The World Journal of Biological Psychiatry, 2005; 6(1): 0–00

REVIEW

Cognitive neuropsychiatry: Conceptual, methodological and philosophical perspectives

JAKOB HOHWY[1] & RABEN ROSENBERG[2]

[1]*Department of Philosophy, Aarhus University, Aarhus, Denmark, and* [2]*Centre for Basic Psychiatric Research, University Psychiatric Hospital in Aarhus, Skovagervej 2, DK-8240 Risskov, Denmark*

Abstract

Cognitive neuropsychiatry attempts to understand psychiatric disorders as disturbances to the normal function of human cognitive organisation, and it attempts to link this functional framework to relevant brain structures and their pathology. This recent scientific discipline is the natural extension of cognitive neuroscience into the domain of psychiatry. We present two examples of recent research in cognitive neuropsychiatry: delusions of control in schizophrenia, and affective disorders. The examples demonstrate how the cognitive approach is a fruitful and necessary supplement to the otherwise successful biological psychiatry paradigm, which tend to bypass the cognitive level. Philosophy concerns some of the core concepts involved in psychiatric illness, particularly concerning rationality, thought and action, reality testing, and the self. We present concrete examples that illustrate how philosophical conceptual tools can be particularly important for the construction and interpretation of the cognitive models relevant to the understanding of psychiatric illness. We conclude that cognitive neuropsychiatry is a fruitful and necessary supplement to biological psychiatry. Furthermore, cognitive neuropsychiatry itself may benefit significantly from employing philosophical conceptual tools in the interpretation and construction of its cognitive models. The cognitive and philosophical approaches may thus be further steps towards a scientific psychopathology.

Key words: *Neuropsychiatry, cognitive neuropsychiatry, philosophy, delusions of control, affective disorder*

Introduction

Cognitive neuropsychiatry is a new branch of psychiatric research. It brings together cognitive and neurobiological approaches to the study of mental disorders. It promises to advance our understanding of psychiatric illness by finding the right theoretical and methodological balance between otherwise successful paradigms, such as biological psychiatry and cognitive psychology.

We review conceptual and scientific aspects of cognitive neuropsychiatry and present examples of research in this field. The examples demonstrate that the cognitive approach is a useful and necessary supplement to neuropsychiatry.

Philosophy may be especially important for the study and understanding of psychiatric illnesses that often touch on philosophical notions concerning rationality, thought and action, reality testing, and the self. We present some concrete examples that illustrate the relevance of philosophical analysis to psychiatric research.

We conclude that there is reason to believe that cognitive neuropsychiatry will be a fruitful and necessary supplement to biological psychiatry, and that philosophical analysis will be a useful aid to cognitive neuropsychiatry. Both the cognitive and the philosophical approaches may therefore constitute further steps towards a scientific psychopathology.

Neuropsychiatry and cognitive science

Neuropsychiatry is claimed to be a discipline for the future (Sachdev 2002) and several recent papers have focused on the integration of psychiatry, neurology and neuroscience (Cowan et al. 2000; Martin 2002; Yudofsky et al. 2002), thereby broadening the concept of biological psychiatry with its primary focus on neurobiology. To influential

Correspondence: Raben Rosenberg, Centre for Basic Psychiatric Research, University Psychiatric Hospital in Aarhus, Skovagervej 2, DK-8240 Risskov, Denmark. Tel: 45 7789 3511. Fax: 45 7789 3549. E-mail: rro@psykiatri.aaa.dk

(Received 28 April 2003; accepted 21 October 2004)

ISSN 1562-2975 print/ISSN 1814-1412 online
DOI: 10.1080/15622970510029867

scientists, such as Kandel, modern neuroscience theory and methodology represent a new framework for psychiatry (Kandel 1998) that hopefully will lead to better understanding and treatment of psychiatric disorders, based on dysfunction of specific genes, molecules, neuronal organelles and specific neuronal systems.

