Facoltà di Medicina e Chirurgia

50 YEARS OF SCHIZOPHRENIA RESEARCH: A PERSONAL VIEW

Gerd Huber

Laurea Honoris causa
in Medicina e Chirurgia

Parma, 13 dicembre 2005

Lectio doctoralis di Gerd Huber
in occasione del conferimento
della Laurea Honoris causa in Medicina e Chirurgia

50 YEARS OF SCHIZOPHRENIA RESEARCH: A PERSONAL VIEW

Gerd Huber

After 12 years of mental decline the time after the 2nd World War has been a very fruitful by the psychopathology of Kurt SCHNEIDER determined decade (1945-1955). No longer war, the feeling, once more got off, and then a hard time as a student.

In Heidelberg I was going among others to the lectures of Alexander MITSCHERLICH, the psychoanalyst, Viktor v. WEIZSÄCKER, the psychosomatic, then also of Kurt SCHNEIDER, and, above all Karl JASPERS, the philosopher, psychologist and psychopathologist. I had already acquired at the beginning of my clinical studies the 4th edition of JASPERS' »General Psychopathology« of 1946 [84] (**slide** – 2 papers), and, immediately after its publication (1948 – [34]) the »Verstehende Psychologie« (»Understanding Psychology«) of GRUHLE.

Because even unpaid jobs at that time could scarcely to be got, I was glad, as K. SCHNEIDER, did promise me a position as assistant in 1949 and 1951. Before I wanted to work at Max Planck Institute in Munich with Willibald SCHOLZ, the neuropathologist,

In Munich I came for the first time in contact with the schizophrenia research. I intended to show that the brain findings, described by the VOGTs, can support the illness hypothesis in schizophrenia. But, I found to my big disappointement that the described alterations in the medial nucleus of the thalamus, also occur in not pathological cases with the same frequency and quality. Much later, after we had described, based on neuroradiological and clinical findings, a basal ganglia syndrome in schizophrenia and a defect of the thalamic filter (HUBER 1957, 1964, 1971 – [39, 44, 47]) and had named a subgroup of schizo-

phrenia as »limbopathy«, B. BOGERTS could demonstrate by postmortem studies an atrophy of amygdala, hippocampus, pallidum internum and periventricular structures of the 3rd ventricle; what could also speak for my hypothesis of disorders of subcortical-cortical circuits [6, 8, 39, 44] (**slide**).

Kurt SCHNEIDER, who only after the war in 1946 changed from Munich to the chair at the University Clinic in Heidelberg, unfortunately already in1955 became Professor emeritus. The follower of K. SCHNEIDER (since 1955), Walter v. BAEYER told to me that the classical psychiatry has been finished with K. SCHNEIDER, the future would be the »«aseinsanalyse" in the direction of HEIDEGGER, HUSSERL and BINSWANGER, with it he also was occupied writing papers about the »Begegnung« in psychiatry.

How was the situation of schizophrenia research in 1950? In 1932 GRUHLE had summarized in the famous Heidelberg Schizophrenia Volume [33] (**slide**): The schizophrenia represents an endogenous organic disease; because the psychology would be largely investigated, could shed some light on the matter most likely the discovery of new somatic symptoms.

20 years later, the change in the view of schizophrenia has been revealed very clear in the comprehensive review of schizophrenia of Manfred BLEULER in 1951 [2] (**slide**): Neuropathology and neurophysiology could not provide, as he wrote, positive contributions, the results of the family research could no longer be regarded as conclusive arguments of heredity; all classical assumptions of the schizophrenia doctrine and, especially, that schizophrenia would be a somatically conditioned illness and not psychogenic, would be shaken severely. Nearly at the same time, on the 1st International Meeting for Neuropathology in Rome (1952), the opinion became generally accepted, that pathological changes of the brain could not be expected in schizophrenias. Just in these years I had begun my neuroradiological-clinical investigations of the brain structure in schizophrenias [35, 36, 39] (**slide**).

I proceeded in the 50s from the view that schizophrenias have a pathological somatic basis and cannot be understood as a »riddle of the human being«, unapproachable for the methods of the scientific medicine, as Walter

SCHULTE, the follower of Ernst KRETSCHMER in Tübingen, did balance the World Congress of Zurich »The group of schizophrenias« (1957). For me was, in contrary to the main trends of psychiatry of that time, the search for empirical indications of the somatosis hypothesis in schizophrenias, an aim of research having priority [47, 55, 57]. I tried to assóciate findings gained with the available somatic methods with clinical syndromes and the course of the disorder [17, 28, 39, 43, 79, 83] (**slide**).

These investigations went already since the monograph of 1957 »Pneumencephalographic and psychopathological pictures in endogenous psychoses« [39] hand in hand with the gradual development of the basic symptom concept (BSC) [38, 42, 45] and our course- and early recognition research [18, 59, 64, 77, 86] (**slide**).

I originated with three observations, made at the Heidelberg Clinic. These were (1.) the “cenesthetic schizophrenia” (1957); (2.) the “asthenic pure defect” [39, 42, 45, 46] (**slide**) and (3.) lethal catatonias, patients who were diagnosed clinically as genuine schizophrenias [37, 41]. These cases only could be separated as symptomatic from the idiopathic schizophrenias by neurohistopathological findings of post-mortem examinations and, e.g., diagnosed as sporadic, atypical encephalitis [37, 40] (**slide**). As in epilepsy, and especially temporal lobe epilepsy [49, 50], we could shown that schizophrenic syndromes and all individual schizophrenic symptoms, including the basic symptoms, occur also in definable brain diseases [50, 75].

