INTERPRETATION OF SCHIZOPHRENIA

The Central Nervous System in Schizophrenia

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The Central Nervous System in Schizophrenia

It is natural that the central nervous system should have been the object of much research in schizophrenia. Emil Kraepelin, who was the first to individualize the disorder at a clinical level, always hoped that science would one day duplicate for dementia praecox the triumphant actualization of the medical model, as it took place in reference to general paresis. Such hope has not been realized.

I
Neuropathology

The founding fathers of neuropathology, Nissl, Alzheimer, and Spielmeyer, initiated a great deal of histologic research. From their time to the present the works of neuropathologists have remained as indefinite as those of other people doing other somatic research. Many histologic alterations were found in the brains of schizophrenics, but not consistently, and their significance was uncertain. Alzheimer (1897) found a local disappearance of ganglion cells in the outer layer of the cerebral cortex. Josephy (1930) described cell sclerosis and fatty degeneration in the cortical layers. Winkelman (1952) found a diffuse

loss of nerve cells. Buscaino (1921) reported grapelike formations in the brains of schizophrenics. Other authors could not confirm these findings, which were considered artifacts.

Klippel and Lhermitte (1906) described areas of focal demyelination (later denied by Adolf Meyer and by Wolf and Co wen, 1952). Bruetsch (1940) found rheumatic foci in brains of schizophrenics. He felt that the incidence of these foci was greater than in the general population and that rheumatic fever must have played an important etiologic role. (In 1952, Lidz denied the importance of this role.)

In an effort to overcome the technical defects that many authors found in neuropathological studies of schizophrenia, C. and O. Vogt adopted a refined technique that consisted of a serial study of the cerebral hemispheres in slices 8 *p.* (micron) thick. Quantitative computations of the alterations were made and control studies in normal people of the same age were carried out. The Vogts (1954) concluded that they were able to find anatomical alterations in all cases of schizophrenia that they studied. The localization, intensity, and histologic aspect of the lesions were found to vary considerably.

The most important features were:

1. So-called wasting cells (*Schwundzelle* in German; *cellule fondante* in French). In these cells there is a progressive disintegration and disappearance of the tigroid substance, increase in fat content, and decrease in volume. The nucleus becomes pale at first and then disappears (karyolysis).

2. Alveolar degeneration. The cells lose the Nissl substance and undergo vacuolation of the cytoplasm.

3. Liposclerosis. The cells are shrunken and presumably filled with fat.

Other alterations (such as cell shrinkage, balloon cells, and senile alterations) seemed to play a less important role. This impressive work by the Vogts was not completed. All the alterations described by them are found to some degree in many pathological conditions and even in the so-called normals. The only finding that would be significant would be a quantitative difference between schizophrenics and normal persons. But again even then there would remain the problem of whether the quantitative change is part of the basic schizophrenic process or the outcome of the abnormal schizophrenic way of living, especially in patients who have been sick for a long time.

Several researchers have studied biopsy material in an attempt to avoid the artifacts liable to be found in postmortem specimens. Papez (1948) and his associates reported inclusion bodies, presumably microorganisms, for example, zoospores. The significance of these findings was disputed by Wolf and Cowen (1952) and by Alfred Meyer (1954).

Roizin (1952) made morphological and biochemical studies of biopsy specimens from twenty-four patients who had undergone topectomy and electric shock (or insulin coma, or both) prior to psychosurgery. He found several alterations in the medium-sized and large-sized pyramidal neurons. He also found changes in the activity of indophenol-oxidase, cytochrome (oxidase) peroxidases, and acid phosphatases. Roizin's work is an excellent piece of research, but, as the author himself admits, it is inconclusive because the findings might have been the results of the therapies to which the patients were subjected.

Dunlap (1928), Spielmeyer (1931), Wolf and Cowen (1952), Weinstein (1954), Ferraro (1954), and Dastur (1959), among many distinguished researchers, reached negative conclusions about these

neuropathological findings in schizophrenia, minimizing their importance on various grounds: these changes are not specific, are of mild intensity, and may be found also in control brains of normal persons; they may be the result of age, malnutrition, concomitant disease, or abnormal way of living of the patient.

Other authors feel that the relatively primitive techniques of traditional neuropathology have led to the negative findings and look forward with great anticipation to the results of new techniques, such as electronic microscopy, tissue culture, and microchemistry. An alteration affecting the brain at a molecular level is not detectable with histological procedures.

II
Electroencephalographic Findings

When Hans Berger discovered the electroencephalograph (EEG) the hope arose that EEG studies would reveal what neuropathology had failed to disclose. Such hope too was doomed to failure. The results were inconsistent and open to severe criticism. Early authors, like Berger himself (1931, 1933) and Lemere (1936), reported that the EEG was normal in schizophrenia. Davis (1940, 1942) described what

she termed a “choppy activity,” which is a disorganized pattern of low voltage ranging from approximately 26 to over 50 cycles per second (cps). This pattern could not be related to age differences. Davis did not consider this EEG record specific for schizophrenia, but she asserted that it was much more common in this disorder than in others. In fact, she found it in 61 percent of the records of schizophrenics in contrast to 39 percent of manic-depressives. Hill (1957) found the “choppy pattern” in only 20 percent of schizophrenics.

Walter (1942) observed slow wave changes in catatonic stupor. He found that the alpha activity shows instability and slow activity at 2 to 6 cps. Low voltage was also apparent. These findings were not constant and could not be interpreted.

Hill, who made an excellent review of the literature on this subject up to 1957, concluded that the EEG is abnormal in a proportion of patients suffering from schizophrenia, particularly of the catatonic type, but that no specific pattern is found in these patients. Subcortical type discharges, similar to those found in some epileptics, were found, particularly in young catatonics, but were not interpreted

by the author as evidence of an organic-pathological process. Hill stated that the available EEG data "do not provide adequate evidence for a genetic progressive pathological cerebral process in schizophrenia."

Landolt (1957) found that temporal foci and spastic potential often exist in noncatatonic schizophrenia before manifestation of the disorder; they subside when the illness manifests itself overtly, and they recur when a remission takes place.

More recently several authors have studied the EEG in the course of the schizophrenic illness. Igert and Lairy (1962) found consistent normal records in sixty-two female patients throughout the illness and in spite of the therapeutic procedures with which the patients were treated. Small and Small (1965) found that the most severe and prolonged forms of schizophrenia were among the patients who had a normal EEG. Various authors have found that the most normal EEG in cases of schizophrenia is paradoxically associated with poor prognosis.

