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J. Mendlewicz, Brussels and H. M. van Praag, Utrecht

S. Karger Basel Mnchen Paris London New York Sydney

Temporal Lobe Epilepsy, Mania, and Schizophrenia and
the Limbic System

Symposium presented during the 3rd World Congress of Biological Psychiatry,
Stockholm, June 28-July 3, 1981

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Mania, and Schizophrenia
and the Limbic System

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The authors and the publisher have exerted every effort to ensure that drug selection and dosage set forth in this text are in accord with current recommendations and practice at the time of publication. However, in view of ongoing research, changes in government regulations, and the constant flow of information relating to drug therapy and drug reactions, the reader is urged to check the package insert for each drug for any change in indications and dosage and for added warnings and precautions. This is particularly important when the recommended agent is a new and/or infrequently employed drug.

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October 1981
Werner P. Koella
Michael R. Trimble

Introduction - Reasons and Aims of this Symposium

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Schizophrenia, mania (the 'high' phase of manic-depressive illness) and temporal lobe epilepsy (the complex partial seizures) usually are looked upon as three separate diseases. Still there can be little doubt that any two, if not all three, of these nosological entities exhibit a good deal of similarity in their overt psychopathological manifestations, or - to put it differently - reveal an often considerable overlap in their symptomatological profile. This is true mainly for the 'acute' phases of these afflictions, and sometimes it is extremely difficult, if not impossible, to come to a diagnosis initially - which even then, later after lengthy and involved examinations, may still turn out to be wrong.

Furthermore, patients with epilepsy, in particular those with temporal lobe epilepsy, in the interictal interval often reveal psychiatric symptoms which may resemble mania, or assume the presentation of a florid, schizophrenia-like psychosis. Associations between such psychopathologies and epilepsy have been observed for several hundred years, but have only in this century, with the introduction of newer nosological systems in psychiatry and epilepsy, become crystallized. The early writers discussed concepts of the 'epileptic personality', an idea that mutated with time, and which recently has become once again the centre of much discussion. Originally characterized by a variety of adjectives, mostly pejorative, such a personality was seen as the inevitable consequence of having fits. It then assumed, in the 'psychosomatic era' of the midcentury, the role of a predisposition, the epileptic personality, on account of various drives, motivations and conflicts, being prone to epileptic seizures. This was followed by a period of rejection of the whole concept, a position still argued by some today, although renewed interest in the idea of an organic personality change which occurs secondary to chronic temporal lobe lesions, of which complex partial seizures represent the paradigm, has been seen when more sophisticated methods of investigating such changes were developed. Thus the idea that patients with epilepsy suffer from not only seizures, but also from aberrant behaviour traits, on account of their brain lesions, is still actively being investigated, and attempts are being made to define which patients with which lesions are most prone to which disorders. The concept of personality change due to temporal lobe lesions has, however, broadened to include other disorders, especially the schizophreniform presentations and its relationship to similar disorders in non-epileptic populations.

In addition to these clinical relationships, in the past 30 years it has
been possible to define overlap between at least pairs of the above-mentioned triad in therapy in the sense that identical drugs are used to combat any two of the three afflictions. Neuroleptics have been and are used in the treatment of psychosis and mania, and - as found quite recently - carbamazepine, the drug of choice for temporal lobe epilepsy, may turn out to be useful in the treatment of mania.

If we accept the notion that a particular symptom - or most generally speaking, a piece of aberrant 'internal' and/or 'external' behaviour - is the manifestation of a specific aberrant neural performance in the system(s) that form(s) the organizational basis of that behaviour, we must conclude that the three disease entities overlap also in their pathogenesis or neuropathology. Several of the participants of this symposium and many others, on a variety of occasions, have pointed out that this (at least partial) common pathology involves structures within the limbic system including the hippocampus, the amygdala, the septum and basal limbic forebrain such as the nucleus accumbens and the olfactory tubercle and some of the interconnecting pathways.

On account of these relationships we thought it timely to bring together clinicians and basic researchers to discuss these issues. As a background for a better understanding of common pathophysiological features for temporal lobe epilepsy, psychosis and mania, Drs Koella and Andersen will review some of the more pertinent aspects of the gross psychophysiology of the limbic system, and the 'microscopic' physiology of the hippocampus, respectively. Their data offer important information for the physiological background of drug therapy.

Some clinicians from the field of neuropsychiatry will discuss and define again the questions of coexistence and overlap of symptoms in temporal lobe epilepsy and psychosis, and the 'therapeutic' overlap between temporal lobe epilepsy and mania. Dr. Trimble, Dr. Sherwin and Dr. Stevens will - from their particular point of view - present data from their own research and from the literature, that hopefully will allow further crystallization of these complex issues. These chapters explore those groups of patients most susceptible to develop behaviour disturbances in epilepsy, highlighting some of the methodological difficulties of past studies. The issue of what type of epilepsy is related to what type of psychosis is elaborated on, and some recent data emphasizing the association between left-sided or dominant temporal lobe lesions and schizophreniform psychosis is presented. The link between affective, in particular manic-depressive disorders

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and epilepsy is discussed in detail by Post. The possible role of kindling as a model for the development of psychosis in patients is introduced, as well as a theoretical basis for the use of certain anticonvulsants in the treatment of non-epileptic disorders in which limbic system dysfunction may occur is discussed.

When the two editors started to organize the workshop presented at the Third World Congress of Biological Psychiatry in Stockholm in July 1981, on which this book is based, Dr. Robert Heath of Tulane University (New Orleans, La.) was also invited to present his views on the role of the limbic system in the pathogenesis of psychosis. Unfortunately, Dr. Heath was unable to attend the meeting, but in view of his great contributions to this field the editors wished him to participate in this venture and thus have included a chapter by him.

The results presented support the idea of a link uniting psychosis, mania and epilepsy. We hope that the material assembled in this book will be of value, not only in introducing new ideas and avenues for further research, but also in emphasizing the biological basis for human behaviour and the role of somatic structures, especially the limbic part of the brain in the pathogenesis of behaviour disorders. 'The myth of mental illness' will some day disappear and we would have liked to have played a small part in that process.