Cenesthetic Schizophrenia

I saw in the early 50s many diagnostically unclear patients with psychopathologically more or less peculiar disturbances of bodily sensations, who only in later course developed schizophrenic, usually after short time remitting psychoses. The prodromes of this cenesthetic type, which I described on the basis of 50 patients, lasted until the transition into the first psychotic manifestation 7 years in average [38, 46, 65] (**slide**). The type had a pilot function for the development of the basic symptom concept, because in its course the basic sympto-

matology determined as well the prodromes before the first psychotic episode, as after that also the postpsychotic pure defect syndromes, into which two thirds of cenesthetic schizophrenias terminate; then, also because with this type the first time has been observed that from initially quite uncharacteristic basic symptoms (level 1 basic symptoms) qualitatively peculiar basic symptoms (level 2 basic symptoms) and then distinct psychotic symptoms, i.e. bodily hallucinations are arising. Our follow-up studies have demonstrated that the positive psychosis and nonpsychotic, pre- and postpsychotic prodromal and residual basic stages belong together, represent only different stages of the course of the schizophrenic disorder. Next to the cenesthesias also other categories of basic symptoms, namely the central-vegetative and dynamic and cognitive perception disorders have been at first described with the cenesthetic type [26] (**slide**). The prodromal early initial stages of schizophrenia were investigated in Germany by MAYER-GROSS [99] and later by Klaus CONRAD [7] and child psychiatrists, in Japan by NAKAYASU [100] (**slide**). In Italy the BSC has been dealt with and further developed by MAGGINI and his group [95, 96, 97, 98] (**slide**). K. KOEHLER and H. SAUER [92, 93] described the BSC in their papers »HUBER's basic symptoms: Another approach to negative psychopathology in schizophrenia« and »JASPERS' sense of presence in the light of HUBER's basic symptoms and DSM-III« (**slide**).

The »Pure Defect«

In the nonpsychotic cross-section picture of the pure defect also K. Schneider could not recognize any hints to a schizophrenic disorder. Only, if the case history of such patients yielded psychotic episodes many years dated back, or patients, who were hospitalized because of florid psychosis, at later catamnestic investigations did show the same uncharacteristic pseudoneurasthenic syndromes, became the assumption of a nonpsychotic basic stage or residuum of a schizophrenia possible. We described these psychosyndromes as »asthenic« [38, 39] or »pure defect« [42, 45, 59] (**slide**) [26, 27, 65, 77, 81]. It is determined by manifold dynamic, cognitive and cenesthetic basic symptoms, presented with the aid of self-delineations of the patients in the BSABS [26, 27].

Neuroradiological findings and limbic system

In patients with “pure defect” we described in pneumencephalogram, then with echoencephalogram and computed tomography (CT), neuroradiological changes, explained as brain atrophy or brain hypoplasia (neurodevelopmental alterations) [24, 35, 36, 39, 42, 44] (**slide**). According to Mantosh DEWAN [9] by our studies of at the beginning 195 and later further 430 patients for the first time has been established a correlation between psychopathological and somatopathological findings in schizophrenias, that is in »pure residual syndromes«; moreover a parallel running progression of neuroradiological morphological and psychopathological changes (**slide**). These findings together with the topical predilection of the alterations preferring the third ventricle and the sections of the lateral ventricles close on the basal ganglia, spoke for a relation to the schizophrenic illness. Our findings, the »neuroradiological basal ganglia syndrome« [44] and the quantitative-morphometric and MRI-changes of sections of limbic system, are indications, as we hypothesized in 1973 (**slide**), that at least for a subgroup of schizophrenias disturbances in limbic key structures are of importance, which can explain basic symptoms and first rank phenomena, arising from distinct cognitive basic symptoms [26, 27, 39, 51, 52, 57, 81, 85].

Basic Symptom Concept (BSC) and long-term course. Bonn-Study

In spite of his programmatic statement of 1951, in which Bleuler the conception of schizophrenia as brain disorder regarded as out of date, he carefully paid attention to all new findings, that seemed to be important, even if they were not in line with his own opinion. Hence, ensued in the next decades a continuous discussion of us with him, also and particularly with reference to the basic symptom concept and the long-term courses with our common paper »The long-term course of schizophrenic psychoses. Joint results of two investigations« [5] (**slide**).

In this paper, BLEULER and we stated [5, 51, 80, 83] (**slide**), that our studies, did lead to a revision of the classical doctrines of an enduring continual pro-

gression and a general unfavourable long-term prognosis. Further, that there are no differences regarding the frequency of the origin from the different social classes. Only at the time of the last catamnestic follow-up we found an unequal distribution in favour of the lower social classes; because it cannot be found before the onset of the disorder, it can only be the consequence of the disease in the sense of the »drift hypothesis« [81, pp 317ff.]. According to both studies, nonpsychopathic prepsychotic personalities with good abilities to communicate have a more favourable course than prepsychotic psychopathic personalities [51, 81, 83].

In our follow-up studies we could consider some in earlier investigations not included findings. Hither belong the precursor stages, as also the reversible and irreversible postpsychotic basic stages, which are likewise, as the prodromes and outpost syndromes, determined by basic symptoms; then, the differentiation of minus symptoms in basic and negative symptoms, that is essential for the early recognition of the disorder. The basic symptoms are accessible for insight and self-perception and are identifiable as complaints and disturbances by the patients, who are able to develop coping strategies against the basic symptoms, whilst this is not possible in comparable manner with the true negative symptoms.

The following data about the long-term course and outcome (**slide**) of schizophrenia (ICD-10: F20, F22-25), are sufficiently ensured [21].

(1) The long-term prognosis is more favourable, if the patients are treated somatically as early as possible [15] (**slides**). The longer prodromes persist untreated, the more rarely are full remissions. The patients with early somatic treatment show a more favourable long-term prognosis than the later or not somatically treated patients.

(2) The majority of schizophrenias is beginning with basic symptoms. Out of distinct cognitive level-2-basic symptoms develop distinct positive symptoms, f.e. delusional perceptions out of cognitive perception disorders, bodily hallucinations out of cenesthesias, thought withdrawal out of thought blocking, thought insertion out of thought interference and the so-called passivity

phenomenon (»Willensbeeinflussung«) out of motor interference and motor blockade. Only a minority of the patients (11% in the Bonn-Study) are beginning with true negative symptoms; we spoke of »primary negative anosognosia schizophrenias«.

(3) In the long courses does **not** occur a progressive deterioration. Frequently the disorder comes to a standstill 5 until 10 years after the first psychotic manifestation and/or shows still in the later course far-reaching improvements in the sense of a »second, positive bend« with remission to pure residues.

(4) 22% each of the Bonn- and the Zurich-Study did show complete and enduring psychopathological remission. At least the half of the patients revealed social remissions: In the Bonn-Study have shown 38% complete and further 18% far-reaching social remissions to the premorbid or slightly below the premorbid level; in these 56% you can speak of practical social recovery.

