A NEUROPHYSIOLOGICAL THEORY OF SCHIZOPHRENIA*

By

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IN the psychiatric literature in English there is a tendency today to deprecate speculative neurophysiological theories of the "functional" psychiatric disorders, while little is done to halt the flood of far-fetched and untestable hypotheses of the "dynamic" psychopathologists. The present author believes that it is legitimate to postulate a neurophysiological basis for schizophrenia in the hope that those with a more adequate knowledge of neurophysiology will be obliged to re-examine the problem of schizophrenia more fruitfully. Conrad (1958, and Fish, 1960) has given a very interesting interpretation of acute schizophrenic symptoms, so that his approach to schizophrenia forms a very convenient starting point for further speculation. This worker's views will therefore now be presented in some detail before the present author's theories are discussed.

A.—THE GESTALT THEORY OF SCHIZOPHRENIA

Using Gestalt theory, Conrad has put forward the view that in schizophrenia there is a loosening of the coherence of perception and thought which results in the emergence of new gestalts† and the fragmentation of psychic activity. He divides the acute schizophrenic shift into five phases, viz.:

1. The Trema.
2. The Apophanous Phase.
3. The Apocalyptic Phase.
4. The Consolidating Phase.
5. The Residual Phase.

We will now consider each of these in turn.

1. *The Trema*

This word comes from the German stage slang for the stage fright which is experienced just before the actor makes his entrance. In this phase the patient feels that he has lost his freedom of action, is somehow shut in and unable to communicate with his environment. Often there is marked anxiety, but depression is also common and usually associated with ideas of guilt and disgust with life. Frequently during the trema the patient carries out rather silly actions which he rationalizes, but which are out of keeping with his personality and the total situation. These senseless actions may lead the patient into difficulties which may then be incorrectly understood as reactive factors in the production of the depressive mood or the schizophrenic symptoms. In many patients there is a general feeling of suspicion which pervades all experiences and leads to the feeling that there is "something going on". In the end a delusional mood occurs in which the environment is experienced as being changed in a strange and

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† In order to avoid confusion, the word "gestalt" will be treated as an English technical word, so that the plural form is gestalts, not gestalten, which is the German plural.

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threatening way. Conrad believes that there is scarcely a case of early schizophrenia in which this phenomenon does not occur. The delusional mood marks the point of transition from the trema to the apophanous phase. The trema itself may last for days, weeks or months and may even subside before the next phase can occur.

2. *The Apophanous Phase*

Delusional experiences in which a delusional significance occurs in connection with a psychological event such as a perception or a sudden idea have been considered by most German workers to be the primary irreducible elements of schizophrenic delusions. Since the German technical terms for these phenomena are cumbersome, Conrad has introduced the term apophany to include all these delusional experiences. This word, which is Greek for "becoming manifest", is a very suitable term for these strange experiences, in which a new meaning becomes manifest in connection with a psychological event. This word also has the advantage that the adjective apophanous can be derived from it, so that it is possible to refer to apophanous ideas, moods, perceptions and so on. Conrad has therefore called the phase in the schizophrenic shift in which delusional experiences occur the apophanous phase.

Before we consider the apophanous phase in detail, we must discuss delusional perception, since this was the first schizophrenic symptom which was subjected to Gestalt analysis (Matussek, 1952, 1953). Delusional perception occurs when an abnormal significance, usually in the sense of self-reference, is attributed to a normal perception in the absence of any rational or emotional reason. The older German investigators all insisted that delusional perception was not due to a disorder of perception itself, but a disorder of thought. However, recently Matussek (1952, 1953) has pointed out that there are two varieties of delusional perception, one in which perception is disordered and the other where verbal associations are important. An example of this second variety is a patient of mine who heard a floorboard squeak as a colleague stood on it and looked down to see the linoleum. The word "Lino" came into his mind and was followed by the thought "No lie", which he took as an instruction from his colleague that he was not to lie. This resembles the play upon words of the obsessional ruminator who repeatedly finds indications of his obsessional thoughts in his environment. In the other variety of delusional perception, Matussek believes that there is a loosening of the coherence of perception which allows the essential properties of the object to come into undue prominence.

