Cognitive processing in schizophrenia

I read the short report by Hall et al (2004) with interest. The authors reported a marked impairment in the ability of people with schizophrenia to make social judgements from facial expressions. Their findings complement and extend earlier studies by us and others (Hellewell et al, 1994; Edelstyn et al, 1996, 2003) that have reported the presence of impairments in facial recognition memory. However, these abnormalities in facial and emotion recognition do not appear to lead to obvious difficulties in day-to-day life; for example, individuals do not appear to exhibit problems with the recognition of familiar people. This apparent inconsistency between experimental findings and real-life situations raises issues about the role played by these cognitive abnormalities in schizophrenia. It is likely that these impairments are stable abnormalities rather than being transient indicators of dysfunction. This would be consistent with structural or functional abnormalities in schizophrenia, which only become evident when the processing systems are placed under high levels of stress, for example, during the prodromal or psychotic phases of a functional illness. This line of reasoning is supported by Hall et al’s finding that individuals with positive symptoms are unable to identify even basic facial emotions. These inherent weaknesses within the processing system may remain hidden during quiescent periods, but may be artificially exposed in the laboratory by challenging the processing system with particularly difficult tasks.

In an attempt to understand the basis of their findings, Hall et al drew attention to the roles of the frontal and temporal cortices as well as the amygdala. In addition to these, we believe that abnormalities in the non-intentional, automatic acquisition of knowledge about the structural relations between objects or events may contribute to impairments in social cognition. Lewicki (1988) and others have suggested that intuitive knowledge can influence how people form impressions, draw inferences and react to situations and people. Interestingly, a number of recent studies have reported the presence of implicit learning abnormalities in people with schizophrenia (e.g. procedural learning, word-stem completion, lexical and semantic priming) (Schwartz et al, 2003). Future research might examine how those with schizophrenia acquire implicit knowledge of regularities in social contexts and how this knowledge relates to adaptive functioning in schizophrenia.


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Authors’ reply: Professor Oyebode draws attention to a number of interesting issues and face processing in schizophrenia. A key question raised by our study is why deficits in emotion recognition were state-dependent, being limited to individuals experiencing positive symptoms, while impairments in social cognition were stable. One possibility, as discussed by Professor Oyebode, is that those who are free of positive symptoms are able to use alternative cognitive strategies to identify basic facial emotions. This view is supported by a functional magnetic resonance imaging study in which individuals with schizophrenia, none of whom was experiencing positive symptoms, were able to identify facial emotions correctly but nevertheless showed deficits in amygdala activation when processing facial affect (Gur et al, 2002). These findings suggest that other brain regions compensate for the normal functions of the amygdala in facial affect processing when individuals with schizophrenia are free of positive symptoms. More difficult tests, such as our social cognition task, may prevent such compensation and thus reveal an underlying stable deficit.

Professor Oyebode also points out the apparent discrepancy between the finding that people with schizophrenia have impairments in facial recognition memory on formal testing, but are able to recognise familiar people in day-to-day life. In our study we found no deficit in the ability of those with schizophrenia to recognise the identity of novel faces presented concurrently, suggesting that the deficits seen in previous studies resulted from the mnemonic and attentional demands of the tasks used, which may be lower for familiar people.


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Early interventions for psychosis

The last Cochrane systematic review of early intervention for those with psychosis included cognitive–behavioural therapy (CBT), family therapy and medication, and reported no significant decrease in the development of psychosis at 12-month follow-up (Marshall & Lockwood, 2004).
The implications of the recent study of CBT for the prevention of psychosis (Morrison et al., 2004) need to be realistically interpreted with this background.

First, two people were excluded from the cognitive therapy arm after the trial had begun, which would have led to a non-significant result. This should have been acknowledged in the abstract, as an abstract has the most impact with service planners.