(5) The enormous heterogeneity of the (long-term) outcome has been confirmed [69] (**slide**). In the Bonn-Study there were four groups of course types: The prognostically favourable, relatively favourable, relatively unfavourable and unfavourable group, each accounting for one quarter of all schizophrenias, with extremely different social remission rates from nearly 100% in types I and II until 2% in type XII (**slide**). After a course of on average 22 years 13% were permanently hospitalized.

(6) 27% of the patients of the Bonn-Study were only once hospitalized in the whole course. Probably is a treatment as inpatients today, due to the psychopharmacological therapy, for many patients no longer needed. The incidence of favourable and of subclinical courses seems to be today still higher than earlier.

(7) Yet, already in the 50s of the last century the majority of patients (70%) revealed not a simple progressive development to marked typical schizophrenic defect psychoses (**slide**), but a course in episodes or shifts with in average 4.4 psychotic relapses, either to full remissions (22%) or to predominantly only slight pure (40%) or mixed(16%) residues (Bonn-Study).

This means that the most schizophrenics show the most time in their life-long courses no typical schizophrenic psychotic symptomatology, but more or less uncharacteristic syndromes that are, determined by basic symptoms.

(8) The frequency of schizophrenic catastrophic courses is in the Bonn-Study with 4% clearly lower than the percentages in the 40s, found by BLEULER (5 to 18%).

The results of the European long-term studies have, as Zubin wrote, revolutionized our knowledge in schizophrenia and released this disease from the burden of inevitable chronicity [21, 69] (**slide**). The findings of the Bonn-Study have moreover shown that schizophrenias, if they are progressing, not always lead to a specific schizophrenic psychopathological personality alteration, that is sharply distinguishable from chronic psychosyndromes in definable brain diseases. Thus, also the classical doctrine of a principle and continuous psychopathological heterogeneity and »numinous singularity« of the schizophrenias opposed to organic brain illnesses, could not longer be maintained [81, 83].

Precursor stages and residual states. Bonn-Cologne early recognition study

After the elaboration of the long prodromes in cenesthetic schizophrenias, we described precursor syndromes in the whole group of schizophrenia spectrum disorders, first systematically, differentiated in outpost syndromes and prodromes and based on 290 patients by G. Gross [12] (**slide**). We were showing that minus and aliter, i.e. prodromes, psychotic episodes and postpsychotic pure deficiency syndromes, thus also positive and negative (ANDREASEN), type I and type II (CROW) schizophrenias, must be understood as stages of one an d the same disease process. The interval between the onset of the prodromes, passing over continuously into the psychosis, and the first psychotic episode, can be very differently and amount between a few months and decades, on average 3 to 5 years.

The phenomenological description of the basic symptoms in the pre- and postpsychotic basic stages has been the presupposition for the gradually elaborating of the Bonn Schedule for the Assessment of Basic Symptoms – BSABS [26, 27] (**slide**). The patients perceive already in the precursor stages the new developing basic symptoms as ailments, complaints and disturbances; they learn to develop strategies of coping, defence and avoiding, that aim at the inhibition of releasing and increasing of the basic symptoms and with it the transition into the psychosis.

In our Bonn-Cologne prospective early recognition study (review: see Huber 2005, pp 412ff. – [74]) (**slide**) and in the transition rows study of Klosterkötter has been shown (**slide**), that distinct level-2-basic symptoms, i.e. cognitive thought-, perception- and action basic phenomena, out of which distinct first rank symptoms arise, are predictors for the schizophrenic psychosis.

Subdiagnostic syndromes and formes frustes

We must take into account in schizophrenias as in other mainly genetic conditioned diseases, that subclinical, subdiagnostic, subthreshold syndromes and formes frustes [104] (**slide**) frequently occur, perhaps more frequently than full developed florid forms. Already Eugen BLEULER believed, that the “latent schizophrenia” would be the most frequent, even if most rarely as such recog-

nized type of the illness. To this area of subdiagnostic types and formes frustes belong also the endogenous juvenile-asthenic failure syndromes [11], that are characterized by cenesthesias and other basic symptoms as depersonalization and derealization (BSABS B.3.4, C.2.11) and a subjectively perceived thought disorder. Catamnestic inquiries after in average 6.5 years showed in 56% a transition into schizophrenic psychoses.

To the formes frustes of schizophrenias belong also »circumscribed cenesthopathias« [61] and a subgroup of the endogenous obsessive-compulsive illness [25] (**slide**), that takes its course in shifts or simple-progressive. A proportion of the patients shows in the course schizophrenic episodes. The anancastic symptomatology can often be understood as coping reaction against thought and action basic symptoms.

In the »circumscribed cenestopathias« bodily sensations occur e.g. exclusively in the region of throat, mouth or tongue (»glossodýnia«). Alike as in cenesthetic schizophrenias with their prevailing subdiagnostic stages also in these types of formes frustes frequently occurs a frustrating diagnostic and therapeutic »caréer«. As the most patients with subclinical variants of the schizophrenia spectrum are also the patients with cenesthopathic disorders predominantly not in psychiatric treatment and receive rarely an adequate therapy.

The follow-up of the long courses of the Bonn-Study (1965-1974 – **slide**) revealed a certain sequence of different stages of the disorder: First often year-long diagnostically neutral, quite uncharacteristic, then characteristic prodromal stages with cognitive level-2-basic symptoms and transition rows to the positive symptoms of the psychotic episodes, that frequently remit again to nonpsychotic stages.

Basic Symptom- and Process Activity Concept. Continuum Hypothesis. Coping strategies

This »diachronia«, the development of the psychosis on a temporal longitudinal axis, we had described with PENIN as “course dynamic aspect” [79]. Proceeding from it and the basic symptom concept we developed the clinical-

psychopathological defined concept of process activity [66, 67, 79, 88, 101] (**slide**). Functional-dynamic, e.g. neurochemical deviations as to neurotransmitter or abnormal rhythmization in EEG (parenrhythmias) occur, as we demonstrated, only transitory-intermittent, associated with process active symptomatology [76, 82, 101, 102] (**2 slides**). These deviations are state dependent changes, only present in psychopathologically as process active definable stages [28, 30] (**2 slides**).