This concept of the essential property comes from Metzger (1954) who considers that percepts may have three types of gestalt property, structural properties, total quality properties and essential properties. Structural properties are those of arrangement and organization, such as figural form, brightness, colour profile and so on. The properties straight, round, closed and constant are examples of structural properties. The total quality properties are material properties which are not simple sensory qualities independent of structure. The properties indicated by the adjectives transparent, rough and soft are examples of total quality properties. Essential properties are expressions of the essence of the object and include all the physiognomic or expressive properties such as character, ethos, habitus, mood, emotional value and so on. Solemn, friendly, proud and elegant are examples of these properties. Although they have been called "subjective impressive qualities" or subjective impressions, Metzger insists that they are perceptually given properties which immediately make an impression on us and affect our own essence.

The apophanous phase can be divided into two subphases, apophany of external space and apophany of internal space. In the first, all external events which are experienced acquire a new significance, while in the latter internal psychic events acquire a special meaning. One can use a more familiar jargon and talk of apophany of perception and apophany of the mediating processes.

Delusional perception is the most common definite external apophanous experience and Conrad has differentiated three stages of this phenomenon, viz.:

Stage 1. The perceived object indicates to the patient that it concerns him, but he cannot say to what extent. This is pure apophany.

Stage 2. The perceived object indicates to the patient that it concerns him and he knows the extent of this immediately. Thus, for example, he may know that it has been put there to test whether he observes it. Thus the patient has the experience that the object or sequence of events has been made for him, or, in other words, he has prefabricated experiences.

Stage 3. The perceived object signifies something quite definite, and the essential properties of the percept have come into prominence because of a change in the total structure of perception. This is delusional perception in the strict sense of the word.

This undue prominence of essential properties explains the misidentification of persons, which is common in the apophanous phase. Unknown persons may be recognized as friends or acquaintances, while relatives and friends are not recognized as such by the patient. The emerging essential properties can be considered as causing these confusions of identity.

The patient is, of course, in the ptolemaic position in that he experiences himself at the centre of things. This being so, he may attribute a delusional significance to his actions and have the experience of omnipotence. Conrad calls the experience of being the centre of the world "anastrophe" and regards it as the subjective aspect of apophany or, as he puts it, "Whenever there is apophanous experience, the ego must, at the same time, be anastrophically changed."

From Conrad's case material it seems as if there is a barrier which stops the schizophrenic process from passing quickly from the perceptual field to the mediating processes. In some cases apophany only affected external space, while in others it was some time before internal space was affected. Once internal space is affected, there is a loosening of the coherence of the mediating processes. Memory images may lose their connection with the total field and be experienced as delusional inspirations. Thought broadcasting in which the patient's thoughts become manifest to the environment can be regarded as the reverse of delusional perception, where a new significance of a perception becomes manifest to the patient.The loss of adequate figure ground relationships in conceptual thinking naturally leads to the patient hearing his own thoughts spoken aloud, so-called Gedankenlautwerden. As the disorder becomes worse, all personal indication of the thoughts is lost and hallucinatory voices occur. Bodily hallucinations can be understood as the effect of apophany on bodily sensations and the body image.

3. *The Apocalyptic Phase*

If the schizophrenic process is very severe, the loosening of the coherence of perception and of the mediating processes may lead to fragmentation of psychic life or the apocalyptic phase. This is another name for catatonia, since the release of the representations of bodily sensations and body movements

leads to a gross motor disorder. Sense continuity is destroyed, and this accounts for the fact that frequently only fragments of the total experience can be remembered after an acute catatonic illness. Rarely the gross fragmentation of psychological activity in the apocalyptic phase leads to death. This is the acute, deadly catatonia of Stauder.

