SES18.03
BIPOLAR PERSONALITY DIMENSIONS AND THE CHOICE OF MOOD STABILIZERS IN LONG-TERM TREATMENT

H.S. Akiskal. *International Mood Center, University of California at San Diego, 9500 Gilman Drive, La Jolla, CA 92093-0603, USA*

That bipolar temperamental dimensions could predict response to mood stabilizers is a provocative possibility about which there is little solid data from control trials. In this presentation I will summarize clinical considerations which make this approach attractive. The most consistent associations between temperament and bipolar types or course patterns are: 1) cyclothymic and bipolar II and rapid-cycling course; 2) depressive temperament and dysphoric mania; 3) hyperthymic temperament and recurrent or chronic mania. The first and the second have clinical evidence for better response to divalproex; the third one is notorious for mood stabilizer compliance and is often treated with depot neuroleptics. Carbamazepine seems to work best in the absence of bipolar family history. Lithium, by contrast, seems to work best in bipolar family positive euphoric manic states without inter-episode dysfunction, i.e., presumably without temperamental disturbance. Lithium augmentation of antidepressants tends to work in individuals with pseudo-unipolar depressions arising, according to our clinical observations, from the substrate of sunny or hyperthymic temperament. Extremely dysphoric irritable, hostile patients with depressive dips (considered "borderline") tend to respond thus to carbamazepine, divalproex, and lamotrigine; however, there is some data that hostility per se in these patients might respond to lithium. In brief, these clinical ideas need to be tested empirically.

SES18.04
PERSONALITY DIMENSIONS AS PREDICTORS OF TREATMENT OUTCOME IN AFFECTIVE DISORDERS

P. Tyrer, H. Seivewright, T. Johnson. *Department of Public Mental Health, Imperial College School of Medicine, London W2 1PD and MRC Biostatistics Unit, Cambridge, UK*

Background: Previous evidence from many quarters has indicated that people with both anxiety and depressive disorders who also have personality disturbance have a worse outcome than those with no personality abnormality. This was investigated more closely in a 12 year prospective study of 210 patients with DSM-III panic disorder, generalised anxiety disorder and dysthymic disorder.

Design: Randomised controlled trial of drug treatment, cognitive and behaviour therapy and self-help for 10 weeks followed by treatment in similar 'mode' wherever possible for next two years. Follow-up of as many as possible of original cohort after 12 years. Premorbid personality status recorded using the Personality Assessment Schedule (PAS)(Tyrer and Alexander, 1979) at baseline, with classification by personality severity as well as type (Tyrer and Johnson, 1996).

Results: 203 (97%) of original sample followed up. 17 had died. There was no difference between the proportions of those who had died with regard to initial personality status. Significantly worse outcome was shown in those with baseline personality disturbance and this was greater for those with more severe personality disorders.

(1) Tyrer, P. & Alexander, J. (1979) Classification of personality disorder. *British Journal of Psychiatry*, **135**, 163–167.
(2) Tyrer, P. & Johnson, T. (1996) Establishing the severity of personality disorder. *American Journal of Psychiatry*, **153**, 1593–1597.

SES18.05
TREATMENT OF SOCIAL PHOBIA: THE ASSOCIATION OF PERSONALITY AND AFFECTIVE SYMPTOMS

P. Bech

No abstract was available at the time of printing.

S54. Functional brain imaging: the future for psychiatry

Chairs: R.M. Murray (UK), P.K. McGuire (UK)

S54.01
THE PATHOPYSIOLOGY OF FORMAL THOUGHT DISORDER

T.T.J. Kircher*, P.F. Liddle, M. Brammer, S.C.R. Williams, R.M. Murray, P.K. McGuire. *University Hospital of Psychiatry, Osianderstr. 24, 72076 Tuebingen, Germany*

Formal thought disorder (FTD) is one of the core features of schizophrenia. However, little is known about its functional-anatomical correlates. In a series of experiments with functional Magnetic Resonance Imaging (FMRI), schizophrenic patients with and without FTD and healthy controls were investigated, using language perception and production paradigms. In a first set of experiments, subjects read sentence stems out aloud and had either to complete it in a meaningful way or choose between two words that made sense. A reading condition served as a baseline. In a second set, Rorschach inkblots were presented and subjects asked to speak about them, while echoplanar images were acquired. The utterances were recorded, the phenomenology of FTD was analysed and related to the BOLD effect. The neural correlates of the rate of articulation, positive and negative TD, usage of peculiar words and sentences were analysed using event related and correlational analysis. Our data showed, that schizophrenic patients with FTD differ fundamentally in the differential activation of the right and left superior temporal gyrus. The pathophysiological correlates of single symptoms, such as neologisms could be isolated for the first time and phenomena, such as 'conctretism' explained. The results are in line with findings in brain lesioned patients and neuropsychological experiments in controls as well as structural imaging studies in schizophrenia that linked FTD to abnormalities in the superior temporal gyrus. A theory that links these and other findings will be outlined.

S54.02
USING PET TO STUDY DOPAMINE RELEASE IN VIVO

P.M. Grasby*, C. Bench, M. Koepp, R. Gunn, C. Messa. *Department of Psychiatry, Imperial College School of Medicine, MRC Cyclotron Unit, Hammersmith Hospital, 150 Du Cane Road, London W12 ONN, UK*

Theoretically, PET has the potential to detect neurotransmitter release in vivo associated with behavioural and pharmacological challenges if sufficient endogenous neurotransmitter is released to cause appreciable change (via receptor occupancy) in the number of 'available' receptors targeted by a radioligand. Increasing evidence suggests radioligand displacement by pharmacological manipulation of endogenous neurotransmitter release can be detected in vivo. For example, dosing human subjects with d-amphetamine, which