subsequent investigations led him to revise this theory, and suggest that negative emotion in dreams may also reflect the direct influence of non-appetitive ('destructive') instincts (Freud, 1940).

So, do we believe that the preponderance of negative emotion in dreams is incompatible with Freud's theories? To the extent that the available neuropsychological evidence speaks to the question, our answer is NO, in relation to both theories (perhaps Hobson meant to say YES here?). Because executive (pre-frontal) control of undiminished appetitive (dopaminergic) influences is *indeed* weakened in dreams, and because the latter (unlike other instinctual and emotional systems) *do* seem to be uniquely necessary for the generation of dreams (Solms, 1997b, 2000), Freud's first theory is not unreasonable. But because 'negative' instinctual systems, centered on the amygdala and anterior cingulate gyrus (responsible for fear, rage and separation distress) are indeed directly activated in dreams (Braun et al., 1997; Maquet et al., 1996; Panksepp, 1998), Freud's second theory, too, is not unreasonable.

4. "Do you believe that the poor memory within dreams and following awakening from them is due to re-repression? Yes or No? I say no."

To the extent that 're-repression' might plausibly be translated as 're-activation of pre-frontal control', with all the attendant state-dependent implications, we disagree with Hobson and say YES. The re-engagement of executive control is surely likely to 're-set' many aspects of cognition, memory included. A trite example might be the well-known memory failures experienced by binge-drinkers (White, 2003; White et al., 2004), who are often amnesic for episodes from the previous evening – a period during which they were clearly both *uninhibited* and *conscious*.

5. "Do you believe that dream interpretation according to Freud is scientifically valid? Yes or No? I say no."

To the extent that dreams are a distorted and degraded form of thinking, the meaning of which (or motivation behind which) is not immediately apparent to the dreamer, some form of 'interpretation' might indeed be possible. Incidentally, the same applies to *other* instances of degraded/distorted cognition, such as semantic paraphasias and amnesic confabulations. The (manifest/declarative) products of degraded language and memory systems are not random. Rather, they are derived from the specific upstream (latent/non-declarative) motivations and cognitions that generated them, as can often be inferred ('interpreted', if you will) from the context. The same applies to dreams.

We are not, of course claiming that *all* motivational precursors can be understood on the basis of the port-distortion relics. The proportion of interpretable material may be rather modest, and there may be many occasions when we are *not* able

to infer from the manifest dream content the motivational primitives. The process would be akin to establishing the likely target-word in a semantic or phonemic paraphasia, which can often be established with some certainty, but where the distortion process is sometimes so impenetrable that the utterances must be classified as jargon aphasia. Thus, in principle, we believe that a process of reverse engineering is viable – though we concede that an exact science is not currently (and possibly never will be) available.

6. “If you agree with me that the answer to any of the questions is no, do you also agree that psychoanalytic dream theory is erroneous and that its central tenets cannot be rescued by any conceivable scientific observation. Yes or no? I say yes.”

We disagree with the whole ‘fundamentalist’ approach to science that generates such questions, and find it remarkable that Hobson should suggest that Freud’s dream theory might not be rescued by “any conceivable scientific observation”. We have cited several lines of neuroscientific evidence which we and others regard as entirely consistent with the classical psychoanalytic theory of dreams. However, Hobson seems to avoid mentioning these findings, perhaps because they run counter to aspects of the theory that his own group developed some time ago, and explicitly touted as anti-Freudian (Hobson and McCarley, 1977). It is unfortunate that Hobson does not seem to be able to grasp the ways in which the more recent data *do* suggest a central role for motivation in dream generation. Dare we surmise that he is so resistant to this body of evidence *because* it might support a Freudian idea?

Readers may be interested to know that Hobson put his Question 6 to a formal Oxford-rules debate at the most recent Tuscon Consciousness meeting (2006). After listening to Hobson’s arguments for the motion, and Solms’ against it, the vote went overwhelmingly against Hobson (by roughly a 2:1 ratio). We feel justified in rejecting it here, again, despite our agreeing with him on at least some aspects of his earlier questions. Hobson seems keen to offer a greatly-simplified version of Freud’s dream theory, ask us to ignore all the modern scientific evidence in its favour, invite us to agree that any *single* component of the parody is open to question, and then consider this necessary grounds for rejecting the *entire* argument. This is not a useful approach to tackling any scientific question, much less one as complex as the neuropsychology of dreams.

CONCLUSION

Reading the commentaries has been an edifying experience. It has confirmed our sense of optimism

that common ground can be found between the psychoanalytic and neuroscientific theoretical traditions, driven by a shared interest in the nature of the mind, and more specifically a growing interest in the ways that emotion and motivation shape cognitive processes. The past century has seen an unfortunate legacy of analytic over-speculation, coupled with empirical under-investigation. However, it seems to us that a corner has been turned, not least because modern neuropsychology now has both the interest and the tools to address many of the complex issues that were so long the preserve of psychoanalysis. This can usefully be coupled with a role for analytically-inspired ideas in shaping some aspects of neuropsychological research and theory. However, writing this response also reminds us of the invisible hurdle of terminology, where problems of translation can hinder a progressive discussion between disciplines with a common cause: the daunting challenge of understanding the human mental apparatus in all its complexity.

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