Cognitive science is a discipline that attempts to understand the mind in terms of information processing, that is, representational structures in the brain and computational procedures that operate on them. With the development over the last 30 years of sophisticated neuroimaging and neurophysiological methods for studying the living brain, neuroscience has become a core component of cognitive science. Now the dominating science of the mind is the amalgamation of neuroscience and cognitive science in *cognitive neuroscience* – optimistically called the 'biology of the mind' (Gazzaniga et al. 1998).

Cognitive neuropsychiatry

Cognitive neuropsychiatry (CNP, hereafter) is the extension of the methods of cognitive neuroscience and cognitive neuropsychology to the study of psychiatric disorders. It was advanced as a scientific discipline in its own right in the beginning of the 1990s, and got its own journal (*Cognitive Neuropsychiatry*) in 1996. Instrumental in promoting CNP were Hadyn Ellis, Anthony David, Peter Halligan and John Marshall (see, e.g., David 1993). The state of the art in terms of scientific results has been elegantly reviewed (Halligan et al. 2001).

CNP attempts to understand psychiatric disorders as disturbances to the *normal function* of human cognitive organisation and it attempts to link this functional framework to relevant brain structures and their pathology. The focus is on symptoms rather than syndromes or diagnoses because phenomenologically based diagnoses rarely lend themselves to a differentiated functional analysis or correlation with specific brain structures. Thus, insight into normal psychological processes is essential for a scientific analysis of psychiatric symptomatology. CNP has a somewhat broader approach to the analysis of psychiatric disorders than neuropsychiatry because it focuses on a cognitive and often more philosophical analysis of human action and behaviour (see below). CNP and philosophical analysis could therefore be a fruitful and necessary supplement to neuropsychiatry, something that allows us to better understand *how* and *why* biological malfunctions and biological damage give rise to mental disorders.

Examples of research in cognitive neuropsychiatry

Current research in CNP includes studies of the various positive symptoms found in schizophrenia such as auditory hallucinations, persecutory delusions and passivity experiences, delusions of misidentification; as well as autism, neuropsychiatric symptoms and signs associated with motor control, and emotional and affective disorders. We now briefly review two sets of studies in order to demonstrate how taking a cognitive approach enables us to better understand how psychiatric disorders arise from malfunctions and damage to brain activity.

Delusions of control

Delusions of control is a first-rank Schneiderian symptom of schizophrenia. Patients believe that actions they perform are really performed by other agents. It is classed with other passivity experiences, such as thought withdrawal and thought insertion. It is possible to begin to explain this delusion in terms of the general functioning of our sensorimotor system and our awareness of it (Frith et al. 2000).

The account begins with cognition, or information-processing at the most basic level: when we issue a motor command, an efference copy of it is also issued. This copy serves to predict how the state of the system will change given the performance of the movement, as well as what the sensory feedback will be given the new state (Wolpert et al. 2000). The first prediction is used in comparisons with the desired state and allows rapid error correction, before re-afferent feedback is available (Blakemore et al. 2002). The second prediction probably serves to attenuate activity in the inferior parietal cortex associated with the sensory consequences of our own movement (Blackwood et al. 2000; Blakemore et al. 1998). It may be that the feeling of being in control of our movements is associated with activity in this area: when there is activity it is not our movement, when activity is attenuated via the prediction, we can tell it is our movement (Frith et al. 2000).

If there is no awareness of the predicted limb position, then the subject will be aware of the goal, of the intention to move and of the movement having occurred, but not of the exact specification of the movement. The subject may thus be unaware of having initiated that movement.

Delusions of control may therefore arise when there is an abnormality in the production of forward models of movement such that the patient feels that, though he or she intended the movement, it was initiated by some external force. This account predicts that patients should be relatively poor at

rapid error control, in the absence of visual feedback, and this has been experimentally confirmed (Frith et al. 1988). Spence et al. (1997) scanned delusional schizophrenic subjects while they performed joystick movements. During the scan they experienced delusions of control. As compared to normal controls and other groups of schizophrenic subjects, this group showed overactivity in the inferior parietal cortex, consistent with the claim that activity in this area is normally attenuated in expectation of the sensory consequences of one's own movements. This overactivity was associated with relative underactivity in the dorsolateral prefrontal cortex that may be an area involved in modulating activity in the inferior parietal cortex.