The understanding of the subjective experiences of the patients, reported as basic phenomena, provides a communicative bridge [13, 51, 54, 77] (**slide**). It becomes e.g. recognizable that first incomprehensible, f.i. secondary autistic behavioural attitudes, are understandable coping strategies with the function to prevent the inducing or intensifying of basic symptoms up to psychotic relapses. The application of psychotherapeutic strategies, referring to the disturbances, requires knowledge of the relationships between specific vulnerability, strain and the manifestation of basic symptoms [104] (**slide**). The reduced capability to be burded by certain stressors can be understood and the patient encouraged e.g. to use intentionally avoidance and shielding reactions. The information of related persons about the basic symptoms in the frame of the psychoeducation supports to understand the behaviour of the patients and to correct errors and misapprehensions [105].

»Prodromal and residual symptoms« (PRS) in the DSM and the basic symptom concept (BSC)

In the modern diagnostic systems the prodromal and residual symptoms are not or insufficiently, i.e. only as negative symptoms defined. This seems to be an essential reason for the delay of early recognition and treatment [66] (**slide**).

The differentiation of the “Prodromal and residual symptoms” of schizophrenias from the unspecific symptoms of patients with neurotic and personality disorders became meanwhile possible by means of the transition relevant level-2-basic symptoms [14, 18, 19, 28, 29, 31, 32, 66, 68, 87, 89, 90, 91] (**2 slides**). Distinct cognitive basic symptoms are early warning signals for the later occurrence of a schizophrenic psychosis (**3 slides**). Lilo SÜLLWOLD and we attained

to a concept of the significance of these basal disorders for the theory of schizophrenia, the understanding of the inner world of the patients and the early recognition before the first and later psychotic episodes [104] (**slide**). That the most researchers do not yet distinguish between basic and negative symptoms, a precondition for a successful early intervention, is surely also the prize for the operationalism. What can be won as to reliability, leads by the methodological reductionism to a loss of psychopathological differentiation and with it of validity. The both symptom dimensions, basic symptomatology and positive symptoms, are in a causal functional connection, as with increasing of process activity and rise of cognitive level-2-basic symptoms exceeding a certain threshold, ensues a psychotic syndrome with positive first and second rank symptoms.

Level-2-prodromal basic symptoms turned out to be able to put up on them gradually concepts of early recognition and treatment [89] (**slide**). But, it is necessary, to intervene also with psychopharmaca, i.e. atypical neuroleptics, earlier as it has been proposed: Namely already when the cited predictors of psychosis in form of distinct cognitive level-2-basic symptoms in the prodromes occur, and not only, if »brief limited intermittent psychotic symptoms« (BLIPS) have developed [s. 74, pp 412ff.] (**slide**).

Psychiatry and Society. Discrimination and Stigmatization

Our issue cannot be dealt with without inclusion of the history of our discipline [48, 56, 60, 62, 64, 72, 73, 78] (**slide**), the questions »Psychiatry and Society« and discrimination, stigmatization and tabooing of psychiatric and particularly schizophrenic illnesses. In this respect, can we be content with that what has been achieved in the last 50 years?

Surely not without reservations. It is true, there are meaningful advances, but, it cannot be overlooked the enormous discrepancy between the scientific progress and the reality of care of many psychiatric, especially psychotic patients [22, 23]. We had in the last decades also referred to the imminent or already present loss of psychiatric and psychopathological competence [16, 20, 57, 70, 71, 72, 73, 74, 103] (**slide**), and had admonished, to correct this neglect.

Perhaps such a monitum may be more effective, if it, as recently (1998), comes from the United States, where Nancy ANDREASEN requested an intensive training of a new generation of psychiatrists in psychopathology, because, without this, “applying technology by high tech scientists without the companionship of clinicians with specific expertise in psychopathology may be a sterile and fruitless enterprise. We need the psychopathology according to the axiom: »First things first« [16, 20, 72, 73].

The patients should be in our university institutions not only the »suppliers« of material for the research; production of often superfluous papers, »publish or perish« and »science impact factor« should not determine the scientific scene. The phenomenological psychopathology is also meaningful for communication with the patients, for atmosphere and spirit of psychiatric hospitals, and the overcoming of prejudices against psychiatric patients.

As to the removal of prejudices the balance, unfortunately, is still negative. According to a Swiss study exist against psychiatry unchangeable prejudices as with regard to no other discipline in medicine [23]. American scientific journals as »Molecular Psychiatry« and »Nature« [10, 23, 94] (**slide**) see psychiatry as »Medicine’s least respectable branch« and allege the reasons for this omega-position. Also according to John WING are above all the antipsychiatric prejudices responsible for this situation; the obstacles conditioned by this attitude, still increased by the public opinion, could scarcely be overcome.

Susceptibility to ideologies and political abuse

If psychiatric, particularly schizophrenic disease, does mean the same as before a capitis deminutio by the society, the way from here is not too far to the idea of the »licence for the extermination of not life-worth life«, as advocated by two German professors of psychiatry (HOCHE) and jurisprudence (BINDING) in 1920, i.e. of the so-called euthanasia and other of HITLER realized wiping out measures. Principiis obsta; commencements, which are not prevented can anew clearly be recognized. Many psychiatrists gave voice to the susceptibility of our discipline to ideologies, thus Weitbrecht in his university speeches of the

60s [53, 106] (**slide**). He has shown how also at that time the psychiatry was risked to be misused for political purposes and to be usurped by a new intolerant dogmatism [56, 58].

In the course of the change to the biological psychiatry, induced by the break down of the Community Psychiatry in the United States [1, 56, 75] (**slide**), were unfortunately replaced old by new errors, thus the psychoanalytic and sociological myth by a one-sided biological psychiatry and the loss of psychopathological competence, connected with it [56, 74].

For the susceptibility of psychiatry to ideologies there are many examples, e.g. the events on the occasion of the renewed filling of the Heidelberg chair in 1972. Six selected professors presented themselves. Huge streamers (»Erich Wulff on the psychiatric chair«; »Huber, marsh flower of the bourgeois psychiatry«), pamphlets: For a materialistic-marxistic founded psychiatry; schizophrenia originating from the capitalistic society; only if this can be eliminated, or can put to vanishing the so-called »Geisteskrankheiten«. Discussion, dominated by the chief ideologists of the 68-scene with the tenor: Schizophrenia and the whole psychiatry must be completely separated from medicine and has to be understood as pathology of social communication. A for many plausible and fascinating universal thesis, that, seemed to be suitable to explain all psychosocial evils from one point.