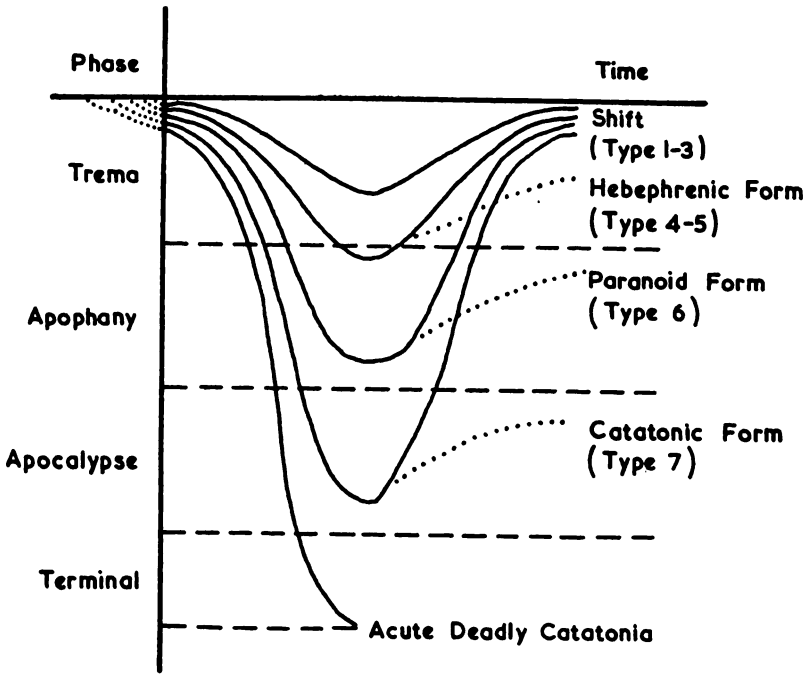
4 and 5. *The Consolidatory and Residual Phases*

The phase of consolidation begins after a few weeks or months, and a final residual phase occurs. In this phase there may be no active symptoms, but the patient feels less capable intellectually than before the shift, but this change is often more subjective than objective. Conrad suggests that every individual has his own energy potential or his particular ability to direct and apply his energies and that this potential is lower than normal in the residual phase.

Conrad has described seven different types of course of illness in a schizophrenic shift. These are shown in Figure 1. They are:

Type 1. The process does not pass beyond the trema, only abuts on the apophanous phase and usually subsides in a few weeks. The loss of energy potential is minimal, so that the defect is very slight.

Type 2. The process passes through the trema, enters the apophanous phase and subsides after a few weeks, leaving behind only a slight loss of energy potential.



VARIETIES OF ACUTE SCHIZOPHRENIC SHIFTS
(AFTER CONRAD)

FIG. 1.

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Type 3. The trema and apophanous phases are quickly passed through, and the apocalyptic phase is briefly touched on before the process subsides: the residual defect is very slight.

Type 4. The trema and the apophanous phase are rapidly passed through without the psychosis being recognized, but a marked loss of energy potential occurs, so that the residual state may make adaptation difficult. This corresponds to the type of schizophrenia called dementia simplex by some authors, but it can also be regarded as a variety of hebephrenia (Fish, 1957).

Type 5. The process does not pass beyond the trema, but there is a severe loss of energy potential. This is another way in which a chronic hebephrenic illness can occur.

Type 6. The process reaches the apophanous phase and is arrested there, so that a chronic paranoid schizophrenia occurs.

Type 7. The process reaches the apocalyptic phase and is arrested there, giving rise to a chronic catatonic clinical picture.

Conrad's theory links together all the phenomena which occur in acute schizophrenia by supposing that they are the result of a loosening of the coherence of psychological activity produced by a neurophysiological disorder. His concept of loss of energy potential in the defect state has nothing to do with Gestalt theory and is merely another term to explain the peculiar psychological disability in this condition. It is difficult to understand why Conrad has not attempted a Gestalt explanation of the defect states. Is it not possible, for example, that the so-called loss of energy potential can be explained as a partial loss of the normal integration of psychological functions or the mediating processes, while the paranoid and catatonic syndromes are abnormal re-integrations of the mediating processes?

