free-running circadian rhythms but only when the administration time coincides with the light/dark transition (e.g. onset of activity in nocturnal mammals, offset of activity in diurnal ones) and the phase angle difference depends on the duration of the Mel signal. The molecular mechanisms underlying these effects of exogenous Mel on the clock rhythmicity are not clarified yet. Contrary to what has been described for photic or non-photic cues, clock gene mRNA's are not the initial targets for Mel action on the SCN.

SS-16-03

Sleep deprivation and antidepressant treatment

U. Voderholzer, M. Berger, D. Riemann. *University of Freiburg, Freiburg, Germany*

Acute sleep deprivation (SD) for one night improves mood in about 60% of depressed patients the day after. In this respect, among all types of antidepressant treatments, SD elicits the fastest results. The main limitation, however, is the transient nature of the effect, since the majority of the improved patients experience a relapse after the next night of sleep. A variety of studies focussed on strategies to avoid relapsing and additionally treated the patients with light therapy, lithium, or other drugs. A further strategy has been to advance the sleep period to an unphysiological time. Several studies showed that a phase advance of the sleep period, over a course of either six or three nights consistently stabilized the antidepressant effect of SD in about 60% of those patients who responded positively to SD (1,3). Up to now, only one study also included a control group which participated in a phase delay protocol after SD instead of a phase-advance protocol (2). Significantly more patients relapsed in the phase-delay protocol, supporting the hypothesis that sleeping at certain phases of the circadian rhythm, i.e., especially late in the night and in the morning, has depressogenic effects. The major limitation of the phase advance studies is, that the effect has been shown over a period of one week; further studies have to be included a follow up over the course of four to six weeks.

References

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Riemann et al. (1999) Eur Arch Psychiat Clin Neurosci 249: 231-237.
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SS-16-04

Sleep-wake rhythm disturbances in major depression and primary insomnia: A study of sleep microstructure

L. Staner. *FORENAP, Rouffach, France*

Objective: A close relationship between the regulation of sleep and the regulation of mood has been suggested by several studies showing that insomnia and depression are two closely linked clinical entities. In the present study we investigate whether a same or a different mechanism is operating in the sleep onset disturbances of primary insomniacs (PI) and major depressive insomniacs (MDI).

Methods: For this purpose, the time course of EEG power density during the period preceding sleep onset and during the first non REM period was examined in three age and gender matched groups of 10 women and 11 men (MDI, PI and healthy controls - HC).

Results: In contrast to HC and MDI, PI did not experience a gradual decrease of their alpha and beta1 power during the sleep onset period and had a lower delta activity in the 5 minutes preceding sleep onset. Compared to the 2 other groups, MDI exhibit less dynamic changes in slow wave activity during the first non REM period.

Conclusion: The present results suggest that increased wake propensity (Process W) may mainly be implicated in PI whereas a lower sleep pressure (low Process S) is related to MDI. The paper will be discussed in light of ongoing studies on sleep microstructure in 2 different model of sleep disturbances: post-nap sleep and sleep after a transient tryptophan depletion paradigm

SS-16-05

Treatment of seasonal affective disorder

K. V. Danilenko. *Centre for Chronobiology Institute of Internal Medicine, Novosibirsk, Russia*

Bright light is the treatment of choice for winter seasonal affective disorder (SAD). An accepted algorithm is to begin treatment with 10'000 lux fluorescent light for 30 minutes daily upon awakening. Although response usually occurs within a week, treatment should last longer for a stable response, sometimes for the entire winter. Meta-analyses reveal that light has antidepressant action beyond its placebo effect. Dawn simulation is a low intensity form of applying the light signal during sleep. The pathophysiology of SAD is not yet clear, nor is known the mechanism by which light is antidepressant. Beyond direct neurobiological effects (e.g. to rapidly increase serotonin turnover) morning light phase advances circadian rhythms - which tend to be phase delayed in SAD - and the advance is correlated with mood improvement. Regular exposure to outdoor light is also therapeutic. Other treatment modalities have been studied much less than light therapy. Pharmacological approaches (fluoxetine, bupropion, reboxetine, agomelatine, some others) provide clinically equivalent results as light, though not as rapidly and with more side effects. High-density negative air ionisation is surprisingly effective compared to (placebo) low-ion density administration. Physical exercise and behavioural therapy have been the focus of a few positive studies. Sleep deprivation is effective in some SAD patients, similar to the response in non-seasonal depression. Evening melatonin administration is not clearly antidepressant. Tests of a morning carbohydrate-rich diet have also been ineffective, and hypericum, a herb which increases light sensitivity, is an interesting possible adjunct at present under investigation.

