and requires a high level of therapist expertise. However, given the considerable efficacy-effectiveness gap observed with medication alone, CT and other evidence-based brief therapies have an important role to play in the treatment of individuals with bipolar disorder.

S33.2

Cognitive therapy in relapse prevention in unipolar depression

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This paper reports a study designed specifically to investigate relapse prevention by cognitive therapy. 158 subjects with residual depressive symptoms after antidepressant treatment for major depression were randomised to receive clinical management plus antidepressant alone, or with 20 sessions of cognitive therapy over 5 months with two booster sessions. Subjects were followed up and treatment controlled for a further year. Cognitive therapy significantly reduced relapses from 47% to 29%. Effects on remission were smaller and symptom ratings affected were predominantly those of negative cognitions. Social adjustment was improved. Extreme thinking in either negative or positive direction predicted later relapse and was modified by cognitive therapy. Costs of other treatment were reduced by cognitive therapy, but did not fully compensate for therapy costs, giving a net cost per relapse avoided. This study is now one of five controlled trials in which cognitive therapy after the acute episode has been found to reduce relapse or recurrence in depression.

S33.3

Cognitive behaviour therapy versus pharmacotherapy in unipolar depression

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Both, cognitive behaviour therapy and pharmacotherapies are well-established treatments of unipolar depression. There are several large-scale studies available which demonstrate the efficacy of each of these treatments in comparison to control conditions. Looking at the direct comparison of psychotherapy and pharmacotherapy does not yet show a clear picture. This presentation will discuss results of own research on this topic. In particular, I shall discuss the question of efficacy in relation to dropouts, to side effects, to short-term and long-term response rates, as well as the question of combination treatment. I'll also address the question of severity of depressive symptomatology and the appropriateness of cognitive behaviour therapy and/or pharmacotherapy. In addition, I'll present some recent approaches of cognitive behaviour therapy with inpatients, with minor depression, with older depressed patients, and cognitive behaviour therapy with depressed stroke patients.

S33.4

Interpersonal psychotherapy in depression

C.B. Pull*, M.-C. Pull. *Centre Hospitalier de Luxembourg, Luxembourg*

Interpersonal Psychotherapy (IPT) was developed by Klerman, Weissman et al. for the treatment of depressive disorders, in association with, or without pharmacotherapy.

The procedures and strategies of IPT have been described in detail by its authors. As such, IPT may be considered a manual-based psychotherapy.

IPT is practical rather than theoretical It is based on the assumption that depressed patients have difficulties in interpersonal relationships. The difficulties are identified as belonging to one out of four major problem areas, i.e. grief, life transitions, interpersonal disputes, interpersonal deficits. IPT is designed to help depressed patients to solve these problems and to improve their depression.

The authors will describe the basic principles underlying IPT and provide information on recent developments in IPT.

In addition, the authors intend to present recent data concerning controlled trials that have been completed to assess the efficacy of IPT in depression.

S34. Disturbances of the self construct in schizophrenia

Chairs: K. Vogeley (D), P. Falkai (D)

S34.1

Perceiving the self in schizophrenia: Abnormalities in the awareness of action

S.-J. Blakemore*. *INSERM Unit 280, Processus Mentaux et Activation Cérébrale, Lyon, France*

It has been proposed that an impairment in 'self-monitoring' underlies certain symptoms associated with schizophrenia[1]. Recently, the self-monitoring mechanism has been interpreted in terms of a forward model of the sensorimotor system[2]. Forward models predict the sensory feedback from self-produced movements, thereby enabling us to recognise the sensory consequences of our own actions. It is proposed that an impairment in this predictive mechanism might give rise to certain psychotic symptoms. If self-produced sensations are interpreted as being generated by an external source, then thoughts might be interpreted as external voices (auditory hallucinations) and self-produced movements might be interpreted as externally generated (delusions of control or passivity phenomena). Psychophysical studies have shown that self-produced tactile stimulation is perceived as less intense than externally produced stimulation, which might be due to the sensory predictions made by a forward model[3]. Functional neuroimaging studies have demonstrated that such perceptual attenuation is mediated by somatosensory cortex and the anterior cingulate cortex: these areas are activated less by a self-produced tactile stimulus than by an identical stimulus when it is externally produced[4]. Evidence suggests that the cerebellum is involved in generating the prediction of the sensory consequences of movement[5]. Furthermore, psychotic patients with auditory hallucinations and passivity phenomena show no perceptual attenuation of self-produced sensory stimulation[6]. This supports the proposal that these symptoms are associated with an impairment of the functioning of the forward model.

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