In a recent study (Blakemore et al. 2003), delusions of control were induced in normal subjects. Hypnotised subjects were scanned in three conditions: while actively raising their arm, while passively having their arm raised, and while actively raising their arm but under the hypnotic suggestion that it was being passively raised. In the last condition they believed they were not in control, even though they did actually raise their arm, and this was associated with increased activity in the inferior parietal cortex.

This empirically tested cognitive approach to delusions of control yields a conceptual framework for beginning to understand what the observed brain activity means, and how it may give rise to delusions of control rather than other disorders. Without this *cognitive* account, the brain activity (e.g., reduction of activity in inferior parietal cortex) in itself would be difficult to make sense of.

Affective disorder

A major task for psychiatric nosology has been to classify and to define subgroups of affective disorders. As evidenced by current classification systems, psychopathological description of symptoms evaluated cross-sectionally or during lifetime course have been the most influential approaches. Although primarily defined by mood changes, affective disorders are characterized by significant impairment of cognitive functions. Numerous neuropsychological studies of unipolar (Ravnkilde et al. 2002) and bipolar disorders (Chowdhury et al. 2003) have revealed abnormal functioning of a variety of cognitive functions, including attention, memory, psychomotor speed and executive functions, indicating that cognitive deficits are not just epiphenomena but essential aspects of the disorders. Neuroimaging techniques are extensively used to characterize the brain mechanisms underlying the clinically important cognitive dysfunctions in depression (Videbech 1997; Videbech 2000). The classical symptoms of guilt feeling, self-reproaches, lack of initiative, and concentration difficulties have been linked to changes in normal neuronal activation patterns (Davidson et al. 2002). Important pathways include the orbitofrontal cortex, dorsolateral prefrontal cortex, the anterior cingulate, hippocampus and amygdala (Davidson et al. 2002; Videbech et al. 2001), and dysfunction in different parts of this circuitry are indicated by several studies.

Furthermore, precipitation of depressive episodes by life events are interpreted in terms of stress related to cognitive coping strategies and neuroendocrinological reactions (Gould et al. 1999; Nestler et al. 2002). Finally long term deviations of cognitive functions in depressive patients have been correlated to atrophy of hippocampus (Lee et al. 2002; MacQueen et al. 2003).

The clinical efficacy of antidepressant drugs is suggested to involve neurogenesis in hippocampus (Kempermann 2002) and by changing serotonergic and noradrenergic neurotransmission, antidepressant drugs may influence stress responses, cognitive functions and mood. Cognitive therapy is efficacious in treating mild to moderate depressive episodes (Scott 2001) and increasing evidence has been obtained for the importance of the treatment modality in relapse prevention (Jarrett et al. 2001), also on the potential benefits of combining cognitive therapy with medication (Arean and Cook 2002). In a recent PET study, cognitive therapy as well as paroxetine were suggested to induce clinical recovery by modulating the functioning of specific sites in limbic and cortical regions (Goldapple et al. 2004).

In conclusion, analysis of cognitive functioning in affective disorders has transpired as a fruitful approach that might lead to a comprehensive theory integrating clinical phenomenology, neuropsychology, neuroimaging, neuropathology and neuropharmacology.

Philosophical perspectives on cognitive neuropsychiatry

Psychiatric disorders hold particular interest for philosophy because they concern core philosophical concepts such as belief, perception, experience, reality, self, other, emotion, and consciousness. Specifically, the construction and interpretation of cognitive models in CNP may benefit from philosophical analyses of the involved concepts—at least to the extent these analyses are apt for application in ongoing empirical research. We now present some concrete examples of philosophical analysis of psychiatric disorder. When there are relatively well-developed cognitive models, as in the case of delusions of control, philosophical analysis may be

needed to address outstanding questions. And philosophical analysis will also be needed in cases, such as affective disorder, where the cognitive approach is in its beginning phases.

