

THE LONG-TERM IMPACT OF THE PHYSICAL, EMOTIONAL AND SEXUAL ABUSE OF CHILDREN: A COMMUNITY STUDY

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The associations between giving a history either of physical, or emotional, or sexual abuse in children and a range of mental health, interpersonal, and sexual problems in adult life were examined in a community sample of women. Abuse was defined to establish groups giving histories of unequivocal victimization. A history of any form of abuse was associated with increased rates of psychopathology, sexual difficulties, decreased self-esteem, and interpersonal problems. The similarities between the three forms of abuse in terms of their association with negative adult outcomes was more apparent than any differences, though there was a trend for sexual abuse to be particularly associated to sexual problems, emotional abuse to low self-esteem, and physical abuse to marital breakdown. Abuse of all types was more frequent in those from disturbed and disrupted family backgrounds. The background factors associated with reports of abuse were themselves often associated to the same range of negative adult outcomes as for abuse. Logistic regressions indicated that some, though not all, of the apparent associations between abuse and adult problems was accounted for by this matrix of childhood disadvantage from which abuse so often emerged.

BIOLOGICAL INVESTIGATIONS IN OCD AND TOURETTE SYNDROME

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A high prevalence of obsessions and compulsions is recognized in patients with Tourette-syndrome (TS). A comparison of a group of patients with TS and another with OCD, using the DSM III-R criteria showed significantly higher scores in obsessions and compulsions than a control group. However, it was possible to show specific differences of obsessive compulsive symptoms in OCD and in Tourette syndrome [1]. This investigation was repeated in other groups of GTS- and OCD patients. The results of the cross-validation will be presented. Moreover, we performed neuroendocrinological tests in both groups of patients. Since in OCD an alteration of the serotonergic neurotransmission is likely, we performed the fenfluramine-test in both groups of patients. Own investigations have shown, that in TS the growth hormone response to clonidine is blunted, these results pointing to a disturbance in the noradrenergic neurotransmission in GTS [2]. Therefore we compared the clonidine-test in 10 patients with OCD and 10 patients with TS. The results of the psychopathologic and neuroendocrine investigations, pointing to a psychopathological and pathophysiological difference between OCD and GTS will be presented. Moreover, some recent immunological data point to parallels between GTS and Chorea Sydenham. These results will be shown.

- [1] Müller, A. Putz, A. Straube, N. Kathmann: Obsessive compulsive disorders and Gilles-de-la-Tourette-Syndrome. *Differential diagnosis of organic and psychic obsessions and compulsions. Nervenarzt* 66: 372–378, 1995.
- [2] Müller, A. Putz, U. Klages, E. Hofschuster, A. Straube and M. Ackenheil: Blunted growth hormone response to clonidine in Gilles-de-la-Tourette-Syndrome. *Psychoneuroendocrinology* 19: 335–341, 1994.

SACCADIC EYE MOVEMENTS IN GILLES-DE-LA-TOURETTE-SYNDROME

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Gilles-de-la-Tourette's syndrome (GTS) is presumed to be an inherited disorder whose pathophysiology is still unclear. An involvement of the basal ganglia is suspected. Beside vocal tics, one of the main symptoms is the presence of motor tics. As eye movements are a specialized part of the motor system, we investigated whether they deviated in some way in GTS patients. It is known that in other diseases of the basal ganglia, such as Parkinson's disease (PD) and Huntington's disease, there are typical oculomotor symptoms, especially in the control of voluntary saccades. To study the control of saccades in GTS, different paradigms were used to elicit saccades, which were either externally triggered and visually guided or internally triggered and without visual target. GTS patients (n = 10) showed an increase of the saccade latency, especially in the internally guided saccades; an highly impaired performance of sequences of memory-guided saccades and a reduction of the peak velocity in the antisaccades. Overall the results were similar to those described with similar paradigms in PD patients.

This suggests that the cortex, especially the frontal cortex, is not activated in the normal way by ascending loops from the basal ganglia to the thalamus and the frontal cortex. The general function of the frontal cortex — frontal eye field, prefrontal cortex — does not seem to be impaired, because the performance of memory-guided saccades and the effect of a fixation target on saccadic latency ("gap effect") were normal.

DEPRESSIVE DISTURBANCES AS A CONSEQUENCE OF CEREBROVASCULARE DISEASE

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Our study covered a group of 160 patients with cerebrovascular disorders. According to GHQ and Hamilton scale for depression, a prevalence of 35–60% of medium to severe depression which lasted for 6 months to one year, was established. In our study we have seen a significant correlation to previous personal history. Subcortical atrophy and lesion of the basal ganglia of the left cerebral hemisphere. Only 50% of the patients with high score of GHQ were given antidepressive drugs and the remaining patients received combined therapy. Contrary to depressive disorders, mania is very rarely seen in cerebrovascular disorders. The clinical features and response to standard therapy are not different than in manic states in endogenous psychoses. There is a strong interaction with lesions of the limbic system of the right hemisphere. Predisposing factors in mania are especially the genetic component, subcortical atrophy, dysfunction of the frontal lobe and the length of hospitalization with good neurologic status. This last factor has a significant role in depressive disorders as well.

The authors underly the significance of the follow-up of the patients after the cerebrovascular insult in view of an increased incidence of affective disorders after 6 months. In view of this, a better cooperation between the neurologist and the general practitioner is needed.

FREQUENCY OF ICD-10 PSYCHIATRIC DIAGNOSES — AN INTERNATIONAL SURVEY

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Chapter V of the Tenth Revision of the International Classification