Perhaps the most conclusive work remains the one by Colony and

Willis (1956), who studied EEG in a thousand cases of schizophrenia and found the frequency of abnormal rhythm to be 5 percent, that is, even less than in their control normal cases.

Strauss (1959) corroborates the findings of Colony and Willis. He believes that schizophrenics show no significantly greater degree of abnormality than normal controls, and certainly no temporal spikes.

EEG studies of schizophrenics have also been made during sleep. Gibbs and Gibbs (1963) have described a so-called mitten pattern, which seems to be connected with psychotic conditions during the state of sleep in patients not younger than age 15. It is found in the frontal lobes, but it spreads also to the parietals. Two varieties have been described: the A mitten, in which the fast component lasts longer than 100 msec, and the B mitten, in which it lasts between 80 and 100 msec. The B mitten pattern was found to occur in 37 percent of schizophrenia patients.

Phases of sleep associated with rapid eye movements (REM) have been studied in schizophrenics (Feinberg et al., 1964, 1965; Rechtschaffen, Schulsinger, and Mednick, 1964). Nothing conclusive

has been found that would convincingly show that schizophrenia is literally a dreaming state or that an alteration in the dreaming process is a basic factor in schizophrenia.

III
Schizophrenia and Epilepsy

The interest in EEG studies has renewed the old debate about the relation between schizophrenia and epilepsy. Once these two conditions were considered incompatible, and it was assumed that they could never occur together. As a matter of fact, it was because of this alleged incompatibility that Von Meduna (1937) had the idea of treating schizophrenia with an artificial convulsive chemical method, that is, with intravenous injections of metrazol. Later Cerletti and Bini (1938) replaced metrazol with electric shock.

This incompatibility now seems disproved, because several cases with the concomitant presence of the two disorders have been described. In many cases the question is the one well formulated by Tedeschi (1957): are we in the presence of a patient with epileptic psychosis or of an epileptic patient who suffers also from schizophrenia? Authors vary in preferring one answer to the other.

The authors who prefer the diagnosis of epilepsy speak of schizophrenic-like psychosis of epilepsy, for example, Beard and Slater (1962). These authors studied sixty-nine patients with a psychiatric syndrome that resembled schizophrenia. The psychosis was not related to the severity of the epilepsy. EEG changes showed that the epilepsy was of temporal lobe origin in over 70 percent of the cases. Zee (1965) speaks of a pseudoschizophrenic syndrome. According to him epileptic pseudoschizophrenic syndromes have one basic characteristic: they occur in hypnoid states, whereas idiopathic schizophrenia emerges in states of lucid consciousness.

Loeb and Giberti (1957) studied psychotic syndromes in psychomotor epilepsy. In the acute cases the psychosis was polymorphous in character. In the chronic cases the syndrome included hallucinations and resembled schizophrenia. During the acute psychotic syndrome the seizures and the EEG alteration disappeared.

Ervin, Epstein, and King (1955) made a study of forty-two patients with temporal spikes. Schizophrenia was diagnosed in 90 percent of those patients who also had temporal lobe seizures, and in

seven out of nine patients who never had an overt seizure. The authors reported in their patients “disturbance in thinking and affect which have come to be associated with the schizophrenic process.” They described the course of the illness as episodic. Strauss (1959), who is an epilepsy specialist, objects to putting these patients in “the wastebasket diagnosis of schizophrenia.” He prefers to call them “epileptics with psychotic episodes.” According to him it is a mistake to think of “acute recurrent schizophrenia” when there are positive EEG findings.

Glaser (1964) studied thirty-seven patients with psychomotor temporal lobe epilepsy, all of whom had definite psychotic episodes, with bizarre and inappropriate behavior, delusions, hallucinations, depressive mood, excessive religiosity, and paranoid ideation. One-third of the patients presented transitory catatonic features. A differential characteristic from schizophrenia was the continued effort on the part of the patient to maintain contact with reality.

After reviewing the literature and studying epileptic patients with chronic delusional psychoses, Bartlet (1957) found no evidence that schizophrenia occurs more, or less, frequently in epileptics than in

the general population. His EEG findings supported the idea that psychosis following epilepsy is related to temporal lobe dysfunction.

The following conclusions can be drawn from the mentioned studies:

1. Schizophrenic symptomatology may coexist with epilepsy, whether grand mal, petit mal, or psychomotor.

2. Dysfunctions of the temporal lobes, detectable with EEG, are often accompanied by symptoms that may be classified as schizophrenic or schizophreniclike.

3. In by far the majority of cases the schizophrenic-like or schizophrenic psychosis in epileptics occurs in the form of recurrent episodes.

I can state the following regarding the patients that I examined and who had been diagnosed as schizophrenics and had positive EEG findings of temporal lobe origin:

1. They had psychotic episodes, varying in length and recurrence.

2. They had a symptomatology of paranoid schizophrenia, often with mystical, religious, and philosophical delusions.

3. They had often presented either a rigid character structure or an angry, antagonistic, at times defiant attitude.

IV
The Possibility of Psychosomatic Involvement of the Central Nervous System in Schizophrenia

Three factors have greatly handicapped neurology in its effort to understand psychological and psychopathological problems. The first factor is shared with the rest of medical science. Physicians, trained in the scientific tradition, are reluctant to use any method that is not strictly empirical. Mental constructions, working hypotheses, are generally frowned upon as armchair speculations, more appropriate to the field of philosophy than to the field of medicine, and complete reliance has been put upon laboratory experiments and clinical observations. Obviously it would be absurd to minimize the tremendous accomplishments obtained through clinical and experimental research. Nevertheless, physicians should once more compare their research with those in the fields of physics and chemistry, where great progress has been made by a combination of experiments and a formulation of theoretical hypotheses. Such an eminent scientist as James B. Conant (1952) thinks that “the history of

science demonstrates beyond a doubt that the really revolutionary and significant advances come not from empiricism but from new theories." Obviously, sooner or later it is necessary to submit any new theory to the empirical test. In spite of the predominant, antitheoretical trend, even in the field of neurology, theoretical concepts like those of Jackson (1932) and the theory of emotion advanced by Papez (1937) have been very fruitful in stimulating important work.

