tor densities in SDAT were partially decreased compared to middle-aged controls, but increased in comparison to age-matched controls. IGF-I receptor densities were unchanged in aging and in SDAT. Tyrosine kinase activity, a key signal transduction mechanism common to both receptor systems, was reduced in SDAT in comparison to middle-aged and age-matched control groups. These data are consistent with a neurotrophic role of insulin in the human brain and a disturbance of insulin signal transduction in SDAT brain and favor the hypothesis that insulin dependent functions may be of relevance for the development of neurodegeneration in sporadic SDAT.

Preclinical evidence for neuroprotection in Parkinson's disease

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The cause of chronic nigral cell death and the nature of the pathological process underlying clinical deterioration in Parkinson´s disease (PD) remains elusive, although the possibility that accelerated ageing, environmental factors, genetic factors, viruses, autoimmune dysfunc-tion, and other factors contribute to the cause of PD has been considered over the years. Partial elucidation of the processes that underlie the selective action of neurotoxic substances, such as 6-hydroxydopamine (6-OHDA), glutamate, kainic acid, quinolinic acid, or 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), has revealed possible molecular mecha-nisms for neurodegeneration. Hypotheses regarding the neurotoxic mechanisms of these substances have evolved based on our understanding of the pathogenesis of cell death in neurodegenerative disorders and have been the rationale for neuroprotective approaches. In co-operation with Peter Riederer we have demonstrated that monoamine oxidase type B (MAO-B) inhibitors such as selegiline and rasagiline, dopamine agonists such as cabergoline and lisuride, and calcium antagonists such as nimopidine exert a neuroprotective effect at the cellular, neurochemical and functional levels in experimental models of PD.

Klüver-Bucy syndrome in men

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In 1937 Klüver and Bucy [1] were first able to demonstrate gross behavioural changes in Macaca rhesus following bilateral removal of major portions of the temporal lobes including most of the rhinencephalic structures (uncus and hippocampal gyrus, nucleus amygdalae). A first description of Klüver-Bucy symptoms in form of total memory loss and signs of hypersexuality was published by Brown and Schäfer 1888 in monkeys after bilateral temporal lobectomy [27].

The behavioural syndrome of the animal now eponymously bearing the authors' names was characterized by the cardinal symptoms psychic blindness or visual agnosia, intensive oral tendencies, extreme distractability or reagibility on visual stimuli, decrease of aggressiveness and loss of fear, hypersexuality and changes in dietary habits [2–4]. While appearing unable to recognize the objects of their surrounding the animals attended to every visual stimulus with a seemingly irresistible impulse to touch every object in sight and to examine all objects by mouth. Aggressiveness was markedly decreased and hypersexuality was present in forms of heterosexual, homosexual and autosexual behaviour. Following these experiments, several authors drew attention to similar behavioural changes in human pathology.

The probably most striking equivalent of a Klüver-Bucy syndrome in human pathology was reported by Terzian and Dalle Ore [5] in a young male patient with intractable temporal lobe epilepsy in whom an almost complete bilateral temporal lobectomy had been performed. He developed bulimia, hypersexuality was present together with placidity, loss of affect and memory, but hyperorality was missed. In the following years further reports on Klüver-Bucy-like syndromes in humans were generally based on the notion that oral tendencies comprise the cardinal manifestation in human Klüver-Bucy syndrome [6–8].

The etiology of human cases is quite diverse and includes degenerative diseases of the brain [9–11], cerebrovascular disease [10], encephalitis, especially of herpes simplex etiology [12–17], traumatic brain injury [7, 18–20], surgical brain lesions, mainly for treatment of temporal lobe epilepsy [5, 6, 21] and also rare cases of metabolic disturbances of the brain [22, 23]. Gerstenbrand [19, 20] could show that a phase with typical Klüver Bucy symptoms is nearly obligatory in the remission state of an apallic syndrome with different etiology. A Klüver-Bucy syndrome can elicit by longer treatment with neuroleptic drugs.

Neuroanatomical and neurophysiological studies of the last decades have shown, that the limbic system is the most important morphologic substrate for the psychoaffective behaviour and the vegetative organic functions seen in Klüver-Bucy syndrome. The anatomical description "le grand lobe limbique" was given by Broca in 1878 [24]. Only after comparative anatomical studies given by Akert et al. [25] in 1963, Brocas' description, given hundred years ago, was taken interest of. Papez [26], described in detail fibre correlations between the hippocampus, gyrus cinguli, anterior part of the thalamus and the corpus mammillare were described in detail, and set up the theory, that the fuctional circle of the limbic system could be the neuronal correlate of affect, emotion and mechanism of expression.

Aichner 1984 [27] described in the complete Klüver-Bucy syndrome in men the following seven symptoms. Hyperorality and hypersexuality are the leading characteristics. *Hyperorality* is the irresistible impulse to grasp every object in sight, taking it into the mouth, trying to bit, chew and swallow it. A piece of soap may be eaten like an apple without disgust, a knife may be bitten without recognition of the possible injury (visual agnosia). In *Hypersexuality* there are manipulations with the genitals (autosexual acts, as well as hetero- and homosexual tendencies), without feelings of shame. *Hypermetamorphism* introduced by Klüver and Bucy describing the copmpulsive attention their attention to every object, coming to the visual field [11], mostly combined with hyperorality [27]. *Amnesia*, firstly not mentioned by Klüver and Bucy in the animal experiment, is in human being an obligatory symptom [27], with severe deficits in memory [28, 29]. Patients with a Klüver-Bucy syndrome show antero- and retrograde-amnesia for the acute accident and lack of learning possibilities. Motivation and affect are blunted, combined with euphoric mood, called as *Placidity*, anxious behaviour is missed, aggressiveness can appear in several patients. *Bulimia* or *hyperphagia* can be found only in 50% of patients with Klüver-Bucy syndrome. The *visual agnosia* is a leading disturbance in the monkey experiment, called "psychic blindness" by Klüver and Bucy [1].

