S47.3
Sleep in schizophrenia

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The polysomnographic (PSG) profile of schizophrenic patients has been studied with somewhat controversial results. Most studies, however, show that schizophrenic patients generally exhibit disruption of sleep continuity, reduced slow-wave sleep and reduced REM latency.

Reduced stage 4 sleep and reduced REM latency has been reported to correlate with psychotic symptomatology, especially negative symptoms. Reduced REM latency has been attributed to cholinergic hyperactivity secondary to increased dopaminergic tone. Neuroleptic treatment, as well as treatment with anticholinergics, has been reported to prolong REM-latencies in schizophrenic patients.

We studied polysomnographic sleep recordings, and morning serum prolactin levels as a measure of dopaminergic tone, in17 drug-free patients suffering from non-affective psychoses. A clear-cut positive correlation between prolactin and REM latency was found, as well as a negative correlation between prolactin and REM sleep. We suggest that short REM latencies and overt psychopathology in patients with-non affective psychoses are not directly associated with dopaminergic tone only, but may at least partly be mediated by hypercholinergic mechanisms accompanying the increased dopaminergic tone.

S47.4
Sleep and neuroendocrinology

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Sleep has an electrophysiological and a neuroendocrine component. Slow wave sleep (SWS,) and the growth hormone (GH) surge occur during the first half of the night, whereas cortisol secretion and REM sleep predominate during the second half of the night. A bidirectional interaction exists between sleep EEG and endocrine activity. Manipulation of sleep-wake activity (e.g. sleep deprivation) affects hormone secretion. On the other hand changes of hormone levels exert effects on sleep EEG.. A reciprocal interaction of GH-releasing hormone (GHRH) and corticotropin-releasing hormone (CRH) appears to play a crucial role in sleep regulation. In young normal men GHRH promotes SWS and GH and inhibits cortisol whereas CRH exerts opposite effects. Changes in the GHRH/CRH ratio may contribute to disturbed sleep in depression (CRH overactivity) and ageing (reduced GHRH activity). Interestingly GHRH appears to exert opposite effects in women and men, possibly one explanation for the higher risk for depression in women.

Also other hormones participate in sleep regulation. NPY and galanin promote sleep, somatostatin impairs sleep. Subchronic glucocorticoid treatment of female patients with multiple sclerosis induced changes of sleep, particularly REM sleep resembling to those in depressed patients. We expect that our research contributes to the development of novel strategies for the treatment of sleep and psychiatric disorders.

S47.5
Sleep in anxiety disorders and stress

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Anxiety disorders have a high prevalence. Their relation to sleep is the focus for this paper. In panic disorder up to 70% of the patients have sleep disturbance, many have sleep-related panic attacks; also abnormalities in REM sleep have been demonstrated. In OCD a decreased total sleep time (TST), increased awakenings and somewhat lower REM latency and less slow wave sleep (SWS) has been reported. In GAD patients a prolonged sleep latency has been demonstrated, but little effects on sleep architecture. In PTSD increased nightmares and fragmentation in REM sleep and less SWS are reported findings. Only a few studies have focused on the relation between perceived stress and sleep habits. We have examined data from three population samples. All three studies were questionnaire-based. Perceived stress was rated on a 5-point scale. The stressed groups had significantly more sleep disturbances, with the most frequent sleep-related symptom being "not being rested by sleep". In a comparison to the none-stressed group the highest odds ratio (OR) was for "awakening too early"(OR 11.2), followed by "awakenings during the night" (NW), OR 7.4, and difficulties falling asleep OR 6.6. Thus, perceived stress during daytime is associated with disturbed sleep.

S47.6
Exploring antidepressant mechanisms by acute and chronic sleep effects

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The use of sleep as a measure of antidepressant drug effects has proved useful and informative in studies of depressed patients. Both changes in sleep architecture, particularly in REM and slow wave sleep, and alterations in sleep continuity are present during different phases of illness and during antidepressant treatment.

We have studied outpatient depressed and anxious patients at home using ambulatory polysomnography. Investigations of TCA and SSRI antidepressants over the course of acute, subacute and chronic treatment have revealed that the expected REM latency increases persist up to 3 months, although early disruption of sleep continuity does not. In a study of paroxetine and nefazodone in depression, measures of sleep continuity are improved early in treatment with nefazodone with little effect on REM sleep, but. after 8 weeks the differences in sleep continuity between treatments were much less. Slow wave sleep distribution in the 2 groups will be discussed. In addition, our studies of lithium and pindolol augmentation of TCAs in resistant depression have revealed marked alterations in sleep which may help to clarify the mechanism of action.

These studies have contributed to our understanding of the role of serotonin and serotonin receptors in sleep.

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