The second factor that has delayed neurological progress toward psychological understanding consists of the poor use of data offered by related fields. For instance, I believe that a more intense application of a genetic approach, such as the one followed by Werner (1957) in the field of psychology, and a greater use of the findings of Cassirer (1953) and Langer (1942, 1949) in their studies on the symbolic functions of the mind could enrich neurology very much.

The third factor is the relative disregard of neurology that dynamically oriented psychiatrists (with a few outstanding exceptions, for example, Roy Grinker and Lawrence Kubie) have recently acquired. Overreacting to previous positivistic approaches, most of them feel

that neurology has not much to offer to the understanding of dynamic psychiatry, and they tend to ignore this field, whereas they maintain much closer relations with other branches of medicine—for example, internal medicine, allergy, dermatology, and so forth.

I am one of the dynamically oriented psychiatrists who believe that neurology has great contributions to make to psychiatry if its boundaries are enlarged.

An area where further expansion is urgently needed is the study of the central nervous system itself in the field of psychosomatic medicine. Only a historically determined trend toward disconnecting psychiatry entirely from neurology is responsible for the fact that whereas every organ or system of the body (such as skin, cardiovascular, and gastrointestinal apparatuses) has been recognized as affected by many psychosomatic disorders, the central nervous system has been given only secondary consideration. And yet the central nervous system is the organ of highest functionality; it is the organ that is first affected by psychogenic stimuli before they are channeled toward the other organs of the body. Could it not be that under a certain psychological stress a more or less specific functional

disintegration of habitual neuronal patterns takes place? A psychosomatic involvement of the central nervous system would require a mechanism different from those responsible for other psychosomatic conditions. In other words, the process would not originate through the action of the autonomic nervous system upon the organs of the body. The central nervous system would be the victim of the psychological conflicts that it produces; the conflicts or turmoils themselves would disrupt the organization of complicated neuronal patterns.

We have seen in Chapter 2 that Jung was the first to advocate a psychosomatic involvement of the central nervous system. Jung felt that the brain, disturbed by its own tumultuous conflicts, would produce toxins that in their turn would further damage the nervous system. In the following pages I shall consider the possibility of a functional alteration of the nervous system in schizophrenia. This alteration will not be considered at a biochemical, or molecular, level but at a preceding level: as a disintegration of usual neuronal patterns.

I shall not start by speculating on the possibility of a primary pathology of the nervous system but, rather, by positing the problem

of what parts of the nervous system are functionally involved in the schizophrenic symptomatology. In other words, I shall consider, not what parts of the nervous system are diseased, but what parts are functioning when a patient has ideas of reference, thinks paleologically, hallucinates, and so on. Then I shall discuss what type of coordination of the functions of these parts of the central nervous system is involved in the schizophrenic syndrome.

I am fully aware that the concepts expressed in this section cannot be fully proved at the present stage of our knowledge. I present them not as final conclusions but as working hypotheses, aiming to stimulate further research toward a closer integration of the psychological and the neurological.

This matter will be discussed in elementary terms. Readers who may be disturbed by the simplicity of the discussion should be reminded that this subject has led to a great deal of misunderstanding, and that therefore we should not be afraid of using too clear or simple language.

Let us start, therefore, with the most elementary question. Is

there any doubt that in schizophrenia the central nervous system is involved, in the sense that many of the symptoms are mediated or produced by the nervous system, although not necessarily because of an organic pathology of the nervous system? Of course not. For instance, it is obvious that the patient would not have delusions or a bizarre way of thinking if he did not have a brain. The central nervous system is as necessary for the production of these symptoms as it is necessary for the production of mental processes in the normal man. We may go further and say that at least most of the schizophrenic symptoms are produced in the cerebral cortex. Here again, from a theoretical point of view, we do not exclude the possibility of an extracortical pathology. Someone, let us say, may conceive that some pathology of the diencephalon is responsible for schizophrenia, or that the membrane of neurons is defective, or that an enzyme is lacking or an abnormal one is produced. In these cases too, however, symptoms such as delusions, hallucinations, ideas of reference, word-salad would require a cortical participation concomitant to the mentioned alterations. Most of the schizophrenic phenomena involve the functioning of the patient in a social and symbolic world; they affect his thinking and his planned actions, and all of these activities require

cortical centers. The schizophrenic symptoms require the function of the cerebral cortex, independently of whether the cortex is healthy or diseased.

The second step in our thinking consists in determining what cortical areas are involved in the pathological symptoms of schizophrenia. At this point we may make a general statement, already implied in the foregoing: all the cortical areas, whose function is well known today, do not seem to be *primarily* involved in the symptoms of early schizophrenia. For instance, it is obvious that the motor, sensory, extrapyramidal areas, and so on, are not primarily involved in schizophrenia. If a patient, as a result of a delusion, commits an absurd action, many cortical areas, such as the visual, pyramidal, extrapyramidal, are of course involved in the execution of this action, but the participation of these cortical areas is not pathological *per se.* It is the motivation, the symbolic meaning, and lack of control of the absurd action that are pathological. If we exclude the sensory and motor areas and the language centers as directly or originally involved in the psychopathology of schizophrenia, only three cortical areas are left to be considered. One is an ill-defined area including most of the temporal lobe and very small parts of the occipital and parietal lobes.

We shall call this area the TOP area—from Temporal, Occipital, Parietal (Figure 46). The second important area occupies the whole prefrontal area. We shall refer to this area with the abbreviation PF. The third area consists of the archipallium and mesopallium, including the rhinencephalon, the hippocampus, the cingulate gyrus, and possibly the posterior orbital gyri. Part of this area borders on the TOP area. We shall take these three areas into consideration separately.

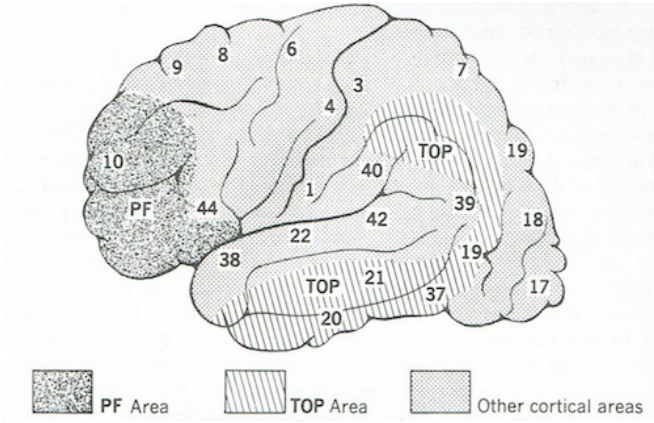

Figure 46

TOP Area

Anatomically, this area includes a large part of the temporal lobe (Brodmann's areas 20, 21, and 37) and a small part of the parietal and

occipital lobes, consisting of the most central parts of Brodmann's areas 7, 19, 39, and 40. The parietal part is crossed by the interparietal sulcus. Histologically in Nissl sections it resembles the structure of the sensory associative areas. It receives projections from the lateral thalamic nucleus and from the pulvinar. The part of area 19 that is included in the TOP area possibly belongs more to the parietal than to the occipital lobe. It also has projections from the pulvinar.

