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.

(1) Frith, CD. *The Cognitive Neuropsychology of Schizophrenia.* Lawrence Erlbaum Associates UK (1992).
(2) Frith, CD, Blakemore S-J & Wolpert, DM. *Brain Research Reviews* 31, 357–363 (2000).
(3) Blakemore, S-J, Frith, CD & Wolpert, DW. *Journal of Cognitive Neuroscience* 11(5), 551–9 (1999).
(4) Blakemore, S-J, Wolpert, DM & Frith, CD. *Nature Neuroscience* 1(7), 635–640 (1998).
(5) Blakemore, S-J, Frith, CD & Wolpert, DW. *NeuroReport* 12 (9): 1879–85 (2001).

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(6) Blakemore, S-J, Smith, J, Steel, R, Johnstone, E & Frith, CD. *Psychological Medicine* 30, 1131–1139 (2000).

S34.2

The cognitive neuropsychiatry of self perception

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Self awareness or insight is a key concept in neuropsychiatry. One approach to insight in psychosis is to search for analogies with anosognosia in neurological conditions. As with anosognosia, insight can be remarkably domain and modality specific. Both have clear cognitive components. However, the clinical course of insight in psychosis is somewhat different being rather more persistent although subject to state changes. Disorders of personal bodily awareness such as anorexia nervosa are well described although these appear to behave quite differently from neurological disorders. However there is growing evidence that depersonalization disorder can also be understood as an abnormality in feedback between representational and bodily-autonomic neural systems or "somatic markers" according to Damasio's terminology. This produces a profound alteration in the subjective sense of the self. Finally, the functional neuroanatomy of self perception is beginning to be understood. It is postulated that The Self should be considered as a separate semantic category.

S34.3

Aspects of the connection between external tasks and internal processing modalities in patients suffering from schizophrenia

B. Gallhofer. *Germany*

No abstract was available at the time of printing.

S34.4

Misattribution of action in schizophrenic patients with Schneiderian symptoms

M. Jeannerod*. *Institute des Sciences Cognitives, Lyon, France*

We tested the hypothesis that Schneiderian symptoms observed in patients with schizophrenia may be explained by a misattribution of their own actions to another agent. We have realized experimental situations where moving hands of an uncertain ownership (i.e., belonging to the subject or to an alien person) were presented. Groups of normal controls and groups of patients suffering from schizophrenia – with Schneiderian symptoms (S) and without Schneiderian symptoms (NS) – were tested. Subjects were requested to identify the action they saw by attributing it to their own hand or to the alien hand. Two types of attribution errors were recorded: misattribution to the self and misattribution to the other. Observation of subjects' performance showed that patients from NS groups and normal controls presented an attribution preference in the same order of magnitude for the two types of attribution of an action, although NS patients made more errors than controls. Patients from S groups, by contrast, showed, first a difference in their decision criteria for attributing an action to them or to another agent and, second a tendency in over attributing an action to the other. The outcome of these experiments bears strong implications on the functioning of social cognition in patients with schizophrenia.

S34.5

Schizophrenia conceived as self-disorders

C. Scharfetter*. *Department of Psychiatry, University of Zürich, Switzerland*

The clinical experience focussing in the self-experience of schizophrenics led to the construct of ego disorder in five basic dimensions: ego vitality, activity, consistence and coherence, demarcation, identity. This clinical concept is in line with a series of historical observations even of the time before the nosological construct schizophrenia was published (Kraepelin, E. Bleuler). Systematic empirical studies with a questionnaire, the Ego Pathology Inventory, on more than 500 schizophrenics confirmed the model of a general factor "ego disorder or disorder of self-consciousness" with 5 subfactors (as mentioned above).

S34.6

Synaptic proteins in schizophrenia: a "bottom-up" approach to neural connectivity

W.G. Honer. *Department of Psychiatry, University of British Columbia, Vancouver, Canada*

Synaptic neurotransmission is dependent on the integrity and function of proteins which allow vesicles to release their contents in a regulated fashion. Three core proteins (the SNARE molecules syntaxin, SNAP-25 and VAMP) are reported to be affected in schizophrenia. In most brain regions studied, the amounts of the proteins are reduced, however in cingulate cortex there may be increases. Recent work demonstrated altered in vitro interactions between these three proteins in tissue from individuals with schizophrenia or depression and suicide as a cause of death. As well as the SNARE proteins, two others, complexin I and complexin II are of particular interest. Both are modifiers of SNARE interactions. Complexin I is enriched in inhibitory terminals, and complexin II in excitatory terminals. We recently described reduced complexin I in prefrontal cortex in schizophrenia, consistent with a loss of inhibitory interneurons as reported by others. Inhibitory processes may therefore be reduced in prefrontal cortex in schizophrenia. In contrast, both complexins appeared to be reduced in hippocampus in schizophrenia, with greater loss of complexin II suggesting reduced excitatory relative to inhibitory processes. Future studies of these proteins in animal models of learning, memory and behaviour may help link molecular and cognitive processes in schizophrenia.

S35. UEMS and AEP in collaboration in education

Chairs: M. Gomez-Beneyto (E), K.-O. Svärd (S)

S35.1

Introduction and presentation of the activities of the UEMS Section & Board of Psychiatry

A. Lindhardt. *Denmark*

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