Klüver-Bucy syndrome can be seen as complete or partial. The Klüver-Bucy syndrome in children shows instead of sexual deviation an increase of demand for tenderness. Aggressive tendencies are rare and temporary.

In the course of a Klüver-Bucy syndrome it has to be divided between a transient and a persistent form. The transient form shows

a preface, a full state and a late phase [27, 19, 20]. The transient form is typically after traumatic and hypoxic apallic syndrome. The appearance of Klüver-Bucy symptoms in the remission course of an apallic syndrome after an acute brain damage, indicating a positive prognostic sign with the explanation that the brain functions are installed in the limbic level after the decrease to the meso-diencephalic functional level during the full stage. Some patients with the transient form show Klüver-Bucy symptoms for years. The persistent form of a Klüver-Bucy syndrom can be seen after encephalitis sometime in combination with Korsakow symptoms.

Partial Klüver-Bucy symptoms can be observed in progressive brain diseases (Pick disease, Alzheimer disease etc.) with various symptoms.

Summarising the complete Klüver-Bucy syndrome of the animal model is rarely observed with human beings. The Klüver –Bucy syndrome men is mostly observed as a partial symptom, for this diagnose three to four single symptoms have to be concerned including the cognitive, emotional and behavioural elements [27]. The Klüver-Bucy syndrome in men is reversible mostly, partial defect symptoms can be seen in 15% (Aichner, [27]). The Klüver-Bucy syndrome after severe brain injury shows a dynamic course with preface, full state and late phase. In neuropathological findings there are local lesions in the limbic system bilateral but in combination with a disinhibiton phenomena due to lesions especially of the frontal lobe. Biochemical investigations have to be enforced, more detailed structural and electro-neurophysiological examinations have to be initiated.

The intensive study of the Klüver-Bucy syndrom is an initial step for understanding the great importance of this neurological disorder and its influence to the emotions and instinctive life of human beings with all the behavioural irritations.

After all documents analysed by Ivan Lesny [32] the Roman emperor Calligula as the little boots called by the legionnaires of his father Germanicus has developed a defect state of a Klüver-Bucy syndrom, Calligula survived a severe form of an encephalitis, probably a herpes simplex encephalitis. After the severe attack lasting some weeks the whole Rome was praying to their different gods the young and hopeful emperor was in his behaviour absolutely changed. His sexual escapades and the unbelievable attitudes as the first man in the antique world are still till today a special chapter in our history books.

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Aspects of dopaminergic psychosis

R. Horowski

be avoided (high risk of severe akinesia) as well as benzodiazepines, and in the case of clozapine, initial weekly hematological controls are necessary to avoid agranulocytosis. Clozapine, which may also act on PD tremor, has strong anticholinergic properties and thus can cause delirium, another severe but rare complication in PD, but so far all newer atypical neuroleptics are less successful. As all dopaminergic therapies suppress REM sleep (to be followed by rebound), dopaminergic psychosis might turn out to be a drug-induced REM-disorder.

HIV-associated dementia-studies on rhesus monkeys

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HIV invades CNS subcortical areas, particularly the dopamine-rich basal ganglia and induces a subcortical dementia, the HIV-associated dementia (HAD). The pathophysiological process which leads to HAD is unclear, since HIV does not infect neurons. To study the role of the dopaminergic system in HAD we used the well-established simian immunodeficiency virus (SIV) infected rhesus monky model. This model is suitable for the investigation of the pathogenesis of AIDS-related CNS disorders.

We infected rhesus monkeys (macacca mulatta) with the virus monocyte-tropic strain SIVmac251. Surprisingly, within two months after SIV infection, a marked impairment of the dopaminergic system was present. To restore observed DA deficits we administered dopaminergic drugs. Although the pharmacological treatment increased DA concentrations, a wide-spread vacuolisation became apparent, and SIV encephalitis was accelerated. These pathological findings were accompanied by an elevation in viral load throughout the brain. Treatment with dopaminergic agonists and antagonists implied that the histological findings were not

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Abstracts of the Symposium

"Interdisciplinary approaches for the understanding of neuropsychiatric disorders"

Passau/Linz/Krems, April 28–30, 2002

Parkinson's disease, namely frontal lobe deficits, appear to be related to a loss of central dopaminergic function. In addition, cognitive deficits may be present prior to the clinical onset of Parkinson's disease.

Neuroprotection in Parkinson's disease: PET foundations

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Parkinson's disease is usually a continously progressing disease in which the decline of the nigro-striatal dopaminergic neurotransmitter pathway plays a dominant role. In former times the view was held that the nigral dopaminergic neurons needed to degenerate at least 80% of normal control values before clinical signs and symptoms would become obvious. If that were true that would make treatment designs which aim at neuroprotection of the remaining nigral dopaminergic neurons an unrewarding option right from the start. However, tracer kinetic data obtained through PET and SPECT revealed that the picture is much more differentiated. When parkinsonian signs and symptoms start to appear it is indeed likely that the endogenous dopamine production in the striatal dopaminergic nerve terminals is very low, but that the pool of nerve terminals itself, thus the structural elements, are still present to a large extent

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