Much less is known anatomically of the temporal part of the TOP area. Krieg (1947) calls this area one of the great *terrae incognitae* of the cortex. Thalamic connections to and from this part of the cortex have not been well established.

Phylogenetically the TOP area is one of the most recently evolved areas. Even such high species as *Pithecanthropus erectus* and especially *Homo Rhodesiensis* had only a rudimentary development of this area, as may be deduced from Tilney's writings (1928). Furthermore, together with other neopallic areas, it has no direct connections with the "reticular system" of the brain stem.

From a point of view of neuropathology, the TOP area is rather

seldom involved in pathological conditions that could reveal its specific functions. Hemorrhages, softenings, or neoplasms are generally unilateral. In addition they either do not involve the totality of this area, or, if they do, they expand to surrounding regions.

In senile psychosis and cerebral arteriosclerosis the TOP area is involved less than the prefrontal area. Maybe this relative resistance to the senile or arteriosclerotic process is to be attributed to a different blood supply. Whereas the prefrontal area receives its supply mainly from the branches of the anterior cerebral arteries, the TOP area is irrigated by branches of all the three major arteries of the brain.

The TOP area is involved in Pick's disease, but this point will be taken into consideration later in this chapter.

From a physiological point of view the TOP area may be considered as the center of functionality of a much larger area including the whole parietal, occipital, and most of the temporal lobes. These three lobes form the part of the brain that receives stimuli from the external world and processes them into progressively higher constructs. We may divide this large part of the brain into four levels.

Before examining these four levels separately, however, we must make it clear that we do not consider them as sharply defined physiological entities. When we attribute a function to a particular cortical area, we mean only that that function is represented predominantly but not exclusively in that area. The cortex is to be conceived, not as a mosaic of distinct localizations, but as a pattern of overlapping representations, which are all more or less connected, and which also have vertical associations. These representations, however, have a relative concentration in certain areas. When we refer to levels, we refer only to these relative concentrations.

The first level consists of what Orton (1929) called the arrival platforms. It includes the borders of the calcarine fissure, Heschl convolution, and the postcentral gyrus (with other contiguous small areas of the parietal cortex and small portions of the precentral gyrus). These are the areas where crude sensation occurs.

The second level is the level of perception or recognition. It consists of area 18 for vision, a portion of the first and maybe second temporal gyrus for hearing, and an undetermined area of the parietal lobe for general sensation. If a lesion occurs at this level, the patient is

not able to perceive, that is, to recognize what he experiences. He suffers from various forms of asymbolias, for instance, psychic blindness. At this level conditioned reflexes occur. This is also the level at which the symbolic activity of the mind manifests itself in the form of *signs* (see Chapter 19).

The third level (parts of Brodmann's areas 19, 22, 39, and 40) is much more complicated. Although it may exist in a very rudimentary form even in some high infrahuman species, it is only in humans that it acquires prominence. The following are some of the functions of this complicated level.

As Nielsen (1946) has described, voluntary recall of past sensations and experiences takes place at this level. This function implies some kind of primitive abstraction because in recalling one must separate or abstract a recognized experience from the other memories with which it was associated. The experience that was recognized at the second level is now abstracted from memories and is voluntarily revisualized. In order to function in this way at this level, the subject must have the ability to reproduce mentally *the image* of the external stimulus. Images are not signs, but *symbols,* inasmuch as

they stand for something that is *not present* (see Chapters 5 and 19).

At this level many other functions occur. Not only images, but external stimuli (caused or not caused by the individual) start to stand for something else that is not present and, therefore, become *symbols.* Phylogenetically, symbols are preceded by *paleosymbols,* which are symbols valid only for the person who creates them. For instance, a person sees a connection between a sound and a situation or object, and that sound becomes the paleosymbol of the object or the situation. The paleosymbol becomes a symbol when its symbolic value has been accepted by at least a second person. The symbol produces in the person who pronounces it the same response that he produces in others (Mead, 1934). Verbal communication and socialization start at this level. Most probably at this level the verbal symbols denote, but have very little connotation power (see Chapters 16 and 19). Connections of this third level with the frontal lobes permit an expansion of the faculty of speech. All the language centers belong to the third level, although, as we shall see shortly, they would not have expanded without the influence of the fourth level.

Most authors follow Orton (1929) in recognizing only the three

mentioned levels of elaboration. I feel that a fourth level must be differentiated, which anatomically consists of the TOP area (Brodmann's areas 20, 21, 37, parts of 7, 19, 39, and 40). This is the area where all the excitements coming from the lower levels are synthesized and elaborated into the highest mental constructs. Of course, as we have already mentioned, we must not consider this area as isolated or functioning by itself. First of all, it is associated with the corresponding area of the opposite hemisphere by means of fibers that pass through the corpus callosum. It is also connected with much lower structures through the archipallium. As we shall see shortly, it has important connections with the frontal lobes, without which it could not function at all. Furthermore, one should by no means think that only the TOP area is required for the highest mental processes. The neuronal network that mediates the highest mental processes extends to much lower levels (see engram in Figure 47). However, the highest engrams, or esthesotypes, to use a very appropriate concept proposed by Mackay (1954), need a place in the TOP area, almost as a center toward which all the associations of which they consist converge. The fact that the esthesotype extends to many lower levels has confused several researchers and has fallaciously led to

conclusions that no cortical localization is possible and that what counts is only the extension of the cortical area.