Delusions and rationality

There may often be an experiential factor in the formation of delusional beliefs (for an overview, see Davies et al. 2001). For example, in the case of delusions of control there may be the unusual and distressing experience that one's intended bodily movements are initiated by another agent. Some cognitive process involving reasoning must then be active in the production of the irrational delusional belief that the movement is in fact initiated by someone else. It is an outstanding question how this process goes (Davies et al. 2000). We need to explain why the alien agent hypothesis presents itself (rather than the more probable hypothesis that something has gone wrong in the patient's brain); why the subject begins to believe the hypothesis; and why this belief is tenaciously maintained in spite of what everyone else says.

Clearly, our explanations in this area depend to a large degree on which concept of rationality we chose to operate with, and on our understanding of the normal processes for belief revision. Philosophers operate with notions of *ideal* and *realistic* rationality, that is, the rules of rationality that people should (but in general cannot) satisfy to be completely rational, versus the rules of rationality that people actually employ, partly as a consequence of the fact that they are not ideally rational (Harman 1986).

Our conceptualisation of pathological belief formation will be influenced by our conception of rationality. Do delusional beliefs arise as rational or as irrational responses to the supposed unusual experiences? Is the experience of such a character that the alien agent hypothesis is in fact rational, or is it necessary to posit a further deficit that results in a preference for irrational, bizarre hypotheses? If some delusions arise as rational responses to unusual experiences, then the prediction is that healthy subjects that have such experiences will develop delusional states. The mentioned study by Blakemore et al. (2003) may bear out this prediction (at least if the subjects' reported states of unintentionality are comparable to delusions of control, and unless hypnosis induces the requisite kind of irrationality).

It may also be necessary to distinguish more departments of rationality than the merely rule-based or procedural. Sometimes, it is the *content* of a propositional attitude like a belief or a desire that appears irrational and pathological, never mind the procedural rules (whether ideal or realistic) that were used to adopt it (Lewis 1986); consider for example the bare desire for a saucer of mud (Anscombe 1957). Further conditions on rationality may also be necessary, for example, disturbances to the sense of egocentricity or self (that *I* am the agent or thinker behind these actions and thoughts) may play a role in the production of irrational belief and the ability to reason coherently (as in delusions of control and thought insertion) (see Frith (1992), for discussion; see Gold et al. (2000)).

These *philosophical* analyses suggest the possibility of a range of cognitive models to account for delusion formation. Delusions of various kinds may arise as the results of disturbances to cognitive systems concerning (a) content formation, and/or (b) procedural rules of belief formation, and/or (c) other cognitive systems, such as those involved in egocentricity (see also Gerrans (2001)).

Reality testing

It seems plausible that reality testing is part of what makes a knowledge gathering mechanism reliable. Reality testing is required when initial beliefs are formed in sub-optimal conditions (bad lighting, background noise, etc), or when they conflict with strongly held prior beliefs. When it comes to sensory modalities such as vision, touch and hearing, we have a range of procedures for reality testing that can override poor initial belief formation. We can turn on the light, feel with our hands, move closer to the source of sound, and so on. If these ordinary procedures for reality testing converge on the same result, then we come to believe the content in question, and the beliefs would be normally be justified.