Second, after 6 months of cognitive therapy, there was a decrease in the development of psychosis compared with the control arm; however, there was similar distress for both groups. Cognitive therapy for psychosis has an aim of decreasing the distress of psychosis as well as the formulation of an explanatory model for that psychosis. It may be that a reframed and normalised explanatory language was taught to the individuals at high risk, and this led to the decreased identification of symptoms at 12 months and the masking of a psychotic episode. This would not ultimately lead to a decrease in distressing psychosis, but to a later identification of psychosis and a possible delay in pharmacological treatment.

The possible risk of harm or hazard was ignored, with a clear bias against the use of medication expressed by the authors in the discussion. Furthermore, the editorial comment alluded to the possibility of premature publication (Tyrer, 2004), but it is the implication of harm which needs to be explicitly stated.


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Author's reply: We welcome Dr Marlowe's comments on our paper and would like to respond to the issues that he identified. The Cochrane review to which he refers examined more traditional approaches to early intervention (i.e. from first episode onwards) rather than a preventive approach in people at high risk, so we are unsure of the relevance of this. Within the manuscript we clearly acknowledge that there were several methodological limitations, including the exclusion of two participants, but we were unable to incorporate these in the abstract as he suggests because of limitations of abstract length imposed by the Journal (indeed, we were asked to further reduce the abstract at proof stage).

We agree that cognitive therapy for psychosis (and the prevention of psychosis) has an aim of decreasing the distress of psychotic experiences as well as the formulation of an explanatory model for a person's difficulties. We also agree that a reframed and normalised explanatory language may be developed by the service users; however, it is unlikely that this would lead to a masking of a psychotic episode. Rather, it is intended to reduce the potential for catastrophic appraisals of psychotic experiences, which are very clearly implicated in the experience of distress (Chadwick & Birchwood, 1994), and the development of normalising appraisals is at the heart of cognitive therapy for established psychosis (Morrison et al., 2003) and the prevention of psychosis alike (French & Morrison, 2004). Even if such a masking were to occur, the assumption that this could cause harm clearly demonstrates a bias against the use of psychosocial interventions, as it suggests that only pharmacological treatments can reduce the potential harm that may result from an untreated psychotic episode, when there is evidence that psychological treatment is also important in this respect (de Haan et al., 2003).

We are accused of being biased against using antipsychotic medication; we certainly are against medication in a population who are yet to develop a psychotic disorder, for the ethical reasons outlined within our paper and elsewhere (Bentall & Morrison, 2002). Finally, it is suggested that we avoid explicitly stating the possibility of harm arising from such an intervention; however, we clearly highlight the possibility of harm resulting from stigmatisation.


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Integration of psychiatric and physical health

In The Netherlands the British Journal of Psychiatry is distributed among Dutch psychiatrists by courtesy of the pharmaceutical industry. For the October issue of the Dutch edition I was asked to write the editorial comment, to be circulated with the Journal as an accompanying letter. My focus is integrated psychiatry in medicine.

Reading the October issue I was struck by the lack of an integrated perspective. Current epidemiological findings underscore how the organisation of our healthcare system is epidemiologically unfair and does not take into account the frequent co-occurrence of psychiatric disturbances and physical illness (Kendell, 2001; Royal College of Physicians & Royal College of Psychiatrists, 2003). The fragmentation of care is seen as one of the major problems of current healthcare (Institute of Medicine, 2001); this applies with regard to treatment of physical disorders in mental healthcare and vice versa.

The editorial by Kingdon et al (2004) on the recommendations of the Council of Europe lacks such an integrated perspective. Among the recommendations the quality of physical care is not mentioned by the Council other than in relation to restraint, and this omission is not mentioned by Kingdon et al.

Similarly, the review by Thornicroft & Tansella (2004) opens with the fact that depression leads to more disability-adjusted life-years than cardiovascular disease and cancer, but it does not report their meaningful interrelation, for instance through compliance (DiMatteo et al, 2000). In the section ‘Acute in-patient care’ it is mentioned that patients with physical comorbidity should preferentially be seen in such facilities and not in community...
Declaration of interest

F.J.H. has received a fee for writing the editorial comment circulated with the Dutch edition of the British Journal of Psychiatry.