But let us go back to the TOP area. In my opinion this area is needed for the highest processes of abstraction. These processes presuppose and require processes of socialization, which in simple forms have already started at the third level. As we have seen in Chapter 19, no high abstraction is possible if the individual in his development has not come into contact with other people. It is through contact with other human beings that verbal symbols continue to increase in number and that those previously acquired develop abstract meaning, that is, *connotation* or *categorical significance.* For instance, the term *mother* eventually refers not only to the mother of the individual or the mother of all the siblings, a concept that is generally connected with one visual image, but to any mother, to mothers in general. In the TOP area, the thought processes become more and more elaborated and more distant from paleosymbols, images, and perceptions. The TOP area also facilitates or permits the full expansion of the third level. In fact, language would consist only of a few words, and only with *denoting* power, if processes mediated at the fourth level did not require a tremendous increase in

vocabulary. This is another instance of the overlapping of levels (Chapter 19).

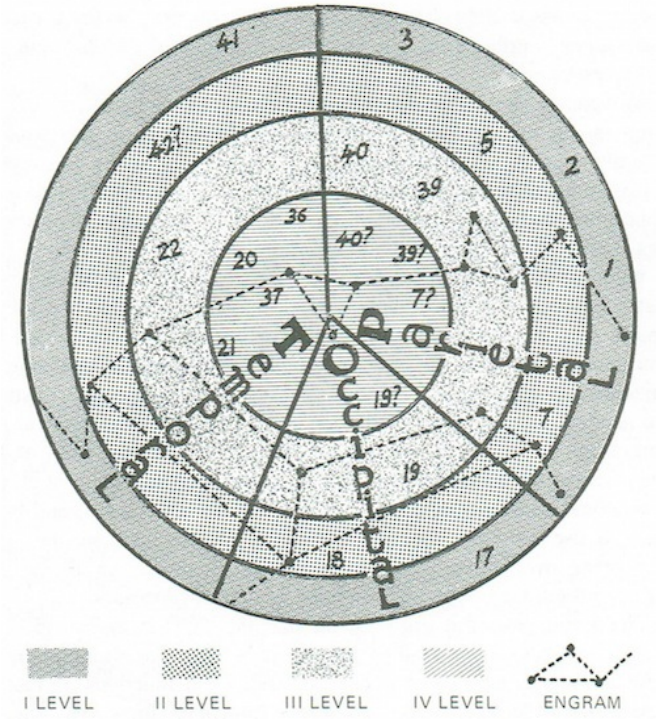

Figure 47

In Figure 47 the temporal (with the exception of the hippocampus and related structures), the occipital, and the parietal lobes are represented in a circular diagram. The four concentric circles represent the four different levels of integration. At the center is

located the TOP area toward which all the other levels of elaboration converge. Like all diagrams, this one is intended merely as a didactical device and, as such, oversimplifies complicated neurological mechanisms. Among the important things that, for the sake of simplification, are omitted from the diagram are: (1) the associations between the different parts of the temporal, occipital, and parietal lobes; (2) the associations with other cortical areas, for example, the frontal lobes; (3) the associations between the two hemispheres; (4) the cortico-subcortical associations. Furthermore, the spatial proportions of the different areas are not exactly respected.

In my opinion there is no doubt that the functions mediated in the TOP areas are altered in the schizophrenic syndrome. We have seen in this book how in schizophrenia there is a gradual return from the abstract to the concrete, from a form of highly socialized symbolization to a form of symbolization that is decreasingly conceptual and social and more perceptual in character. Even the thought processes, deprived of abstract symbolization, abandon our common logic and become paleologic. At first the symbols lose the connotation power and denote only; then they become paleosymbols, that is, they are understood only by the patient; finally they become

completely perceptualized or regressed to the levels of images or perceptions, as in the case of hallucinations. In other words, in progressive schizophrenia there is an increasing inability to use engrams or esthesotypes that need neurons located in the TOP area. At first there is a tendency to lose only the functions of the neuronal patterns that include the central parts of the TOP areas; later, as the illness progresses, there is a tendency not to use at all the functions of neuronal patterns that require any part of the TOP areas, and there is an inclination to use only the functions mediated by the first, second, and third levels. In other words, there is a reduction or simplification in the neopallic neuronal configurations of the highly symbolic mental processes.

The foregoing does not imply at all that the TOP areas of the two hemispheres are affected by some kind of organic pathology. The disintegration or loss of their functions may be viewed as psychogenic. Their functions, in fact, by permitting complicated interpersonal relations and human conflicts, may engender the greatest anxiety in human beings.

How the psychogenic mechanism brings about a dysfunction of

these areas is a matter of speculation. It could be that the intensity of the emotional impact produced by these neuronal patterns is enough to make the complicated chains of these neuronal patterns disintegrate; or it could be that the rest of the nervous system learns to avoid these patterns or to do without them. Whatever the mechanism is, there seems to be little doubt that these areas do not function in the usual way or are not integrated well with the rest of the nervous system.

With this partial or complete psychogenic decline or elimination of the functions of the TOP areas, the schizophrenic does not obtain adjustment at a lower level of integration. In the process of evolution all the nervous areas readjust themselves by means of new associations every time a new area appears, changes, or extends. The entire nervous system and especially the cortex is in a state of maladjustment when the TOP areas are in a state of decreased functionality, even if this decreased functionality is psychogenic in origin. In other words, the schizophrenic regresses but does not integrate at a lower level, just as an experimental animal of a species normally provided with the cortex does not integrate at a noncortical level when the cortex is experimentally removed, because its whole

organism is adjusted or attuned to the presence of the cerebral cortex. The schizophrenic's disintegration will continue. Furthermore, together with the elimination of the functions of high levels (Jackson's negative symptoms), there is a resurgence of inhibited functions (Jackson's positive symptoms), such as paleologic thinking, perceptualization, and so forth. The chronic patients who had been sick for many years, and whom we discussed in Chapters 25 and 26, presented agnosia to pain, temperature, and taste, and a syndrome not too dissimilar from psychic blindness. They also showed primitive habits reminiscent of the ones described by Klüver and Bucy (1937, 1938, 1939) in monkeys after bilateral removal of the temporal lobes. The crude sensations remained. It could be that in these patients the areas of functional impairment extended beyond the TOP areas and involved also the third level and part of the second.

PF, or Prefrontal, Area

For didactical purposes we have taken into consideration the TOP area first, but the PF areas are even more important in the psychogenesis of schizophrenia. The TOP areas elaborate the material coming from the external world, but it is the PF areas that permit this

elaboration to a degree where schizophrenogenic conflicts are possible.

Increasing evidence indicates that the functions of the PF areas are very important, even if these functions are hard to define and, to a certain extent, still obscure. In addition to control of some visceral functions, at least four psychological functions of the PF lobes have so far been recognized. These four functions are certainly interrelated and are possibly different manifestations or different degrees of the same basic process.

