compulsive buying. Twenty outpatients with a lifetime diagnosis of kleptomania and ten suffering from compulsive buying were evaluated by means of the Structured Clinical Interview for DSM-IV, of a specially-designed semi-structured interview and of a modified version of the Family History Research Diagnostic Criteria. The majority of patients reported an early and abrupt onset, with an episodic course of the disorder with no gender prevalence. Lifetime comorbidity for other Axis I disorders was relevant, in particular for mood disorders, obsessive-disorder (OCD), separation anxiety, panic disorder and OCD-related disorders such as pathological gambling and tricotillomania. Family history showed a high prevalence of mood disorders, alcohol abuse and OCD. Our study indicated a clear connection between kleptomania, mood disorders and OCD, the exact nature of which has yet to be clarified.

S04. Behaviour and motor control in psychiatric disorders

Chairs: M.R. Lemke (D), B.G.C. Sabbe (NL)

S04.01

IMPULSIVITY, BEHAVIORAL DYSCONTROL, AND CONSCIOUS AWARENESS

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Impulsivity has emerged as one of the leading risk factors for behavioral disorders including, for example, substance abuse, ADHD, borderline and conduct/antisocial personality disorders. Impulsive acts have been defined clinically as "occurring suddenly, quickly, without much forethought, and without consideration of consequences" (Granit). Theoretical causal models of impulsivity emphasize arousal, varying forms of information processing, attention, and sensitivity to reinforcement in learning paradigms. Barratt and colleagues have proposed that impulsivity is related to temporal information processing which is related to performance on timing and rhythm tasks. In construct validity studies they have demonstrated that self-report measures of motor impulsivity, performance on a wide range of behaviors involving timing and rhythm requirements (verbal tasks [e.g., reading], pursuit rotor and finger tapping) and selected cognitive psychophysiological measures related to behavioral inhibition (N_{200}) and information processing (P_{300}), converge to define impulsivity. In predictive validity studies they have demonstrated that these measures are significantly related to ADHD, conduct disorder and impulsive aggression. This paper will discuss proposed neural circuits (e.g., basal ganglia, thalamus, and frontal lobes) which may be related to the behavioral measures of impulsivity and will include a discussion of a scale of conscious awareness which has impulsivity and premeditated behaviors at opposite extremes. Current research relating the lateralized readiness potential to impulsivity will also be broached.

S04.02

A NEW MODEL TO ASSESS STIMULUS PROCESSING AND BEHAVIOR CONTROL IN HUMANS FOLLOWING EXPERIMENTAL INDUCTION OF EMOTIONS

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Objective: Many psychiatric disturbances including borderline and antisocial personality disorders, substance abuse, eating disorders and suicidal behavior present with dysfunction of processing stimuli into adequate actions. Perceptive and executive components of this process may be modulated by affective stimuli. It was hypothesized that stimuli processing and generation of movements are affected by induction of positive and negative emotions.

Methods: Healthy subjects were subjected to visual stimuli which they could turn of by releasing and pressing two buttons. Neurophysiological methods including EEG, startle reflex, EMG and kinematic measures of hand movements by infrared detection (Proflex) were used to analyze the neuronal process from stimulus perception to movement execution with a specific software program for continuous chronological assessment of the signals with high precision.

Results: Stimuli (International Affective Picture System) induced differend affective valence ratings (neutral/positive/negative) and startle response amplitudes (neutral 48.85 ± 3.28, positive 49.95 ± 4.14, negative 52.04 ± 8.95) Movement analysis revealed differences in onset of movement (neutral 4.86 ± 1.56, positive 5.65 ± 2.12, negative 4.73 ± 1.32 sec), movement duration (neutral 1.48 ± 0.59, positive 1.49 ± 0.42, negative 1.36 ± 0.44 sec), max. velocity (vmax) (neutral 0.45 ± 0.13, positive 0.46 ± 0.15, negative 0.46 ± 0.13 m/s), vmax latency (neutral 37.88 ± 17.18, positive 39.87 ± 14.35, negative 36.11 ± 12.7 sec).

Discussion: We showed for the first time that not only perceptive, but also executive components of CNS behavior control can be experimentally modulated by affective stimuli, The efficacy of psychotherapeutic and psychopharmacological interventions on behavioral control can be evaluated using our model. Future studies will include experimental variation of serotonergic CNS activity and its effects on behavior control.

S04.03

ANXIETY/AGGRESSION-DRIVEN DEPRESSION: A MANIFESTATION OF SEROTONERGIC PATHOLOGY?

H.M. van Praag

No abstract was available at the time of printing.