This failure to deal with the chronic schizophrenic syndromes can be considered as a product of Gestalt theory itself which dogmatically insists that all psychological phenomena occur in figure ground relationships which are not learned, but innate and immutable. If this is so, then once the interfering schizophrenic neurophysiological process subsides, the previous figure ground relationships should reassert themselves. This would lead to the conclusion that in the paranoid and catatonic chronic clinical pictures the process was still active. However, the classical apophanous experiences do not occur in chronic paranoid schizophrenia.

B.—A Neurophysiological Theory of Schizophrenia

There is no doubt that Conrad's views are extremely stimulating, but as they stand it is difficult to use them as in a heuristic way. If we re-interpret his ideas in terms of D. O. Hebb's neuropsychological theories, I believe we have a much more useful set of ideas.

Hebb (1949) has summarized his basic theory as follows:

"Any frequently repeated particular stimulation will lead to a slow development of a 'cell assembly', a diffuse structure comprising of cells in the cortex and diencephalon (and also perhaps in the basal ganglia of the cerebrum), capable of acting briefly as a closed system, delivering facilitation to other such systems and usually having a specific motor facilitation. A series of such events constitutes a 'phase sequence'—the thought process. Each assembly action may be aroused by a preceding assembly, by a sensory event or—normally—by both. The central facilitation from one of these activities on the next is the

prototype of 'attention'. The theory proposes that in this central facilitation, and its varied relationship to sensory processes, lies the answer to an issue that is made inescapable by Humphrey's (1940) penetrating review of the problem of the direction of thought.

"The kind of cortical organization discussed in the preceding paragraph is what is regarded as essential to adult waking behaviour. It is proposed also that there is an alternate 'intrinsic' organization occurring in sleep and in infancy which consists of hypersynchrony in the firing of cortical cells. But besides these two forms of cortical organization there may be disorganization. It is assumed that the assembly depends completely on a very delicate timing which might be disturbed by metabolic changes as well as by sensory events which do not accord with the pre-existent central process. When this is transient, it is called emotional disturbance; when chronic, neurosis or psychosis."

Hebb's views can be partly represented diagrammatically as shown in Figure 2, which is a modification of a diagram by Hebb (1958). C is the central

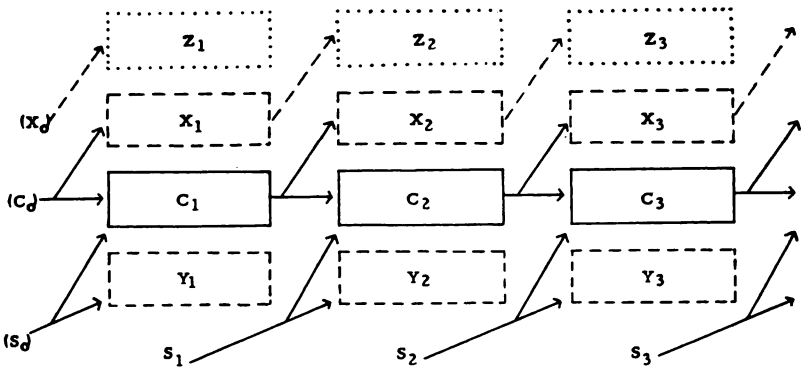

DIAGRAM OF THE SELECTIVE PROCESS IN THINKING (AFTER HEBB)

FIG. 2.

process consisting of simultaneous active assemblies at three successive moments, while S is the corresponding sensory input. X is an assembly subliminally excited by the central process, Y is an assembly subliminally excited by the sensory input, and Z is an assembly which would become subliminally excited if X were excited. In the following discussion these assemblies will be referred to as X, Y and Z assemblies. C consists of assemblies which are active because they receive excitation from both sources, but if one accepts Milner's modification of the cell assembly (Milner, 1957), then priming of X assemblies may lead to the central process deviating to X assemblies and to the sensory input having little influence on the central process for a short time. This diagram does not show the effect of the arousal or reticular system, but this system is, of course, partly responsible for the maintenance of the central process, since it facilitates the firing of cortical neurones.