This commonsense *epistemological* notion of reality testing may connect with the hypothesis that some delusions arise as responses to unusual experiences, caused by biological deficit or damage. These experiences may occur in sensory modalities where we do not have such ready procedures for reality testing, for example, experiences of sensorimotor feedback (also of speech production), including awareness of the output of discrepancy monitors as mentioned in the above account of delusions of control; awareness of introspection of thought, and awareness of emotional and affective states. If there are no or very limited strategies for reality testing, then it may be impossible to override and revise the beliefs formed on the basis of those unusual experiences. Every time the subject tries to reality test, the same result obtains, and there is no alternative route to reality test experiences in these

modalities. This would go some way towards explaining the tenacity with which patients hold on to delusional beliefs that incorporate an unusual experience. On this view, there is no specific reasoning deficit or bias involved in delusion formation. If it was possible to give people unusual experiences in these sensory modalities, then the hypothesis would be supported by finding that normal subjects with no reasoning deficits or biases form delusional beliefs (Blakemore et al. (2003) may be an example; see Hohwy and Rosenberg (submitted) for more detailed discussion).

Decision, reasoning and affect

The exploration of the cognitive aspects of affective disorders (see above) is still in its beginning phases, and intriguing philosophical questions arise. It is plausible that affective and emotional states play a role in decision processes (Bechara et al. 1994). This suggests that affective disorders are related to realistic, rather than ideal rationality, as discussed above. It may be that defective processing of affective and emotional states play a role in producing the cognitive deficits seen in affective disorders. On the other hand, it may also be that the cognitive deficits cause failures in the way affective states are brought to bear on decision making and belief formation. Deciding on these issues again partly depend on how we analyse and organise philosophical notions of rationality, decision, belief and emotion.

A scientific psychopathology?

The move away from Freudian psychoanalysis to a biologically based psychiatry in the last century has made psychiatry a more scientific enterprise, even though most new drugs have appeared due to serendipity and not as a consequence of insight into disease processes. Thus, a majority of current theories of mental disorders have been developed secondary to the pharmacodynamics of drugs, e.g., theories of neurotransmitter deficiencies for schizophrenia and depression. The cognitive approach taken by cognitive neuropsychiatry may help gain a more direct focus on psychiatric disorders because it goes from symptoms to brain activity via cognitive models of normal psychological functioning. Furthermore, philosophical analysis may be a fruitful supplement to cognitive neuropsychiatry. Cognitive neuropsychiatry and philosophy may thus constitute further steps towards a scientific psychopathology.

References

Anscombe GEM. 1957. Intention. Oxford: Blackwells.

Arean PA, Cook BL. 2002. Psychotherapy and combined psychotherapy/pharmacotherapy for late life depression. Biol Psychiatry 52:293–303.

Austin DW, Richards JC. 2001. The catastrophic misinterpretation model of panic disorder. Behav Res Ther 39:1277–91.

Bandelow B, Zohar J, Hollander E, Kasper S, Moller HJ. 2002. World Federation of Societies of Biological Psychiatry (WFSBP) guidelines for the pharmacological treatment of anxiety, obsessive-compulsive and posttraumatic stress disorders. World J Biol Psychiatry 3:171–99.

Bechara A, Damasio AR, Damasio H, Anderson SW. 1994. Insensitivity to future consequences following damage to human prefrontal cortex. Cognition 50:7–15.

Blackwood NJ, Howard RJ, ffytche DH, Simmons A, Bentall RP, Murray RM. 2000. Imaging attentional and attributional bias: An fMRI approach to the paranoid delusion. Psychol Med 30:873–83.

Blackwood NJ, Howard RJ, Bentall RP, Murray RM. 2001. Cognitive models of persecutory delusions. Am J Psychiatry 158:527–39.

Blakemore SJ, Wolpert DM, Frith CD. 1998. Central cancellation of self-produced tickle sensation. Nat Neurosci 1:635–40.

Blakemore SJ, Wolpert DM, Frith CD. 2002. Abnormalities in the awareness of action. Trends Cogn Sci 6:237–42.

Blakemore SJ, Oakley DA, Frith CD. 2003. Delusions of alien control in the normal brain. Neuropsychologia (in press).

Chowdhury R, Ferrier IN, Thompson JM. 2003. Cognitive dysfunction in bipolar disorder. Curr Opin Psychiatry 16:7–12.

