

Highlights of this issue

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Uncomfortable truths . . . about hospitalisation, older antipsychotics, race and employment

This month's editorials question the basis of some accepted beliefs, namely the benefit of minimising the time spent as an in-patient in hospital, and the reasons for favouring atypical antipsychotics in the treatment of schizophrenia. Capdevielle & Ritchie (pp. 164–165) debate the risks and benefits of shorter hospital stays and suggest that the shortest stays occur in countries where community care is most developed, but that the risks of suicide, criminality and readmission may be considerably higher with shorter hospital stays, especially when combined with suboptimal community provision. They advocate a focus on developing best practice guidelines to aid clinical decision-making. Lewis & Lieberman (pp. 161–163) review the results of the recent large antipsychotic treatment studies of schizophrenia, the CATIE and CULASS studies, and conclude that there is not only little difference between the effectiveness of older antipsychotic drugs and newer atypical agents, even on negative symptoms or cognition, but also little difference in their side-effect profiles. Clozapine remains the only antipsychotic medication showing a significant advantage over other treatments. They suggest that these data highlight the urgent need to develop safer and more effective treatments for schizophrenia. Cooper and colleagues (pp. 185–190) report that Black ethnic groups had a four-fold higher incidence of first-onset psychosis, and this was significantly associated with a more deprived socio-economic background and a greater perception of being disadvantaged. The authors conclude that this perception of disadvantage was not related to negative self-esteem or to the presence of psychotic symptoms, and question whether it was due to actual discriminatory experiences. Although there is a literature supporting the role of racism in the development of psychosis, the direction of causality can only really be addressed through a prospective longitudinal study. Employment rates are low for patients with schizophrenia, despite many patients expressing a desire to work. Catty *et al* (pp. 224–231) demonstrate that the use of a targeted individual placement and support (IPS) package shows superiority on all vocational measures, compared with the provision of routine vocational rehabilitation for people with severe mental illness. The quality of the therapeutic relationship and adherence to the IPS model predicted getting a job, and the authors suggest that these are both components that can be addressed through training and supervision.

Self-harm in first-episode psychosis, CBT and childhood sexual abuse

Suicide is the main cause of early death in psychosis. A first-episode psychosis study by Harvey *et al* (pp. 178–184) found high rates of self-harm prior to the presentation to services. Self-harm

was associated with depression, a longer period of untreated psychosis and increased insight. Other associated factors were male gender and higher socio-economic class. They suggest that some of these factors could usefully be addressed by early intervention services. An increase in rates of self-harm has been noted in younger people, and various psychological treatments have shown some benefit here. Slee *et al* (pp. 202–211) report that 12 sessions of cognitive-behavioural therapy, in addition to routine treatment, were effective in reducing self-harm, depressive symptoms and suicidal cognitions. The patients were selected on the basis of having recently self-harmed, and following their CBT also showed significant improvements in self-esteem and problem-solving ability, compared with the control group. The authors suggest that this could usefully be incorporated as an early stage of a stepped care approach, with more intensive input reserved for those unresponsive to this treatment. The aetiology of self-harm is considered to be multifactorial, but childhood sexual abuse has frequently been cited as a prominent factor and there are theoretical accounts of its primary role in this behaviour. Klonsky & Moyer (pp. 166–170) performed a meta-analysis of the available data and report only a small statistical association, perhaps accounting for 5% of the variance, between such abuse and self-harm. This is unlikely to reflect a primary role of such abuse in subsequent self-harm and is more likely to arise as a consequence of publication bias and the presence of mutual psychiatric risk factors.

Facial affect recognition in bipolar disorder and schizophrenia

The processing of facial affect has been shown to be disturbed in both bipolar disorder and schizophrenia, and functional neuro-imaging offers not only a means of examining the cortical basis for these deficits, but also the opportunity to examine the effects of treatment and symptom variation. Jogia *et al* (pp. 197–201) used functional magnetic resonance imaging to demonstrate that unmedicated patients with bipolar disorder showed decreased activation within regions of the prefrontal cortex while processing sad facial affect compared with controls. After treatment with lamotrigine, activation in these prefrontal regions was increased. The authors suggest that lamotrigine could act to normalise activation with the network of areas involved in processing facial affect, although symptom levels did not show a significant difference with lamotrigine treatment. A similar study in patients with schizophrenia processing fearful faces demonstrated significantly attenuated activation within the amygdala and extrastriate visual cortex, including the fusiform and superior temporal cortex. Interestingly, the severity of the patients' negative symptoms was correlated with attenuation of activation within the superior temporal cortex. Michalopoulou and colleagues (pp. 191–196) suggest that impaired activation within this region of the visual cortex may contribute to the failure to recognise fearful stimuli which has been reported to be more prominent in patients with negative symptoms. This region also has a role in social cognition which could provide a mechanism linking negative symptoms and disturbance of facial affect perception.