These ideas remained not theory, but were translated consequently into action, thus in the Heidelberg socialistic collective of patients, according to whose program »to made a weapon out of the disease«, the purpose is the fighting of the patients against the society system, responsible for their disorder.

Consequences of those ideologies are still until today recognizable, in Germany e.g. in leading articles of news magazines as e.g. »Psychopharmaca in the psychiatry – the soft death« or in postulates of the Greens to the psychiatry Politics, circulating as parliament printed paper »Abolition and overcoming of the psychiatry – against the nuclear power station in the interior« [56, 72] (**slide**).

As to the susceptibility of psychiatry to ideologies was always the schizophrenia in the centre of interest. We had reminded of the consequences of the dictatórial biologism in the »Third Reich«. But, also after it the psychiatry has been misused for political purposes, as I have shown in a review to the 65th birthday of WEITBRECHT [53] (**slide**), citing a paper of v. BAEYER »To the psychiatric internment of opposing people in the Sowjet Union« (1973). After the publication of my lecture in the »Fortschritte Neurologie Psychiatrie« (1975) the publisher of the »Fort-schritte« received a letter from Leipzig »Book export foreign trade management of GDR«: My publication would show unequivocally antisowjetic character and »against such agitation we protest rigorously. In the case of recurrence we must expunch your journals from our postlist of newspapers« [72].

After I before, after the death of Weitbrecht (2nd Janu-ary 1975) as his follower has been co-opted in the Editorial Board of the »Fort-schritte«, came immediately after the warning from Leipzig the information, unfortunately the publisher had now to refrain from my provided entering into the redaction. At that time really many Germans remained not unimpressed by such trials of intimidation.

With the internment of the dissenters as mental cases, were Professor HEINRICH and I confronted as German delegates and speakers at the International Psychiatry Congress of the WPA (World Psychiatric Association) in Eriwan and Tiflis (8.-12.10.1973). We had together with John WING and RAWNSLEY refused to go in the Moscow Serbskij Institute for Forensic Psychiatry in order to state, based only on medical files, that it would be a question of schizophrenics and not of opposing persons. But, we remained with this refusal a minority, while all other foreign delegates followed the invitation. The consequences were exactly thus, as our Russian Jewish colleague Erich STERNBERG had prognosticated. STERNBERG emigrated already in 1933 from Germany to Moscow, there in 1936 has been banished to Siberia and only after the death of STALIN (1953) could return to Moscow [58] (**slide**). The international Press reported unisono, that western psychiatrists had confirmed after a discussion in the Serbskij Institute, that the interned persons were not dissenters but schizophrenics [72].

Before, SACHAROW in a long interview in the German magazíne »Stern« designated the names of psychiatrists, who diagnosed dissenters as schizophrenics. These political arbitrators would meet in Eriwan western scientists. »From Germany come the professors Gerd HUBER and Kurt HEINRICH – how will be their attitude to such sowjetic colleagues?«.

The history of Neurology and Psychiatry in the 20th century with the separation of psychiatry from neurology has shown how the esteem of psychiatry has unfavourably changed [78] (**slide**). Not at last because its proneness to the fashions of the »Zeitgeist«, and its tendency to replace the gaps in biological and etiological research just in schizophrenias by partly fascinating speculations and to abandon well substantiated findings, concepts and classifications without having better alternatives. This became evident recently also in the mentioned comments of United States neuropsychiatrists. In an editorial of LICINIO [94], editor of »Molecular Psychiatry«, has been demonstrated, why psychiatry did not succeed to throw off its bad image.

At the end I ask: What should we have learned from the history of schizophrenia research? Impatience on too slow progress of sure knowledge should not seduce get lost in vague speculations and utópias. An attitude, that regards the good agon as necessary and fruitful and e.g. grapples actively also with wrong vievs as those of the principle heterogeneity and numinous singularity of schizophrenia against all other diseases, or with the dogmas of biologism, socialism and Freudism, has been demanded already by KRAEPELIN, JASPERS, Kurt SCHNEIDER, WEITBRECHT, KOLLE and Michael SHEPHERD. I cite: Further carefully observe and investigate, with modest claims to put our questions, to follow-up attentively the acrobatic rhetorical arts of some theorists, but him- or herself take firm standing on the earth, not too much philosophize, as HEIDEGGER himself did admonish us in 1950; and, for all that, not forget the wondering and: Not loose the novelty seeking and taking pleasure and satisfaction in the working with our patients.

References:

