

ments — an assistance to help the rational “part” of the psyche, its self at the highest integration level of the reality principal; to gain ground on the pathological “part”.

But conceiving the psyche in healthy and pathological parts is not a modern psychiatry invention. This metaphor, reifying but necessary to reflection, found an essential step of its advent in the maturation in Esquirol’s work, of the Monomania concept or its synonym, the “partial madness”.

In 1818, Esquirol describes it in a clip of the “Panckoucke”. However, in both clips held in his treatise “Des Maladies mentales” published in 1838, other forms of the partial madness are proposed by the same author. Then, the madman is no longer a stranger to himself and it is possible to aim the healthy part in him. We are far away from the madman so totally different from the healthy man, that he is rejected out of the city walls.

Does the psychological confusion concern the whole mind or only a part of it? Isn’t the debat out of date?

NR18. Clinical aspects of schizophrenia

Chairmen: F Holloway, E Joyce

PSYCHOEDUCATIONAL TRAINING IN SCHIZOPHRENIC PATIENTS

Wilhelm Classen¹, Peter Schwenkmezger², Marion Heinrich², Catherine Schäfer-Krajewski¹, Sybille Boesken¹. ¹ *Center for Psychobiological and Psychosomatic Research, Department of Biological Psychiatry, University of Trier, Friedrich Wilhelm-Str. 29, D-54290 Trier, Germany;* ² *Department of Psychology, University of Trier, D-54298 Trier, Germany*

Many therapeutic techniques are involved in psychoeducational programs (Wright & Schrodt, 1989). The main goal is to reduce relapse rates of psychoses. (Hornung & Buchkremer, 1992). Psychoeducational interventions require patients to be competent to discuss their illness, their behaviour and preventive strategies.

In our study three group of patients: 1. a training groups of in-patients (n = 15; 10 sessions of 90 minutes, 1 session a week), 2. an out-patient group (n = 13), 3. a group of patients awaiting treatment (n = 8) were investigated with respect to psychopathologic symptoms (BPRS), subjective emotional state (BIS), understanding of their illness, protective strategies and attitude towards drugs.

As compared to the control groups psychopathologic symptoms, understanding of illness and protective factors improved in the training group, but their attitude towards drugs didn’t change. Implications of these findings will be discussed.

In a long-term panel possible reduction of relapse rates will be studied.

THE ATTRIBUTION OF INTENTIONALITY, CAUSALITY AND DISPOSITION BY DELUDED PATIENTS

D.J. Done, V. Millard, H.A. Pattinson, M. Papanastasiou, and the Hertfordshire Neurosciences Research Group. *QEII Hospital, University of Hertfordshire, Wellhouse Trust, Hertfordshire, AL10 9AB, England*

Social reasoning has been extensively studied by psychologists but there has been limited application of methods and theory to understanding the abnormal social reasoning that is central to many

delusions. We report results from a study in which deluded patients with a diagnosis of schizophrenia and normal control subjects watched video vignettes depicting negative outcomes occurring to one actor, with the intent of the provocateur manipulated to be either accidental, ambiguous or on purpose. The prediction that patients with delusions will infer intent more than normal controls was supported by the data but this did not result in any increased likelihood of making negative disposition statements about the provocateur as predicted by the Correspondent Inference Theory theory of Jones and Davis [1]. There was also no increased tendency by the patients to make person rather than situation attributions when asked to describe what had caused the outcome, unlike results reported in other studies. Deluded patients were also less affected by the intent manipulation suggesting a failure to perceive salient social information. The method used demonstrates that social reasoning in deluded patients can be readily investigated and the results suggest inappropriate use of information when making judgments about intentionality.

[1] Hewstone M. (1989) Causal Attribution. Oxford, Blackwell.