Churchland PS, Sejnowski TJ. 1999. Perspectives on cognitive neuroscience. In: Gazzaniga MS, editor. Cognitive neuroscience: A reader. Malden, MA: Blackwell.

Clark D. 1986. A cognitive approach to panic. Behav Res Ther 24:461–70.

Cowan WM, Harter DH, Kandel ER. 2000. The emergence of modern neuroscience: some implications for neurology and psychiatry. Annu Rev Neurosci 23:343–91.

David AS. 1993. Cognitive neuropsychiatry? Psychol Med 23:1–5.

Davidson RJ, Pizzagalli D, Nitschke JB, Putnam K. 2002. Depression: Perspectives from affective neuroscience. Annu Rev Psychol 53:545–74.

Davies M, Coltheart M. 2000. Introduction: Pathologies of belief. Mind Lang 15:1–46.

Davies M, Coltheart M, Langdon R, Breen N. 2001. Monothematic delusions: Toward a two-factor account. Phil Psychiatr Psychol 8:133–58.

Elliott R. 1998. The neuropsychological profile in unipolar depression. Trends Cogn Sci 11:447–54.

Frith CD. 1992. The cognitive neuropsychology of schizophrenia. Hillsdale, NJ: Lawrence Erlbaum Ass.

Frith CD, Done DJ. 1988. Towards a neuropsychology of schizophrenia. Br J Psychiatry 153:437–43.

Frith CD, Blakemore SJ, Wolpert DM. 2000. Abnormalities in the awareness and control of action. Phil Trans R Soc London B Biol Sci 355:1771–88.

Furmark T, Tillfors M, Marteinsdottir I, Fischer H, Pissiota A, Langstrom B, Fredrikson M. 2002. Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. Arch Gen Psychiatry 59:425–33.

Gazzaniga M, Ivry RB, Mangun GR. 1998. Cognitive neuroscience: The biology of the mind. New York: WW Norton.

Gerrans P. 2001. Delusions as performances failures. Cogn Neuropsychiatry 3:161–73.

AQ1

Gold I, Hohwy J. 2000. Rationality and schizophrenic delusion. Mind Lang 15:146–67.

Goldapple K, Segal Z, Garson C, Lau M, Bieling P, Kennedy S, et al.. 2004. Modulation of cortical-limbic pathways in major depression: Treatment-specific effects of cognitive behavior therapy. Arch Gen Psychiatry 61:34–41.

Gorman JM, Kent JM, Sullivan GM, Coplan JD. 2000. Neuroanatomical hypothesis of panic disorder, revised. Am J Psychiatry 157:493–505.

Gould E, Tanapat P. 1999. Stress and hippocampal neurogenesis. Biol Psychiatry 46:1472–9.

Green RL, Gazzaniga MS. 2001. Linking neuroimaging and cognitive neuropsychiatry. Cogn Neuropsychiatry 6:229–34.

Halligan PW, David AS. 2001. Cognitive neuropsychiatry: Towards a scientific psychopathology. Nat Rev Neurosci 2:209–15.

Harman G. 1986. Change in view. Cambridge, MA: MIT Press.

AQ2 Hohwy J, Rosenberg, R. Submitted. Reality testing, unusual experiences and delusions of control.

Jarrett RB, Kraft D, Doyle J, Foster BM, Eaves GG, Silver PC. 2001. Preventing recurrent depression using cognitive therapy with and without a continuation phase: A randomized clinical trial. Arch Gen Psychiatry 58:381–8.

Kandel ER. 1998. A new intellectual framework for psychiatry. Am J Psychiatry 155:457–69.

Kempermann G. 2002. Regulation of adult hippocampal neurogenesis: Implications for novel theories of major depression. Bipolar Disord 4:17–33.

Lee AL, Ogle WO, Sapolsky RM. 2002. Stress and depression: possible links to neuron death in the hippocampus. Bipolar Disord 4:117–28.