1. Arnhoff FN (1975) Social consequences of policy towards mental illness. Science 188: 1277-1281.
2. Bleuler M (1951) Forschungen und Begriffswandlungen in der Schizophrenielehre. Fortschr Neurol Psychiat 19:385-452.
3. Bleuler M (1972) Die schizophrenen Geistesstörungen im Lichte langjähriger Kranken- und Familiengeschichten. Thieme, Stuttgart
4. Bleuler M (1976) Prävention der Schizophrenien – winzige Körnchen Wissen in einem Meer von Nichtwissen. In‘: Huber G (ed) Therapie, Rehabilitation und Prävention schizophrener Erkrankungen. 3. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York, 161-174.
5. Bleuler M, Huber G, Gross G, Schüttler R (1976) Der langfristige Verlauf schizophrener Psychosen. Gemeinsame Ergebnisse zweier Untersuchungen. Nervenarzt 47:477-481.
6. Bogerts B (1985) Schizophrenien als Erkrankungen des limbischen Systems. In: Huber G (ed) Basisstadien endogener Psychosen und das Borderline-Problem. 6. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York, 163-172.
7. Conrad K (1971) Die beginnende Schizophrenia. 3. Aufl. Thieme, Stuttgart
8. Danos P, Baumann B, Krämer A et al (2003) Volumes of association thalamic nuclei in schizophrenia: A post mortem study. Schizophr Res 60:141-155.
9. Dewan MJ (1987) Cerebral structure and symptomatology. In: Harvey PD, Walker EF (eds) Positive and negative symptoms in psychoses. Erlbaum, Hillsdale, 216-247.
10. Evans D (2000) Medicine’s least respectable branch. Nature 403:19-20.
11. Glatzel J, Huber G (1968) Zur Phänomenologie eines Typs endogener juvenil-asthenischer Versagenssyndrome. Psychiatr Clin 1:15-31.
12. Gross G (1969) Prodrome und Vorpostensyndrome schizophrener Erkrankungen. In: Huber G (ed) Schizophrenie und Zyklothymie. Ergebnisse und Probleme. Thieme, Stuttgart, 177-188.
13. Gross G (1987) Basic symptoms and coping behaviour in schizophrenia. In: Strauss JS et al (eds) Psychosocial treatment of schizophrenia. Huber, Toronto Bern Stuttgart, 126-135.
14. Gross G (1997) The onset of schizophrenia. Schizophr Res 28:187-198.
15. Gross G, Huber G (1993) Möglichkeiten und Grenzen einer Prädiktion des langfristigen therapieunabhängigen und therapieabhängigen Verlaufs schizophrenr und schizoaffektiver Erkrankungen. In: Heinrich K et al (eds) Prädiktoren des Therapieverlaufs endogener Psychosen. Schattauer, Stuttgart New York, 17-31.
16. Gross G, Huber G (1993) Do we still need psychopathology –and if so, which psychopathology? Neurol Psychiatr Brain Res 1: 194-200.
17. Gross G, Huber G (1995) Psychopathology and biological-psychiatric research. Neurol Psychiatry Brain Res 3:161-166.
18. Gross G, Huber G (1996) The true onset of schizophrenia in its meaning for the view of the disorder. Neurol Psychiatry Brain Res 4:93-102.
19. Gross G, Huber G (1996) Prodromes and primary prevention of schizophrenic psychoses. Neurol Psychiatry Brain Res 4:93-102.
20. Gross , Huber G (2000) The relevance of Jaspersian-Schneiderian psychopathology for today’s psychiatry. Neurol Psychiatry Brain Res 8:53-68.
21. Gross G, Huber G (2000) The long-term outcome of schizophrenic and related disorders. A critical review. Neurol Psychiatry Brain Res 8:121-130.
22. Gross G, Huber G (2004) The discrepancy between scientific advances in psychiatry and the reality of treatment of psychoses. Neurol Psychiatry Brain Res 11:165-170.

23. Gross G, Huber G (2005) Psychiatrie – die am wenigsten respektierte Disziplin in der Medizin. In: Schneider F, Saß H (eds) Entwicklungen in der Psychiatrie. Springer, Berlin Heidelberg New York, 323-329.
24. Gross G, Huber G, Schüttler R (1982) Computerized tomography studies on schizophrenic diseases. Arch Psychiat Nervenkr 231:519-526.
25. Gross G, Huber G, Klosterkötter J (1988) Schizophrenie und endogene Zwangskrankheit. Nervenheilkd 7:113-118.
26. Gross G, Huber G, Klosterkötter J, Linz M (1987) BSABS. Bonner Skala für die Beurteilung von Basissymptomen. Bonn Scale for the Assessment of Basic Symptoms. Springer, Berlin Heidelberg New York. (1992) Italian, (1994) Danish, (1995) Spanish, (1996) Japanese edition.
27. Gross G, Huber G, Klosterkötter J (2005) BSABS. Bonn Scale for the Assessment olf Basic Symptoms. 2nd edition. Shaker, Aachen (in press).
28. Gross G, Huber G, Klosterkötter J (1990) Psychopathological-neurochemical correlations in idiopathic psychoses. In: Stefanis C et al (eds) Psychiatry: A world perspective, Vol. 2. Excerpta Medica, Amsterdam New York Oxford, 203-208.
29. Gross G, Huber G, Klosterkötter J, Gnad M (1990) Früherkennung idiopathischer Psychosyndrome. In: Huber G (ed) Idiopathische Psychosen. Psychopathologie – Neurobiologie – Therapie. 8. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York, 157-180.
30. Gross G, Huber G, Klosterkötter J et al (1990) Zustandsabhängige neurochemische Parameter bei schizophrenen und affektiven Erkrankungen. Fortschr Neurol Psychiat 58:154-160.
31. Gross G, Huber G, Klosterkötter J (1991) Früherkennung der Schizophrenien. Fundamenta Psychiatrica 5:172-178.
32. Gross G, Huber G, Klosterkötter J (1992) Early diagnosis of schizophrenia. Neurol Psychiatry Brain Res 1:17-22.
33. Gruhle HW (1932) Die Schizophrenie. Die Psychopathologie. In: Bumke O (ed) Handbuch der Geisteskrankheiten. Bd.IX, Spez. Teil V. Springer, Berlin, 135-210.
34. Gruhle HW (1948) Verstehende Psychologie. Thieme, Stuttgart.
35. Huber G (1953) Zur Frage der mit Hirnatrophie einhergehenden Schizophrenie. Arch Psychiat Z Neurol 190:429-448.
36. Huber G (1955) Das Pneumencephalogramm am Beginn schizophrener Erkrankungen. Arch Psychiat Z Neurol 193:406-426.
37. Huber G (1955) Zur nosologischen Differenzierung lebensbedrohlicher katatoner Psychosen. Schweiz Arch Neurol Psychiat 74:216-244.
38. Huber G (1957) Die coenästhetische Schizophrenie. Fortschr Neurol Psychiat 25:491-520.
39. Huber G (1957) Pneumencephalographische und psychopathologische Bilder bei endogenen Psychosen. Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie, H. 79. Springer, Berlin Göttingen Heidelberg.
40. Huber G (1961) Zur klinischen und anatomischen Diagnose der primären, atypischen, sporadischen Virusencephalitiden. Zbl Ges Neurol Psychiat 161:174.
41. Huber G (1961) Zur klinischen Diagnose sporadischer Spontanencephalitiden. Nervenarzt 32:491-497.
42. Huber G (1961) Chronische Schizophrenie. Synopsis klinischer und neuroradiologischer Untersuchungen an defektschizophrenen Anstaltspatienten. Hüthig, Heidelberg
43. Huber G (1961) Klinische und neuroradiologische Untersuchungen an chronisch Schizophrenen. Nervenarzt 32:7-15.
44. Huber G (1964) Neuroradiologie und Psychiatrie. In: Gruhle HW et al (eds) Psychiatrie der Gegenwart. Forschung und Praxis, Bd.I/1 B. Springer, Berlin Göttingen Heidelberg, 253-290.

