Highlights of this issue

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NO MAN IS AN ISLAND

John Donne's words are aptly used by Hobson (pp. 193–195) to illustrate not only how much of our mental life occurs as a dialogue with significant people around us, but also how the mind may develop under the influence of these relationships. He sets out the mutual benefits to be gained from integrating psychoanalytic method with the research ethos of experimental psychology. Lanman *et al* (pp. 255–260) take a preliminary step in this direction. Seven psychotherapists independently used the Personal Relatedness Profile to assess videotaped couple interactions. Their inter-rater reliability was good and correlations between items supported the Kleinian distinction between depressive and paranoid–schizoid states. The strength of this approach is that it can now be used to test the clinical utility of these states and quantify the predictive validity and likelihood of change in these positions.

CORTICAL ASYMMETRY IN EARLY-ONSET SCHIZOPHRENIA AND THOUGHT DISORDER

If there are brain changes underlying the development of schizophrenia, then early-onset cases may be expected to show these to a greater extent. Le Provost *et al* (pp. 228–232) studied the paracingulate sulcus in 40 male patients with early-onset schizophrenia and 100 healthy volunteers, and found a decrease in the normal asymmetry within the patient group. As the paracingulate sulcus develops by 36 weeks of gestation, they suggest that this may be a marker of early neurodevelopmental abnormality. Neurodevelopmental abnormalities within the language system have been suggested to be the core deficit within psychosis, and Ceccherini-Nelli & Crow (pp. 233–240) use the Clinical Language Disorder Rating Scale (an instrument to assess the form of language production) to demonstrate its utility in diagnosing ICD–10 schizophrenia. They suggest that this instrument may be useful in forensic settings where such language disturbance may be more reliable than Schneiderian first-rank symptoms.

COGNITION AND COGNITIVE TREATMENT OF DEPRESSION

Porter *et al* (pp. 214–220) demonstrate that unmedicated patients with depressive illness show impairment on a range of neuropsychological tests, perhaps unsurprisingly including attention and memory, but are comparable to controls in their psychomotor functioning. The severity of the depression correlated with performance on learning and memory measures, but not executive function. They suggest that the executive dysfunction may be a trait marker of depressive illness. Meanwhile, there is no dispute as to the usefulness of cognitive therapy in the treatment of depression, but the cost to benefit ratio is much less clear. Scott *et al* (pp. 221–227) examined the cost-effectiveness of add-on cognitive therapy in 158 people with partially remitted major depression receiving clinical treatment. Patients treated with cognitive therapy in addition to standard management (and antidepressant medication) were significantly less likely to relapse over the follow-up period of 17 months. However, there was a significant additional financial cost associated with cognitive therapy. Thus, it would appear that these additional short-term costs need to be balanced with better outcomes and concomitantly lower costs in the long term.

NEUROBIOLOGY OF EMOTION AND PROTEIN DRINKS

Perception and experience of emotion is essential for survival in any social environment. In an illuminating editorial, Phillips (pp. 190–192) discusses current thinking on the neurophysiology underlying normal emotion perception and its relevance for psychiatric disorders – not only affective disorders, but also schizophrenia and anxiety. The conclusion is that specific abnormalities in the structure and function of neural regions important for the identification of, and response to, emotional material are likely to be associated with prominent social difficulties in schizophrenia, emotional lability in bipolar disorder, and negative bias and depressed mood in major depressive disorder. Although tryptophan depletion has been used in research settings to precipitate depressive symptoms, Scarnà *et al* (pp. 210–213) demonstrate that the administration of a protein drink may also be a useful therapeutic agent. They were able to decrease measures of manic symptoms in patients with an acute manic episode treated with an amino acid mixture lacking the catecholamine precursors tyrosine and phenylalanine. They suggest that this acts by diminishing catecholamine synthesis and attenuating central dopamine transmission.

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