Lewis D. 1986. On the plurality of worlds. Oxford: Oxford University Press.

Libet B, Gleason CA, Wright EW, Pearl DK. 1983. Time of conscious intention to act in relation to onset of cerebral activity (readiness-potential). The unconscious initiation of a freely voluntary act. Brain 106:623–42.

MacQueen GM, Campbell S, McEwen BS, MacDonald K, Amano S, Joffe RT, Nahmias C, Young LT. 2003. Course of illness, hippocampal function, and hippocampal volume in major depression. Proc Natl Acad Sci USA 100:1387–92.

Martin JB. 2002. The integration of neurology, psychiatry, and neuroscience in the 21st century. Am J Psychiatry 159:695–704.

Nestler EJ, Barrot M, Eisch AJ, Gold SJ, Monteggia LM. 2002. Neurobiology of depression. Neuron 34:13–25.

Ravnkilde B, Videbech P, Clemmensen K, Egander A, Rasmussen NA, Rosenberg R. 2002. Cognitive deficits in major depression. Scand J Psychol 43:239–51.

Sachdev PS. 2002. Neuropsychiatry – A discipline for the future. J Psychosom Res 53:625–7.

Scott J. 2001. Cognitive therapy for depression. Br Med Bull 57:101–13.

Spence SA, Brooks DJ, Hirsch SR, Liddle PF, Meehan J, Grasby PM. 1997. A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). Brain 120:1997–2011.

Spence SA, Frith CD. 1999. Towards a functional anatomy of volition. J Consc Stud 6:11–29.

van Balkom AJLM, van Oppen P, Vermeulen AWA, Van Dyck R, Nauta MCE, Vorst HCM. 1994. A meta-analysis on the treatment of obsessive compulsive disorder: A comparison of antidepressants, behavior, and cognitive therapy. Clin Psych Rev 14:359–81.

van Balkom AJ, Bakker A, Spinhoven P, Blaauw BM, Smeenk S, Ruesink B. 1997. A meta-analysis of the treatment of panic disorder with or without agoraphobia: A comparison of psychopharmacological, cognitive- behavioral, and combination treatments. J Nerv Ment Dis 185:510–6.

Videbech P. 1997. MRI findings in patients with affective disorder. A meta-analysis. Acta Psychiatr Scand 96:157–68.

Videbech P. 2000. PET measurements of brain glucose metabolism and blood flow in major depressive disorder: A critical review. Acta Psychiatr Scand 101:11–20.

Videbech P, Ravnkilde B, Pedersen AR, Egander A, Landbo B, Rasmussen NA, Andersen F, Stodkilde-Jorgensen H, Gjedde A, Rosenberg R. 2001. The Danish PET/depression project: PET findings in patients with major depression. Psychol Med 31:1147–58.

Videbech P, Ravnkilde B, Pedersen AR, Egander A, Landbo B, Rasmussen NA, Andersen F, Stodkilde-Jorgensen H, Gjedde A, Rosenberg R. 2003. The Danish PET/depression project: Severely disturbed cerebral circuit in major depression. In prep. AQ3

Wolpert DM, Ghahramani Z. 2000. Computational principles of movement neuroscience. Nat Neurosci 3(Suppl):1212–7.

Yudofsky SC, Hales RE. 2002. Neuropsychiatry and the future of psychiatry and neurology. Am J Psychiatry 159:1261–4.

AUTHOR'S QUERY SHEET

Author(s): J. Hohwy & R. Rosenberg SWBP 157
Article title:
Article no:

Dear Author

Some questions have arisen during the preparation of your manuscript for typesetting. Please consider each of the following points below and make any corrections required in the proofs.

Please do not give answers to the questions on *this* sheet. All corrections should be made directly in the printed proofs.

AQ1 Blakemore et al. 2003, in press – any details?

AQ2 Hohwy & Rosenberg, submitted – any details?

AQ3 Videbech et al. 2003 – any details?