45. Huber G (1966) Reine Defektsyndrome und Basisstadien endogener Psychosen. Fortschr Neurol Psychiat 34:409-426.
46. Huber G (1971) Die coenästhetische Schizophrenie als ein Prägnanztyp schizophrener Erkrankungen. Acta Psychiat Scand 47:349-362.
47. Huber G (1971) (ed) Ätiologie der Schizophrenien. Bestandsaufnahme und Zukunftsperspektiven. 1. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York.
48. Huber G (1972) Zukunftsperspektiven einer Struktur der psychiatrischen Versorgung in Deutschland. Fortschr Neurol Psychiat 40:515-533.
49. Huber G (1972) Klinik und Psychopathologie der organischen Psychosen. In: Kisker KP et al (eds) Psychiatrie der Gegenwart. Bd.II/2. 2.Aufl. Springer, Berlin Heidelberg New York, 71-146.
50. Huber G (1973) Psychopathologie der Epilepsien. In: Penin H (ed) Psychische Störungen bei Epilepsien. Schattauer, Stuttgart New York, 7-22.
51. Huber G (1973) (ed) Verlauf und Ausgang schizophrener Erkrankungen. 2. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York
52. Huber G (1973) Zum Stand der Verlaufsforschung bei den Schizophrenien. In Huber G (ed) Verlauf und Ausgang schizophrener Erkrankungen. 2. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York, 259-267.
53. Huber G (1975) HJ Weitbrecht und die Psychiatrie. Fortschr Neurol Psychiat 43:54-70.
54. Huber G (1976) (ed) Therapie, Rehabilitation und Prävention schizophrener Erkrankungen, 3. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York.
55. Huber G (1976) Indizien für die Somatosehypothese bei den Schizophrenien. Fortschr Neurol Psychiat 44:77-94.
56. Huber G (1979) Neuere Ansätze zur Überwindung des Mythos von den sog. Geisteskrankheiten. Fortschr Neurol Psychiat 47:449-465.
57. Huber G (1980) Hauptströme der gegenwärtigen ätiologischen Diskussion der Schizophrenie. In: Peters UH (ed) Die Psychologie des 20. Jahrhunderts, Bd.X. Kindler, Zürich, 397-420.
58. Huber G (1981) Erich Sternberg 1903-1980. Arch Psychiat Nervenkr 200:1-3.
59. Huber G (1983) Das Konzept substratnaher Basissymptome und seine Bedeutung für Theorie und Therapie schizophrener Erkrankungen. Nervenarzt 54:23-32.
60. Huber G (1984) Die Bedeutung von Karl Jaspers für die Psychiatrie der Gegenwart. Nervenarzt 55:1-9.
61. Huber G (1987) Hypochondrie und Coenästhopathie. Fundamenta Psychiatrica 1:122-127.
62. Huber G (1987) Kurt Schneider. The man and his scientific work. Zbl Neurol Psychiat 246: 177-191.
63. Huber G (1989) Advances in schizophrenia research. In: Deecke L (ed) From neuron to action. Springer, Berlin Heidelberg New York, 471-477.
64. Huber G (1991) Psychiatrie als öffentliches Anliegen. Zbl Neurol Psychiat 259:1-15.
65. Huber G (1992) »Cenesthetic schizophrenia« – a subtype of schizophrenic disease. Neurol Psychiat Brain Res. 1:54-60.
66. Huber G (1995) Prodrome der Schizophrenie. Fortschr Neurol Psychiat 63:131-138.
67. Huber G (1995) Basissymptom- und Prozeßaktivitätskonzept endogener Psychosen. In: Bräunig P (ed) Emotionspsychopathologie und zykloide Psychosen. Schattauer, Stuttgart New York, 51-65.
68. Huber G (1996) Was wissen wir über initiale Stadien schizophrener Erkrankungen? In: Gross G, Huber G, Morgner J (eds) Persönlichkeit – Persönlichkeitsstörung – Psychose. 10. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York, 171-189.
69. Huber G (1997) The heterogeneous course of schizophrenia. Schizophr Res 28:174-185.