The first function is the ability to maintain a steadfastness of purpose against distracting impulses from the environment (Malmo, 1942); in other words, it is the function of focal attention. The importance of focal attention as a prerequisite for higher mental processes has been very well illustrated by Schachtel (1954). As Schachtel wrote, each act of focal attention does not consist just of one sustained approach to the object to which it is directed but of several renewed approaches. Focal attention requires ability to suppress secondary stimuli and to delay the response.

It is obvious that the high elaborations of stimuli described in connection with the TOP areas could not take place if this function of the PF areas would not permit it.

A second function of the PF areas is the ability to anticipate the future. Whereas animals are able to anticipate events that will occur only within a very short period of time (from a few seconds to a few minutes after the stimulus takes place), man is able to anticipate mentally distant events. The importance of this faculty in the engendering of anxiety cannot be overestimated; in fact, anxiety is based on *anticipation* of danger, as Freud repeatedly wrote. Without the faculty of distant anticipation, anxiety is possible, but only a short-circuited anxiety similar to that experienced by infrahuman animals (Arieti, 1947; also Chapter 5 of this book). In animals, anxiety is experienced when the stimulus indicates a present danger or a danger that will follow shortly after the stimulus or when the animal is at the same time stimulated by two conflictful stimuli. In addition to this type of anxiety, human beings experience long-circuited anxiety, that is, an anxiety due to anticipation of distant danger. This anxiety persists even when the external stimulus has disappeared, because the external stimulus is replaced by an internal one, that is, by a chain of

mental processes that permit the anticipation of distant future. Anticipation of distant future would not be possible if the individual could not receive high forms of symbols from the third and fourth levels of the posterior brain. For instance, a calf considers his mother to exist only when he perceives her, that is, when the visual stimulus of the cow or another stimulus that is simultaneously associated with the cow or follows at a very brief interval is perceived. But a man can think of his mother even when the mother is absent, because he is able to substitute mother with the symbol "mother." The symbol "mother" places mother in the three temporal dimensions: past, present, future.

A third function of the PF areas is the ability to permit planned or seriatim functions. By seriatim functions is meant the organization or synthesis of skilled acts or thoughts into an orderly series (Morgan, 1943). Although some high species, like monkeys and apes, are capable of simple seriatim series, this function expands very much in man. Seriatim functions imply ability (1) to anticipate a goal and (2) to organize and synthesize acts or thoughts in a given temporal sequence for the purpose of reaching the anticipated goal (Brickner, 1936).

The fourth function of the PF areas is the ability to make choices

and to initiate the translation of the "mental" choice into a motor action.

Some experiments have proved that infrahuman animals also learn to choose; that is, they learn what choice to make in certain experimental sets and what choice not to make (Hamilton, 1911; Yerkes, 1934). These experiments are valuable in the study of "equivalent stimuli" and of a function of primitive abstraction, but they do not really demonstrate the presence of the ability to choose as it exists in human beings. Long-circuited choice as it occurs in human beings requires many steps, as we have seen in Chapter 17, section V.

These steps could not take place if the posterior brain did not provide high forms of symbolism.

There is no doubt that these functions of the PF areas are disturbed in schizophrenia. The function that comes first to our mind is the ability to anticipate the future. This function is an absolute prerequisite for long-circuited anxiety and is the basis of any complicated mental conflict. In schizophrenia it tends to be teleologically replaced by more primitive processes that require

shorter circuits (see Chapter 16). The same thing could be repeated for seriatim thinking. Greenblatt and Solomon (1953) feel also that the frontal lobe circuits sustain the emotional tension of the psychotic, and Freeman and Watts (1942) have well described the importance of future anticipation in the engendering of psychoses. These functions do not seem to be very disturbed in well-systematized paranoids, possibly because at the beginning of the illness these patients resort predominantly to the mechanism of projection in the attempt to remove anxiety. At a subsequent stage, however, these functions become impaired in paranoids too. The delusions change from the persecutory to the grandiose type and are more and more related to the present time: "I *am* a king."

The other PF functions are also disturbed in schizophrenia. The ability to make choices or to translate thoughts into actions is particularly altered in catatonia. As to the ability for steadfastness or focal attention, we know that it is very much impaired in schizophrenics. Their span of attention is very limited.

Archipallium and Hypothalamus

The archipallium and the hypothalamus may be thought of as playing an important role in schizophrenia. Because great importance is attributed to these structures in the mechanism of emotions, we could even be inclined to think that an altered functionality of the archipallium could explain the affective impairment that is so pronounced in schizophrenia. One could even be induced to the hasty conclusion that the original cause of this mental condition is to be found in this part of the nervous system. At the present stage of our knowledge, such possibility cannot be denied categorically. However, such an interpretation seems improbable to me.

I do not mean that the archipallium is not involved in schizophrenia. On the contrary, a neopallic dysfunction, even if functional, is bound to have important repercussions upon the archipallium too. If we follow again Jackson's principles, we may think that the hypofunctionality of neopallic areas must be accompanied by a release and hyperfunctionality of the archipallium. Because we know so little about the functions of the archipallium and of its single parts, we cannot explain how this release is manifested. At first one would think that the release of the archipallium would increase the emotionality of the patient, but we have to remember that with the

term *archipallium* we include many structures representing possible different sublevels, and that their specific action in the experience of emotions is unknown. At any rate, the release of the archipallium will increase the inhibitory power that this part of the brain exerts over lower structures, such as the hypothalamus. This inhibition of the hypothalamus may explain the hypofunctionality of the autonomic nervous system, as far as homeostatic reactions are concerned, as well as the decrease in somatic and visceral expressions of those emotions that are still experienced in schizophrenia, in spite of the neopallic-symbolic disintegration.

The interesting study by Bard and Mountcastle (1947) may support this point of view. These authors found that removal of the neocortex produces a state of placidity in cats. Anger or sham rage never occur. Nociceptive stimuli elicit mild responses. If these animals are subjected to ablation of the cingulate gyrus or to various parts of the rhinencephalon, they become angry and ferocious. These experiments suggest that the rhinencephalon restrains the hypothalamus. This action is opposed to the one exerted by the neocortex; in fact, removal of the cortex increases the inhibitory effect of the rhinencephalon.