? A NEW AND FAMILIAL VARIANT OF SCHIZOPHRENIA

G.A. Doody, W.J. Muir, E.C. Johnstone, D.G.C. Owens. *University Dept. of Psychiatry, Royal Edinburgh Hospital, Morningside Park, Edinburgh. Scotland. EH10 5HF*

It is widely believed that the point prevalence of schizophrenia in individuals with mild learning disability is three times that of the general population. This large Edinburgh study seeks to explore reasons for this observation. Three sex and age matched populations are under study; subjects with a dual diagnosis of mild learning disability and schizophrenia (obtained from a National register, N = 20), subjects with DSMIII-R schizophrenia and normal premorbid I.Q. (randomly matched from the Lothian Psychiatric Case Register, N = 20) and subjects with mild learning disability alone (N = 17). A detailed family history has been obtained in 85% of cases.

40% of the schizophrenic group have a family history of schizophrenia in first or second degree relatives. One schizophrenic subject also has a first degree relative with a dual diagnosis of schizophrenia and mild learning disability. Only one subject with learning disability alone has a psychotic relative.

Over 60% of the dual diagnosis group have a family history of schizophrenia. 50% of these subjects with schizophrenic relatives have a family history of schizophrenia alone, and the remaining 50% have a family history of a dual diagnosis also occurring in relatives.

Karyotypic analyses of 16 of the 20 dual diagnosis probands show chromosomal variants to be common. The proband with learning disability, who has psychotic relatives, also shows evidence of chromosomal variance.

We wish to suggest that the excess of schizophrenia in the mildly learning disabled population may be partially explained by the existence of a highly familial sub-type of schizophrenia. The phenotypic appearance of which may be polymorphic in families who are multiply affected with mild learning disability, schizophrenia and a dual diagnosis. Genotypically, this sub-type of schizophrenia may be associated with chromosomal variance.

AUDITORY HALLUCINATIONS IN PROFOUNDLY DEAF SCHIZOPHRENIC PATIENTS: A PHENOMENOLOGICAL ANALYSIS

M. Du Feu, P.J. McKenna. *Queen Elizabeth Psychiatric Hospital, Mindelsohn Way, Edgbaston, Birmingham, and Fulbourn Hospital, Cambridge CB1 5EF, UK*

Profoundly deaf individuals who develop schizophrenia sometimes claim to hear voices. Proposed explanations for this counter-intuitive

phenomenon include that such patients are really experiencing thought insertion, hallucinations of vibration, etc, or that only patients who become deaf after speech is acquired hear voices.

Sixteen patients meeting RDC criteria for schizophrenia were interviewed using the 9th edition of Present State Examination (PSE). All were profoundly deaf, having no ability to hear spoken language, and in most cases no intelligible speech.

10 (62.5%) gave definite accounts of hearing voices, with description of content. Both the second and third person and true and pseudohallucinations were described. Six patients experienced non-verbal hallucinations and three described thoughts spoken aloud or thought broadcasting. Verbal auditory hallucinations were not confined to patients with an onset of deafness after speech may have been begun to be understood. Three patients were profoundly deaf from birth, and almost all of the remainder in whom information was available were diagnosed as deaf before the age of 18 months.

These findings confirm that profoundly deaf schizophrenic patients, who may never have experienced spoken language, report hearing voices to much the same extent as hearing patients. They also experience other auditory symptoms. Explanations in terms of misattribution of other symptoms or restriction of the symptom to those who were not prelingually deaf are insufficient to account for this phenomenon.

PSYCHOEDUCATIONAL INTERVENTION ON THE RELATIVES' KNOWLEDGE ABOUT SCHIZOPHRENIA: A TWELVE MONTH FOLLOW UP

M. Economou, M.G. Madianos. *Psychosocial Rehabilitation Unit, Community Mental Health Center, University of Athens, 14 Delou Str., 161 21, Athens, Greece*

A psychoeducational intervention, which was part of a broader psychosocial programme of our Rehabilitation Unit, was offered to key relatives of patients suffering from schizophrenia.

50 relatives related to 38 persons suffering from schizophrenia, living in the community (experimental group) participated in an education programme of 20 hours on a weekly basis. The relatives of the experimental group were assessed by a questionnaire eliciting information on the knowledge about schizophrenia three times during the study period, before and after the end of the education programme and one year later in a follow up assessment.

A control group, comprising of 30 relatives having not exposed to any intervention, was assessed by the same instrument into three also consecutive periods.

