

using the questionnaire. Following main topics were evaluated: a. problems with getting adequate information about HD in early stages of the disease, b. main problems connected with the diagnosis itself, c. cooperation and communication with physicians, d. clinical symptoms of HD most severely affecting the life of CG, e. information about the existence of HD support group and f. the usefulness of HD support group for CG.

**Results:** 14/21 CG had severe problems with getting adequate information about HD in the early stage of HD, while 7/21 had enough information. The risk of HD for their children was found to be the main problem connected with the diagnosis itself. Other main problems were how to explain the risk of HD to their children and the incurable character of HD. Cooperation and communication with physicians were reported as very problematic in 15/21 CG, whereas 6/21 had good experience with physicians. The insufficient interest in family and social problems and almost no contact with partners and CG were reported to be the main controversy. Behavioral and affective changes were reported as most severe symptoms of HD in 12/21 CG (aggression in 12/21, depression in 7/21 and sexual disturbances in 3/21), symptoms of dementia in 10/21, dysarthria in 7/21, involuntary movements and/or gait disorders in 3/21 CG. The information about the existence of HD support group among physicians and medical services was found to be insufficient in 16/21 CG. The usefulness of HD support group was found to be extremely important for all CG, even more important than medical services.

**Conclusions:** What should be done: a. to accept CG as a client and a patient, b. to be aware that CG need sometimes the medical help and always social and psychotherapeutical support, c. to educate physicians and medical services about problems, connected with the character of HD and about the impact of HD on CG, d. to improve the knowledge and the information about HD support group among physicians and medical services and e. to force authorities to work actively in the field of family and social support of HD.

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## P02.277

### AUTONOMIC NEUROCARDIAC REGULATION (ANR) IN PATIENTS WITH MAJOR DEPRESSION AND EFFECTS OF ANTIDEPRESSIVE TREATMENT WITH REBOXETINE

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**Background:** There is growing evidence that major depression (MD) is associated with disturbances in autonomic neurocardiac regulation (ANR). Moreover, antidepressants from various classes distinctly influence ANR depending on the extent to which anticholinergic and/or anti-adrenergic effects are exerted *in vivo*. Reboxetine is the first selective noradrenaline re-uptake inhibitor. Its action on ANR in man remains to be established.

**Methods:** 25 strictly selected, untreated patients with MD (DSM-III-R) and a minimum of 18 points on the Hamilton Depression Scale (HAM-D) were recruited. Exclusion criteria were the presence of any disease known to affect ANR (e.g. diabetes mellitus, coronary heart disease, drug abuse, alcoholism). ANR was assessed using standardized measurements of the 5-min resting heart rate variability (HRV). Reboxetine was started with 4 mg/d; after 10 days the dose was increased to 8 mg/d. Repeated HRV studies were performed before (baseline) and after 2, 10 and 21 days of treatment, respectively.

**Results:** Serial HRV recordings (n = 100) showed that reboxetine (4–8 mg/d) did not reduce the vagally mediated HRV-indices both, in the time domain (CVr, RMSSDr) and frequency domain (HF-power) analysis. Compared to baseline there was a significant decrease of the low frequency bands (absolute and relative values) and the mean LF/HF ratio (p < 0.05), which occurred even after 2 days of treatment. Reboxetine (4–8 mg/d) did not influence ECG time relations; in particular, the mean QTc- and PQ-conduction times remained obviously unchanged during treatment.

**Discussion:** Our results indicate that reboxetine (4–8 mg/d) had no anticholinergic properties *in vivo*. The decrease of both, the relative and absolute LF-power in combination with a decrease of the LF/HF ratio, suggest a reduction of efferent sympathetic nerve activity, possibly due to a centrally mediated inhibition of reticular neurons.

## P02.278

### EFFECTS OF SILDENAFIL (VIAGRA®) ON CARDIOVASCULAR AUTONOMIC FUNCTION (CAF) IN MAN: PRELIMINARY RESULTS

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**Background:** In patients with psychiatric disorders treatment with psychopharmacological agents often causes various sexual dysfunctions. In particular, in males erectile dysfunction is one of the most frequently reported reasons for discontinuation of neuroleptics or antidepressants. It is well known that these drugs exert distinct influence on CAF, which in part may limit their cardiovascular safety, especially when multiple drugs were combined. Standardized measurement of heart rate variability (HRV) allows a quantitative assessment of the CAF *in vivo* (1). The effects of sildenafil on HRV have yet never been investigated. Thus, it is still unclear whether in psychopharmacologically treated patients with sexual dysfunction cardiovascular complications would arise which might result from a sildenafil triggered synergistic amplification of autonomic dysfunction. Therefore studies are needed to clarify the effects of sildenafil on CAF in detail.

**Methods:** This study is still ongoing; 15 men (planned n = 30), who received sildenafil from their urologists because of erectile dysfunction, had been recruited. The CAF test-battery included standardized measurements of the HRV during 5-min resting (1) and during deep-breathing, the 30:15 ratio, a modified Schellong-test and serial conventional ECG recordings at rest and during ergometric exercise (75 Watt) over a period of at least 10 minutes. The test-battery was repeated about 90 minutes after the patients had received a single oral dose of sildenafil (25–75 mg).

**Results:** Although upon questioning patients reported that they were in good physical health and fitness, detailed examination disclosed the presence of multiple somatic diseases (prior myocardial infarction, coronary heart disease, diabetic autonomic neuropathy, arterial hypertension). There is no preliminary evidence that sildenafil alters the HRV or any other of the ECG parameters in the healthy as well as in the medically ill subjects.

(1) Task Force. Heart rate variability. *Circulation* 1996; 93: 1043–1065.