Incidentally, the rhinencephalon may exert a restraining influence not only over the hypothalamus, but also on the thalamus, possibly through some hypothalamic-thalamic connections. We have seen that in the terminal stage of schizophrenia pain and temperature perceptions are decreased or abolished (Chapter 25). We have interpreted this phenomenon as a consequence of some possible psychosomatic dysfunction of the parietal cortex. Penfield and Rasmussen (1952) have, on the other hand, found that pain and temperature are experienced in humans at a thalamic level when the cortical areas are removed. In the terminal stage of schizophrenia an increased ability to experience pain and temperature should occur on account of a theoretically presumed release of the thalamic level. The opposite is the case. It may be that some restraining action exerted on the thalamus by the archipallium, or lack of stimulation from the hypothalamus, is involved in these processes. The fact that frontal leucotomy relieves intractable pain gives additional support to this point of view.

We have to mention that this altered functionality of cortical centers in schizophrenia also puts into a certain disequilibrium the so-called visceral brain (MacLean, 1949), that is, those cortical and

subcortical centers that are now considered the highest representation of visceral functions. A dysfunction of the autonomic nervous system may be partially due to their altered functionality. Also the inhibition of the hypothalamus may reduce the excitement that normally reverberates from the hypothalamus to the cortex, and this lack of excitement may add to the dysfunction of the neocortex. Hypothalamic-thalamic and thalamic-cortical relations may also be altered as a result of this disequilibrium. Figure 48 is a diagram of these interrelations. As every diagram does, it represents an oversimplification of the processes involved.

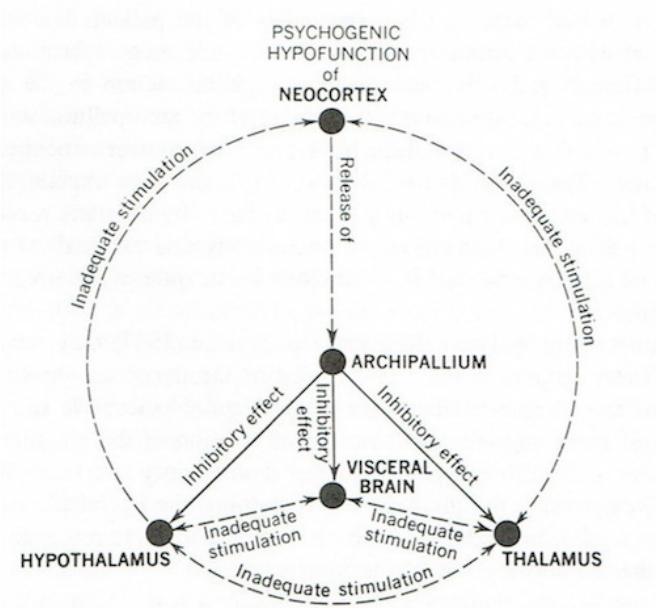

Figure 48

General Interpretation

We shall recapitulate now what we have mentioned about the possibility of a psychosomatic involvement of the central nervous system in schizophrenia.

Whatever is the origin of the series of events that ultimately leads to the psychosis, the overwhelming anxiety, the difficult interpersonal conflicts, and the disturbing high forms of symbolism are

predominantly mediated in those cortical areas that in this chapter have been called the PF and the TOP areas. These areas are the highest in the evolutionary scale; they are the last to appear in phylogeny and the last to myelinize in ontogeny. They are probably the most vulnerable areas of the central nervous system from a functional point of view, at least in those people who have a hereditary predisposition to schizophrenia. This hereditary vulnerability may be direct, that is, immediately involving the functions of these two areas, or indirect, by disturbing another area whose normal functioning is a prerequisite for the good functioning of the PF and TOP areas. The impact of the psychological conflicts is too much to bear for a part of the central nervous system that is already genetically vulnerable. At times it may be too much to bear even if such hereditary predisposition does not exist. On the other hand, the hereditary vulnerability alone, without the psychogenic factors, would not be enough to engender the disorder. A disintegration of the neuronal patterns that involve predominantly these two areas may take place, and a reintegration or formation of simpler circuits that use these high areas less and less, and eventually not at all, may follow.

This neuronal reintegration may thus be seen deterministically

and also adaptationally or restitutionally. In fact, there seems to be a psychosomatic attempt to return to lower levels of integration, levels that do not permit complicated interpersonal symbolism and long-circuited anxiety. Orton (1929) too thought that shorter circuits or crossovers are used in schizophrenia and especially in catatonia, but he thought also that the process involved was organic and affected specifically the centers connected with the longer circuits.

In my opinion, at the same time that these cortical centers or parts of them are in a state of hypofunctionality or dysfunction, other centers or some other parts of them or some lower neuronal configurations or patterns are released and cause characteristic symptoms. Jackson's concepts are valid in relation to schizophrenia too, if correlated with psychodynamic principles. For instance, when logical thinking is impaired, paleologic thinking comes to the surface. When social symbols disappear, paleosymbols replace them. Concepts become more and more perceptual, and anticipation of the future is replaced by thoughts concerning the present.

One could raise the objection that although an organic condition affecting *bilaterally* the PF and the TOP areas should give a

symptomatology similar to schizophrenia, so far this occurrence has not been reported. Actually the only organic disease that would approximate a bilateral involvement of these areas, and these areas exclusively, is Pick's disease. In Pick's disease the symptomatology is similar to that found in cases of very regressed schizophrenics who have been sick for many years, but it has very little in common with the symptomatology of early cases. In both Pick's disease and schizophrenia there is an impairment of the abstract attitude described by Goldstein, that is, there is occurrence of Jackson's negative symptoms. However, in schizophrenia there is also a resurgence of many positive symptoms (paleologic thought, hallucinations, and so on) that are not present in Pick's disease. This may be due to the fact that in Pick's disease, as well as in other organic conditions, several levels are affected at the same time, and therefore a release of inferior cortical processes is prevented. We know in fact from neuropathological studies how diffuse the alterations are. In Pick's disease they involve not only association areas, but also primary centers and many subcortical structures (Ferraro and Jervis, 1936). Even when the lesions are patchy, we can hardly believe that they selectively involve only some higher neuronal configurations and

respect lower ones. This state of affairs explains why Jackson's positive symptoms are not so clearly seen in organic conditions that involve the cortex.