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Quality of life and ECT

The first author of this study (McCall et al, 2004) has an apparent career, if not financial, conflict of interest in the treatment being reviewed. He is the President of the Association for Convulsive Therapy, the industry trade organisation, as well as the editor of its journal which he calls 'the voice of ECT' (McCall, 2004). This ought to have been revealed to readers directly; as it is, it reveals itself in the many flaws of research design which bias the study towards minimising the risks of electroconvulsive therapy (ECT).

The study included those who had had ECT as recently as 4 months previously, thus building into the research design the assumption that the adverse effects of ECT resolve within that time period; but there is evidence that this is not so. If it is not, then the study is simply comparing those who are still suffering the after-effects of ECT with those suffering more severe after-effects, a comparison which tells us nothing about the effects of ECT per se. The fact that those at baseline averaged a score of only 18 on the Mini-Mental State Examination suggests some type of cognitive dysfunction, perhaps due to ECT, even at that point.

The measures chosen by McCall et al in all areas – cognition, amnesia and, most importantly, what he calls quality of life and functioning – are the grossest possible, and cannot register the deficits known to be associated with ECT because they are simply not designed to do so. The authors must be aware of the work of the Service User Research Enterprise (SURE) group (Rose et al, 2003) in which patients describe a highly specific pattern of permanent memory and cognitive deficits post ECT. This was a rigorous systematic review of the literature on ECT's effects, and encompasses what most people would call quality of life and functioning. It revealed that for at least one-third of individuals ECT had deleterious, often devastating, effects on these areas which lasted more than 6 months and appeared to be permanent.

Individuals lost the ability to perform their jobs. They lost memory of up to 20 years of their lives. They were unable to handle schoolwork because of impaired memory function and concentration. They did not recognise persons previously well known to them. They waited anxiously for the promised 'return of memory' which never came. None of this is consistent with improvement in quality of life.

Why then are McCall et al's results so seemingly contradictory? Because he did not ask about these things. Instead, participants were asked, quite literally, whether they could wipe their own backsides. If they were simply able to get out of bed, feed and dress themselves, and use a bus or a telephone they were graded as functioning at the highest possible level. No one has ever reported that ECT affected their ability to use a toilet.

Finally, 4 weeks after ECT is too soon for individuals, who are unlikely to have tried to go back to work or school yet, to be able reliably to assess their altered memories and abilities.


Author's reply: We are grateful for Ms Andre's interest in our paper. She is the director of the Committee for Truth in Psychiatry (CTIP), which is a vocal anti-ECT group in the USA (see http://www.harborside.com/~equinox/ect.htm). As such, we feel that our work must be on target and of some importance to attract their criticism. Ms Andre has some specific complaints with our work, which we address as follows.

First, Ms Andre suggests that I have an apparent 'career, if not financial, conflict of interest' that invalidates the paper, especially as pertains to my role as President of the Association of Convulsive Therapy (ACT). I receive no financial or material support for serving as president of ACT; ACT is self-supporting through the dues of its members. The idea of a 'career conflict of interest' is not a concept endorsed by the American Medical Association Code of Ethics, per section 8.031 (Council on Ethical and Judicial Affairs, 1997). It is just as likely that she has a conflict of interest as director of CTIP in writing her letter – any information that supports the use of ECT threatens the position of CTIP. We would
welcome Ms Andre’s full disclosure of her financial support from CTIP, and disclosure of the source of funding for CTIP since its website states that dues are not a requirement for membership.

Second, she claims that those in our study had an average Mini-Mental State Examination (MMSE) score of 18 at baseline. In fact, the mean baseline MMSE score was 27.4, as shown in Table 2 (McCall et al., 2004: p. 407). The minimum MMSE score for inclusion was 18.

Third, Ms Andre takes us to task for not citing Rose et al. (2003). The Rose et al. paper has merit, but has no direct bearing on our work. Those authors ‘aimed to . . . assess the debated distinction between efficacy, effectiveness, and satisfaction’; the focus of our paper is quality of life (QOL) and function, not ‘satisfaction’. As reviewed by Asadi-Lari et al. (2004) satisfaction and QOL are discrete, non-overlapping ideas.