70. Huber G (2001) (ed) Therapie der Schizophrenien. Grundlagen und Praxis. 13. Weißenauer Schizophrenie-Symposion. Fortschr Neurol Psychiat 69 (Suppl 2):S53-S144.
71. Huber G (2001) Ist die Psychopathologie für die Therapie der Schizophrenie noch von Bedeutung? In: Huber G (ed) Therapie der Schizophrenien. Grundlagen und Praxis. 13. Weißenauer Schizophrenie-Symposion. Fortschr Neurol Psychiat 69 (Suppl 2): S86-S91.
72. Huber G (2002) Psychopathologie – eine versiegende Quelle? Fortschr Neurol Psychiat 70:393-402.
73. Huber G (2002) The Psychopathology of K. Jaspers and K. Schneider as a fundamental method for psychiatry. World J Biol Psychiatry 3:50-57.
74. Huber G (2005) Psychiatrie. Lehrbuch für Studium und Weiterbildung. 7th edn. Schattauer, Stuttgart New York.
75. Huber G, Gross G (1974) Schizophrenie und Pseudoschizophrenie. In: Alsen V (ed) Organische endoforme Psychosen, organische Pseudopsychopathien und Pseudoneurosen. Das ärztliche Gespräch. Troponwerke, Köln, 80-105.
76. Huber G, Gross G (1992) Somatische Befunde bei Psychosen. In: Battegay R et al (eds) Handwörterbuch der Psychiatrie. 2. Aufl. Enke, Stuttgart, 566-574.
77. Huber G, Gross G (1998) Basic symptom concept – historical aspects in view of early detection of schizophrenia. Neurol Psychiatry Brain Res 5:183-190.
78. Huber G, Gross G (2003) The bridges between Neurology and Psychiatry. Neurol Croat 52(Suppl 2):65-77.
79. Huber G, Penin H (1968) Klinisch-elektroencephalographische Korrelationsuntersuchungen bei Schizophrenen. Fortschr Neurol Psychiat 36:641-659.
80. Huber G, Gross G, Schüttler R (1976) Konsequenzen der Verlaufsuntersuchungen für die Therapie und Rehabilitation der Schizophrenien. In: Huber G (ed) Therapie, Rehabilitation und Prävention schizophrener Erkrankungen. 3. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York, 111-131.
81. Huber G, Gross G, Schüttler R (1979) Schizophrenie. Verlaufs- und sozialpsychiatrische Langzeituntersuchungen an schizophrenen Kranken. Monographien aus dem Gesamtgebiete der Psychiatrie, Bd.21. Springer, Berlin Heidelberg New York. Reprint 1984.
82. Huber G, Gross G, Klosterkötter J (1990) Amino acids in process active and inactive stages in schizophrenic psychoses. In: Stefanis C et al (eds) Psychiatry: A world perspective. Excerpta Medica, Amsterdam New York Oxford, 209-214.
83. Huber G, Gross G, Schüttler R, Linz M (1980) Longitudinal studies of schizophrenic patients. Schizophr Bull 6:592-605.
84. Jaspers K (1946) Allgemeine Psychopathologie. 4. Aufl. Springer, Berlin Göttingen Heidelberg.
85. Klosterkötter J (1988) Basissymptome und Endphänomene der Schizophrenie. Springer, Berlin Heidelberg New York.
86. Klosterkötter J (1999) Das Forschungswerk Gerd Hubers. Aktualität und Entwicklungsperspektiven. In: Gross G et al (eds) 50 Jahre Psychiatrie. Schattauer, Stuttgart New York, 199-226.
87. Klosterkötter J, Gross G (1988) Wahrnehmungsfundierte Wahnwahrnehmungen. In: Böcker F, Weig W (eds) Aktuelle Kernfragen in der Psychiatrie. Springer, Berlin Heidelberg New York, 111-121.
88. Klosterkötter J, Gross G, Huber G (1989) Das Konzept der Prozeßaktivität bei idiopathischen Psychosen. Nervenarzt 60:740-744.
89. Klosterkötter J, Gross G, Huber G et al (1997) Evaluation of the Bonn Scale for the Assessment of Basic Symptoms – BSABS as an instrument for the assessment of schizophrenic proneness: Recent findings. Neurol Psychiatry Brain Res 5:137-150.
90. Klosterkötter J, Gross G, Huber G et al (1997) Selbst wahrnehmbare neuropsychologische Defizite bei Patienten mit Neurose- oder Persönlichkeitsdiagnosen, für spätere schizophrene Erkrankungen prädiktiv? Nervenarzt 68:196-204.

91. Klosterkötter J, Gross G, Huber G et al (1997) Early self-experienced neuropsychological deficits and subsequent schizophrenic diseases: 8-year follow-up prospective study. Acta Psychiat Scand 95:396-400.
92. Koehler K, Sauer H (1984) Huber's basic symptoms: Another approach to negative psychopathology in schizophrenia. Compr Psychiatry 25:174-182.
93. Koehler K, Sauer H (1984) Jaspers' sense of presence in the light of Huber's basic symptoms. Compr Psychiatry 25:183-191.
94. Licinio J (2004) A leadership crisis in American psychiatry. Mol Psychiatry 9:1.
95. Maggini C, Dalle Luche R (1992).Edizione italiana: Gross G, Huber G, Klosterkötter J, Linz M. BSABS. Scala di Bonn per la valutazione dei Sintomi di base. ETS Editrice, Pisa.
96. Maggini C, Raballo A, Salvatore P (2002) Depersonalization and basic symptoms in schizophrenia. Psychopathology 35:17-24.
97. Maggini C, Raballo A (2003) Experiences with the basic symptom concept in Italy. Neurol Psychiatry Brain Res 10:151-158
98. Maggini C, Raballo A, Salvatore P (2004) Depressive and basic symptoms in schizophrenia. Neurol Psychiatry Brain Res 11:1-6.
99. Mayer-Gross W (1931) Die Schizophrenie. Die Klinik. In: Bumke O (ed) Handbuch der Geisteskrankheiten. Bd.IX, Spez. Teil V. Springer, Berlin, 293-578.
100. Nakayasu N (2991) Early schizophrenia: A new clinical entity. JMAJ 44:182-188.
101. Penin H, Gross G, Huber G (1982) Elektroenzephalographisch-psychopathologissche Korrelationsuntersuchungen in Basisstadien endogener Psychosen. In: Huber G (ed) Endogenen Psychosen: Diagnostik, Basissymptome und biologische Parameter. 5. Weißenauer Schizophrenie-Symposion. Schattauer, Stuttgart New York, 247-264.
102. Rao ML, Gross G, Huber G (1984) Altered interrelationship of dopamine, prolactin, thyrotropin and thyroid hormone in schizophrenic patients. Eur Arch Psychiatry Clin Neurosci 234:8-12.
103. Schneider K (1992) Klinische Psychopathologie. With a commentary by Huber G and Gross G. 14th edn. Thieme, Stuttgart New York. 15th edition in press.
104. Süllwold L, Huber G (1986) Schizophrene Basisstörungen. Monographien aus dem Gesamtgebiete der Psychiatrie, Bd.42. Springer, Berlin Heidelberg New York.
105. Süllwold L, Herrlich J (2001) Psychologische Therapie schizophren Erkrankter – Trend der Konzeptentwicklung. In: Huber G (ed) Therapie der Schizophrenien: Grundlagen und Praxis. Fortschr Neurol Psychiat 69 (Suppl 2):S127-S131.
106. Weitbrecht HJ (1968) Psychiatrie in der Zeit des Nationalsozialismus. Politeia – Bonner Universitätsreden zu öffentlichen Fragen. H.11. Universitätsverlag, Bonn

Address of the author:
Professor Dr.med. Dr.h.c. Gerd Huber
Auf dem Rosenberg 18
D-53343 Wachtberg-Villiprott/Germany
Tel/Fax: +49 228 322883

Office:
Universitäts-Nervenklinik
(Department of Psychiatry and Psychotherapy of the University of Bonn)
D-53105 Bonn (Venusberg), Germany

Tel: +49 228 287 5725
Fax: +49 228 287 1736