The similarity of regressed schizophrenics to organic patients with involvement of the cerebral cortex is more pronounced than is generally assumed. For instance, Figure 49 reproduces the letter of a patient, the content of which resembles schizophrenic word-salad. Actually, this letter was written by a nonpsychotic patient suffering from motor aphasia. She was a young woman who had been shot by her irate husband. The bullet damaged her Broca's area and surrounding regions. She was not able to talk, but she understood simple spoken language. She was relearning how to write, but she could not find the right words. This letter was written to me on my last day of service at Pilgrim State Hospital. The patient, who knew I was leaving, wanted to say farewell and to thank me. In the letter, however, she seems to express ideas different from, or even the opposite of, those that she wanted to express. The result is something similar to word-salad. The difficulty may be the same one that the regressed schizophrenic presents. Each part of the mental context is equivalent to another; the patient is unable to pick up the right part. An organic

patient who replies to the question, “What city is the capital of France?” with, “La Marseillaise,” probably uses the same mental process as that of the schizophrenic who replied, “White House,” to the question, “Who was the first president of the United States?”

Brown (1972) has stressed the similarities between some aphasics and some schizophrenic patients. According to him the type of aphasia that resembles most the language of the regressed schizophrenic is semantic aphasia, which is a midway state between anomic aphasia and Wernicke’s aphasia. Semantic aphasia, first described by Head (1926), reflects an interruption of language at a prelinguistic phase in the thought-speech transition. According to Brown it is “characterized by a want of recognition of the full significance of words and phrases apart from their verbal meaning, failure to comprehend the final aim or goal of an action, and inability to clearly formulate a general conception of what has been heard, read, or seen in a picture, although many of the details can be enumerated.” Brown collects from the literature cases of aphasias with semantic jargon reminiscent of schizophrenic language and thought. He quotes a patient, reported by Kinsboume and Warrington (1963), who was asked to explain the proverb “Strike while the iron is hot.” The patient

replied, “Ambition is very very and determined. Better to be good and to Post Office and to Pillar Box and to distribution and to mail and survey and headmaster. . .

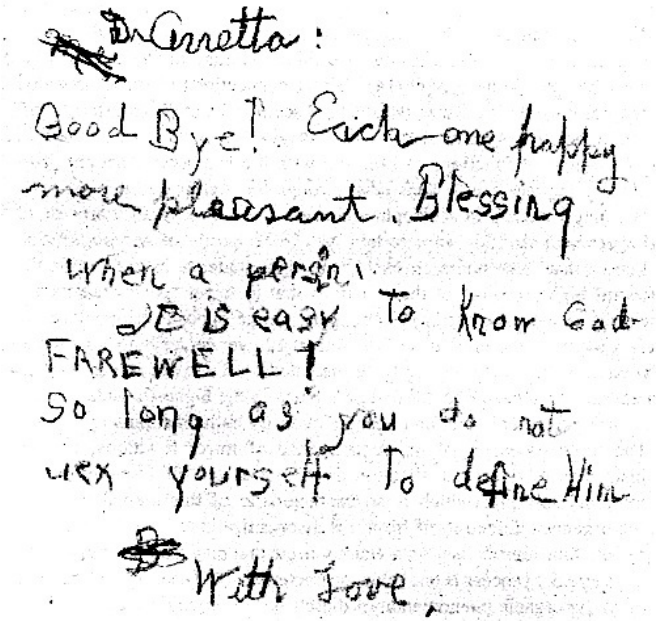

Figure 49

This sample and many others from semantic aphasics, are reminiscent of schizophrenic word-salad and at times of pseudoabstract schizophrenic language. It could be that in the semantic aphasics and in regressed schizophrenics the same cortical areas are involved, although the etiology and pathology differ.

To return to our regressed schizophrenic patients, we can state that the reemerging primitive functions as a rule are not useful, although they include also those restitution symptoms studied by Freud. In fact, as we have already mentioned, the regressed patient does not become adjusted, but maladjusted at a level at which he cannot integrate. The hypofunctionality of the neopallic areas that we have examined must also alter associations with other areas, not only the neighboring one but also the distant, through long association bundles, commissures like the corpus callosum, the cortico-thalamic and thalamo-cortical tracts, and so on. This is a process of functional diaschisis, similar to the organic diaschisis described by Von Monakow (1914). Direct connections of these neopallic areas with the brain stem reticular system do not seem to exist (Livingston, 1955). The brain stem reticular system is of ancient origin and is more concerned with the basic physiological functions of life than with the symbolic functions. However, undoubtedly indirect connections exist through the archipallium.

We may visualize in schizophrenia also what we may call a process of functional *dysencephalization,* in a certain way the opposite of *encephalization.* It is well known that some neurologists have used

the latter term to describe the shifting toward higher centers of the functions that in lower species are mediated by lower or more caudal centers. In schizophrenia the opposite takes place: not only is there a return of the functions of the released lower center but also a tendency of the function of the higher center to be mediated by the lower center. For instance, often abstract thought processes that take place in the highest centers are mediated instead at a perceptual level and become auditory hallucinations.

The three processes of the reemergence of lower functions, of functional diaschisis, and of dysencephalization determine a state of disequilibrium and of psychological splitting, which is so characteristic of this mental disorder. How does the organism defend itself from this disorganization? With further regression. The process thus repeats itself in a vicious circle that may lead to complete dilapidation. This circular process is one of the characteristics of functional regression that is lacking in the organic phenomenon of diaschisis. In organic conditions there is not complete disintegration because the resurgence of the functions of lower levels or sublevels is minimal in comparison to functional conditions. If further deterioration occurs, it is only because the organic process itself expands or produces other

damages—for example, gliosis, hydrocephalus, hemorrhages, and so on. Some intoxications, however, just as those caused by mescaline, may be selective enough in their physiological action to reproduce a specific cortical hypofunctionality, and subsequent subcortical dysfunctions, similar to the ones occurring in schizophrenia.

In conclusion, at least in those cases that progress beyond a very initial phase, the schizophrenic process elicits psychosomatic attempts to integrate the highest cortical functions at a lower level. With comparatively few exceptions this attempt fails because the process engenders other self-perpetuating mechanisms that lead to regressions.

An artificial partial attempt to integrate the schizophrenic at a lower level is made by frontal leucotomy and other psychosurgical procedures. Theoretically these procedures have some validity because they remove, at least partially, the functions of such areas as the PF and do not unchain progressive regression. However, to change a disorder that is at least partially psychogenic for one that, together with symptoms, removes permanently a great part of the essence of man and reduces him to an almost infrahuman state seems more than

questionable to those who today obtain an increasing number of successes with different therapeutic approaches, even in very difficult cases.

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