Fourth, Ms Andre asserts that memory effects of ECT must necessarily affect QOL. Ms Andre is changing the definition of terms to suit her purposes, or else remains unfamiliar with the field. QOL research is ‘. . . widely regarded as a robust measure of outcome assessment . . . and is defined as ‘. . . the patient’s perspective of their own health status’ (Asadi-Lari et al., 2004). It is a violation of the concept for anyone, including Ms Andre, to define a patient’s QOL for them.

Fifth, Ms Andre belittles our work for showing that ECT is associated with significant improvement in activities of daily living and instrumental activities of daily living. She does not recognise that impairment of instrumental activity of daily living may be the deciding factor in referring patients for ECT (McCall et al., 1999) and that ECT is superior to medication in improving instrumental activities of daily living over 1 year of follow-up (McCall et al., 2001).

We do share one goal with Ms Andre – a desire for truth in psychiatry. We choose to reveal truth through the scientific method as opposed to rhetoric.


Author’s reply: Dr McCall responds to my letter but does not answer it. I get very tired of explaining to ECT proponents that the Committee for Truth in Psychiatry is not an ‘anti-ECT’ group, but no matter how many times and in how many contexts I do so, that false statement continues to be made. More about CTIP later, since I cannot leave Dr McCall’s claims unrefuted.

My point about building assumptions about the longevity of ECT’s adverse effects into the research design by including persons who had recently had ECT was not addressed.

Nor was any evidence presented to show that the rating scales chosen by McCall et al are relevant to the types of deficits reported by former ECT patients and illustrated so well in the SURE report. (Nor has there been evidence, which I requested privately from the author, to show that the study participants, who for some reason scored so poorly on both the MMSE and the IADL prior to this course of treatment, are representative of ECT patients as a whole.)

McCall’s point that ex-patients and only ex-patients define what quality of life is and by what standard it should be measured is exactly my own: no ECT survivor or ex-patient ever has or ever would define ‘quality of life’ or ‘functioning’ in the terms Dr McCall uses. He says, ‘It is a violation of the concept for anyone to define a patient’s QOL for them’, yet that’s exactly what he has done. Had he asked patients themselves, an approach taken by the Rose et al group, he would have set off in a productive direction instead of down a blind alley.

His attempt to selectively redefine the work of Rose et al as research on ‘satisfaction’, not relevant to work on quality of life, is without foundation, as a reading of the actual study will show. It was he who brought up the work ongoing in Britain as relevant, by his reference in his first sentence to the National Institute for Clinical Excellence guidelines which came out concurrently with, and used some of the same evidence base as, the report of the Rose group at the SURE.

There is a wide literature on non-financial conflicts of interest, best described as ‘an individual occupying dual roles which should not be performed simultaneously’ (Fava, 2001). Those include treatment researcher and editor of a journal promoting the treatment under study.

If you yourself read what CTIP says, and not what others say about you, you will begin to wonder where the ‘anti-ECT’ claim comes from. We are an international organisation made up entirely of persons who have received ECT. We represent the spectrum of outcomes, from persons who feel ECT is beneficial and have had it more than once, to persons whose lives were ruined by it. None of us was truthfully informed of the risks of ECT before consenting to it, and no one liked being lied to. Our organisation exists for one purpose only: to advocate truthful informed consent for prospective ECT patients. Thus, it makes no sense to say that ‘any information that supports the use of ECT threatens the position of CTIP’.

Whether you are of the opinion that being in favour of truthful and informed consent somehow makes you anti-ECT depends on whether you believe that patients have the right to full disclosure of ECT’s risks – and the right to make a decision for themselves based on that information – or whether you believe that ECT’s risks are such that full disclosure would result in patients en bloc deciding to forego the treatment. That Dr McCall and colleagues are in the latter camp speaks much more eloquently than their article as to what they really believe about ECT’s effects on quality of life.

CTIP, founded in 1984, has never received funding of any kind.


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Editor’s note: This correspondence is now closed.
Integration of psychiatric and physical health
F. J. Huyse
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References
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