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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

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No abstract was available at the time of printing.

S34.4
Misattribution of action in schizophrenic patients with Schneiderian symptoms

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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

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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

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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

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