Sunday, April 3, 2005

W-06. Workshop: Neuropsychological and neuroanatomical correlates of affective-cognitive interaction in major depression

Chairperson(s): Georg Northoff (Magdeburg, Germany), Heinz Boeker (Zürich, Switzerland)
16.15 - 17.45, Holiday Inn - Room 5

G. Northoff. *Universität Magdeburg Klinik für Psychiatrie, Magdeburg, Germany*

Objective: The symposium aims at demonstrating the neuropsychological and neuroanatomical correlates of abnormal affective-cognitive interaction in depression. Therefore, different studies in functional imaging and neuropsychology investigating

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affective-cognitive interaction will be presented. Dr.H.Boeker demonstrates attentional dysfunction in depression in neuropsychological studies. Dr.Grimm will present neuropsychological and imaging results showing disturbed emotional processing in ventral prefrontal cortex (VPC). Hyperactivation in medial regions is accompanied by hypoactivation in lateral regions of the ventral prefrontal cortex. Dr.Bermpohl investigated the modulation of emotional processing by preceding attention. He showed abnormal attentional modulation of neural activity in VPC in depression. Dr.Northoff presents imaging data about abnormal deactivation in depression in VPC by attentional modulation of emotional judgment. Based on correlation findings, such abnormal deactivation in depression in VPC by attentional modulation might be related to the abnormal focus on negative emotions and the inability to shift to positive emotions. In summary, the symposium reveals abnormal function in VPC as a crucial neural correlate of altered affective-cognitive interaction in major depression.

Tuesday, April 5, 2005

W-15. Workshop: Comorbidity in bipolar disorders

Chairperson(s): Peter Brieger (Halle, Germany), Zoltan Rihmer (Budapest 27, Hungary)
14.15 - 15.45, Holiday Inn - Room 4

G. Perugi, A. Erfurth, BHK Augsburg, Augsburg, Germany. *Universita degli Studi di Pisa, Pisa, Italy*
Z. Rihmer. *National Institute for Psychiatry, Budapest 27, Hungary*
I. Maremmani. *Universitá Pisa, Pisa, Italy*
P. Brieger. *MLU Halle-Wittenberg Klinik für Psychiatrie, Halle, Germany*

The workshop's topic is new research and perspectives in the growing field of bipolar spectrum disorders. Andreas Erfurth (Augsburg, Germany) presents research into the field of temperament and bipolar disorders including their complex interactions. Giulio Perugi (Pisa, Italy) discusses the overlap between anxiety, obsessions, impulsivity and bipolar disorders. Icro Maremmani(Pisa, Italy) presents research into the comorbidity of bipolar disorders and substance abuse, with a focus on opiate dependence. Zoltan Rihmer's (Budapest, Hungary) talk is on the comorbidity of bipolar I and bipolar II disorders and anxiety disorders, as well as on the frequency of suicide in the bipolar spectrum. Peter Brieger(Halle, Germany) gives data on subjective quality of life in a large cohort of remitted bipolar patients - and its relation to course and outcome. The overall aim of the workshop is to present new data and modern concepts of different aspects of bipolar spectrum disorders, which are relevant for both clinicians and scientists.

Tuesday, April 5, 2005

W-18. Workshop: Fate of psychotropic drugs at the blood-brain-barrier and in the brain: Pharmacokinetic, pharmacodynamic and clinical consequences

Chairperson(s): Pierre Baumann (Prilly-Lausanne, Switzerland), Georg Nikisch (Fulda, Germany)
16.15 - 17.45, Holiday Inn - Room 4

P. Baumann. *Unite de biochimie et psycho- pharmacologie clinique, Prilly-Lausanne, Switzerland*
G. Nikisch. *Klinikum Fulda Psychiatrie und Psychotherapie, Fulda, Germany*
P. Baumann. *Unite de biochimie et psycho- pharmacologie clinique, Prilly-Lausanne, Switzerland*
J. Tauscher. *Dept. General Psychiatry University Vienna, Vienna, Austria*

The in vivo pharmacological effects of psychotropic drugs are expected to depend on their availability in the brain, but this issue has so far been neglected in research. The principal aim of this symposium is to present the relationship between drug concentrations in blood, brain and/or CSF, and their effect on neurotransmitters and neuropeptides, which are measured in blood or CSF of patients treated with antidepressants, and clinical parameters such as therapeutic or adverse effects. Observations obtained with human studies will be compared with those using animal models (pharmacological models, knock-out animals).

Sunday, April 3, 2005

C-02. Educational course: Teaching general practitioners about depression

Course director(s): John Cooper (Nottingham, United Kingdom), Linda Gask
08.30 - 12.00, Hilton - Salon Orff

Depressive illnesses cause much suffering and disability, even when they are not obvious or severe. They constitute a very large burden on the individual, the family and the community, so it is important that all opportunities to identify and treat patients with depressive illness are taken. Many patients are seen in primary care and in general hospital services who can be treated successfully without referral to specialist psychiatric services. Some studies have been published which demonstrate how to tackle this problem successfully, but some published studies have not been successful. The differences between successful and unsuccessful studies will be discussed in this course, and the teaching modules produced by the World Psychiatric Association will be used to demonstrate some of the components needed for successful courses. Passive presentation of information by itself is not sufficient, and the course will include brief illustrations of practical clinically-based exercises as examples of how participants in such courses can be actively involved in teaching and learning procedures.

Sunday, April 3, 2005

C-04. Educational course: Introduction to cognitive psychotherapy

Course director(s): Stirling Moorey (London, United Kingdom)
14.15 - 17.45, Hilton - Salon Bialas

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