In the case of the control group of relatives, no significant changes between the three assessments were observed.

The relatives of the experimental group showed a significant improvement of knowledge about schizophrenia and a significant increase of optimism about prognosis of illness at the end of the educational programme. Twelve months later, at the follow-up assessment, the same relatives exhibited the same patterns of knowledge and attitudes acquired through the education.

THE USE OF THE EXPRESSED EMOTION INDEX AS A PREDICTOR OF OUTCOME IN FIRST ADMITTED SCHIZOPHRENIC PATIENTS IN A FRENCH SPEAKING AREA OF SWITZERLAND

Ph. Huguelet, S. Favre, S. Binyet, Ch. Gonzalez, I. Zabala.

Summary: A 5 year prospective study of 44 first admission schizophrenic patients was conducted in Geneva, in order to evaluate the prognostic value of Expressed Emotion (EE). The predictive power of the EE index was tested on 3 variables of outcome: relapse rates, social adaptation and hospital stays. The EE index and the out-

come measures tended to be associated. After the third year, patients living with high EE relatives were significantly more maladjusted and relapsed more than those living with low EE relatives. At intake, the patients presenting more premorbid features lived in high EE households. Our results show that initial measure of EE in a first episode cohort is predictive of outcome over a five year period. This may not be causal, as it cannot be excluded that poorer premorbid functioning alone may result in poorer outcome, and may also elicit high EE in the relative.

LATE ONSET SCHIZOPHRENIA — A VALID ENTITY? AN EMPIRICAL STUDY ON RISK FACTORS, PSYCHOPATHOLOGY AND COURSE

A. Riecher-Rössler, H. Häfner, P. Munk-Jørgensen. *Central Institute of Mental Health, J 5, 68159 Mannheim, Germany*

"Late onset schizophrenia" was analysed on the basis of

- a direct investigation of 267 first admitted patients from a defined catchment area in southwest Germany (ABC Study; Häfner et al. 1992) and

- case register data on the course of all 1,423 Danish patients first admitted in 1976.

Results: Late onset schizophrenia did not markedly differ from early onset schizophrenia in terms of *psychopathology*. Especially nuclear psychotic symptomatology was surprisingly similar. Minor differences in unspecific symptoms and illness behaviour could be explained by general influences of the higher age rather than distinct pathogenetic processes.

As regards *risk factors* we found hints of a lower familial risk in late onset as compared to early onset cases.

The *course* of late onset cases seemed to be milder when number and duration of hospitalizations over 10 years were compared.

Interestingly *psychopathology* and *course* of late onset women were significantly worse than that of late onset men.

Conclusions: Late onset schizophrenia essentially seems to belong to the same group of diseases as early onset schizophrenia. But *psychopathology* and *course* are influenced by the higher age of the patients and many age-specific characteristics. The outbreak of the disease seems to be later (a) in women and (b) if there is a lower genetic loading. Based on earlier findings pointing at a protective (antidopaminergic?) effect of estrogens in schizophrenia, we suggest that in some late onset women with a relatively high (genetic) vulnerability the outbreak of the disease is delayed by estrogens. When this protective factor fades off after menopause, there occurs a relatively severe form of the disease as regards *psychopathology* and *course*.

VIOLENT BEHAVIOUR IN SCHIZOPHRENICS — CAUSED BY SCHIZOPHRENIA ITSELF OR BY PREMORBID BEHAVIOUR DISORDER?

T. Steinert, A. Voellner, K. Hermer. *Dep. of Psychiatry I, University of Ulm, Weissenau State Mental Hospital, mailbox 2044, D-88190, Ravensburg, Germany*

Violence generally is the result of complex interactions between the personality of the actors and the dynamic development of a situation; in the case of schizophrenia or psychic disease in general this disease is added as a third factor, whose significance for the origin of violent behaviour is not very clear: Is schizophrenia associated with an elevated rate of violent behaviour due to psychopathology itself (e.g., delusions, command hallucinations) or due to poor social adjustment or does violent behaviour predominantly occur in individuals who showed antisocial behaviour styles already in their premorbid personality? To find answers to this question, 26 violent