S04.04

FACIAL EXPRESSIONS AND PERSONALITY

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Facial expressions are often disturbed in psychiatric patients. Subjects with personality styles such as sensation seeking, extraversion or impulsivity are thought to have a risk to develope psychiatric disorders. To identify those subjects, kinematic analysis of facial expressions could be helpful. An active measurement device was used, allowing kinematic analysis of facial movements in detail. Markers which are fixed in distinct points of the face and send light or ultrasonics in high frequency give a direct measure of facial movements with high spatial-temporal resolution. Healthy

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subjects (n = 44, age: in mean 40.3 (range 23–76) years, 21 males, 23 females) watching a witty movie ("Mr. Bean") were investigated. The speed of the facial expression "laughing" was more pronounced in subjects with high scores of Zuckerman's sensations seeking scale (general, boredom susceptibility) and Neo-FFI (frankness, neurotizism) than in subjects with low scores. In contrast, the speed of voluntary movements of mouth and eyes was not correlated to the personality measurements. Kinematic analysis of facial expressions seems to be useful in identifying subjects with sensation seeking and related personality styles. Higher speed of facial movements found in these subjects suggests enhanced dopaminergic function in such a personality.

S04.05

PSYCHOMOTOR RETARDATION IN DEPRESSION DURING TREATMENT WITH SSRI's

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Earlier research on fine motor retardation in inpatients with a Major Depressive Episode (MDE) and on the effects of treatment with SSRI's, has demonstrated that mainly cognitive slowing improved during treatment, while motor slowing only slightly changed (Sabbe et al., 1996, 1999).

Cognitive and motor slowing and their changes were studied by using a computerised device (PC, digitizer and a specially designed pen), that enables measuring and analysing in great detail writing and drawing behaviour. By analysing different kinematic variables of the hand movements, such as the reaction time, the movement time and their components, and by manipulation of the cognitive and motor demands of the different tasks, cognitive processes and motor processes can be studied at the start and during treatment. Cognitive processes encompass attention, perception, working memory and planning, and motor processes include programming, initiation, coordination and execution of the movement.

In the studies that will be presented three research domains will be further explored: (a) the detailed analysis of cognitive and motor slowing in depressed inpatients (MDE) by weekly measurements during a 6-weeks treatment with sertralin (50–100 mg/d); (b) differences between patients with MDE with diurnal variation of depressed mood and patients without this variation; (c) comparison of cognitive and motor slowing in patients with MDE and with dysthymia. It is concluded that psychomotor changes early in treatment, can differentiate between subgroups of patients which show full, mild and none response to treatment.

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S05. The biology and psychology of memory disorder in schizophrenia

Chairs: S.R. Hirsch (UK), J.M. Danion (F)

S05.01

EARLY MEMORY CHANGES IN FIRST EPISODE STUDIES

E. Joyce

No abstract was available at the time of printing.

S05.02

CHARCTERISATION OF THE MEMORY DEFECTS: ACCESS OR VERSUS STORE DISORDER

T.K. Kondel

No abstract was available at the time of printing.

S05.03

PROBING SEMANTIC MEMORY IN SCHIZOPHRENIA

A. David

No abstract was available at the time of printing.

S05.04

EFFECT OF A SUBANESTHETIC DOSE OF KETAMINE ON MEMORY AND AWARENESS IN HEALTHY VOLUNTEERS

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Background: Ketamine is an NMDA receptor antagonist with psychotogenic and cognitive effects in healthy volunteers and schizophrenic patients which has been proposed to be an useful tool to investigate neurobiological basis of schizophrenia. The present study characterized the effects of a subanesthetic dose of ketamine on memory and related subjective states of awareness in healthy volunteers.

Methods: Twenty-six subjects were given either a 60-minute ketamine (0.5 mg/kg/hour) or a placebo infusion. To obtain constant plasma ketamine throughout the experiment, ketamine was administered using a computer-assisted infusion. Subjects carried out episodic memory tasks involving words presented before and during infusion. Memory performance was assessed with recognition and free recall tasks. Subjective states of awareness were assessed using an experiential approach. Levels of psychopathology were evaluated with BPRS.

Results: Ketamine impaired performance in free recall and recognition of words presented during, but not before, infusion. There were no differences between groups concerning states of awareness associated with recognition memory. Subjects under ketamine had higher BPRS total scores as well as BPRS negative and positive clusters scores than control subjects.

Conclusions: Ketamine decreases episodic memory performance by impairing encoding, but not retrieval processes. It does not selectively impair subjective states of awareness associated with recognition memory as it has been seen in patients with schizophrenia. Ketamine might mimic the memory impairment associated with acute, but not chronic, forms of schizophrenia.

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