Let us suppose that in schizophrenia there is an overactivity of the reticular system. This would lead to the X and Y assemblies being nearer their firing thresholds than usual. It could be argued that the Y assemblies would be more likely to fire than the X when the reticular overactivity is relatively mild, since the sensory input would tip the balance. It may also be that the sensory input at the cortical level is enhanced by reticular overactivity, although there is evidence that it is diminished at lowest levels by reticular stimulation.

Fuster de Carulla (Magoun, 1958) has shown that briefly presented visual clues are perceived better during reticular stimulation, while Lindsley and Griffith (Magoun, 1958) have found that the recovery cycle of the optic cortex was shortened by reticular stimulation. Thus it seems reasonable to assume that sensory events would, under these circumstances, acquire a much greater significance and would tend to produce disorganization of the phase sequence much more often than usual. Such a disorganization would, of course, express itself as anxiety. This anxiety, together with the increased significance of neutral events, would account for the general feeling of unease and suspicion which is so characteristic of the trema. This diversion of the central process would initially be sporadic and the central process would be mainly determined in the normal way by the previous cell assembly and the sensory input. However, as the intensity of the reticular overactivity increased, the central process would be diverted and disrupted by the sensory input much more frequently. When this disruption was occurring very often, then a delusional mood would occur. The uncanny, unpleasant nature of this symptom can be explained as due to the repeated disorganization of the central process by the sensory input and the feeling of some unknown significance produced by the increased impressiveness of all perceptions. In the end, some sensory event would dominate the central process for some time and the individual would experience a kind of fascination for this object. It would therefore acquire a new meaning for him, i.e. it would be a delusional perception.

Here we must pause to consider Matussek's concept of the coming into prominence of essential properties in delusional perception. Objects with marked essential properties have a greater tendency to evoke an emotional response, or, to use Hebb's terminology, to produce a cortical disorganization. Such objects would therefore be likely to form the subject of delusional perception. Since human beings have marked essential properties, the perception of others could lead to cortical disorganization and this could give rise to a lack of recognition, since the appropriate phase sequence would not be aroused, because another X or Z assembly takes over the train of thought. Conversely, a similarity of features may lead to a domination of the central process by the sensory input and the evocation of a memory image (phase sequence) of a relative.

However, once the reticular overactivity passed a certain point, sensory over-determination of the central process would not be the only expression of the disorder, since a central process, consisting entirely of Z cell assemblies, could then become active independent of the specific sensory input.

Any central process consisting of X or Z assemblies could either be the sole central process or a central process running parallel with, but interfering with the "normal" central process which is sensorily over-determined. Such a parallel process seems necessary in order to explain auditory hallucinations which are, of course, experienced as being foreign. It seems legitimate to assume that the sense of self is associated with the central process which is determined by the preceding phase sequences and the sensory input. Normally, the central process gives rise to behaviour leading to environmental modification, which, in turn, leads to a modification in the sensory input which then influences the central process and so on. Thus the interaction of the organism with the environment in which the self is experienced is expressed in the central process. In the trema the sensory over-determination of the central process gives rise to a paranoid attitude. The ability of the central process to detach itself from sensory input, but to be reorganized by impressive sensory input is lost. If a

parallel process were set up and constantly interfered with the central process, then these interferences would be experienced as foreign thoughts or voices, since the only continuous influence on the central process which has been previously experienced is a sensory one. Sudden ideas or "brain waves" can be considered as interference with the central process in normals, but they are, of course, brief and probably have a connection with the on-going central process, which is not conscious, but is expressed by the fact that the idea is not foreign. Since language plays such a great part in human thought, it is easy to understand that the interfering parallel process is often experienced as hallucinatory voices.

This parallel process need not be permanent, so that there could be periods when the sensorily over-determined central process alone was active, other periods when the parallel process was active on its own, and others when the two processes ran parallel. The phenomenon of hearing one's own thoughts spoken aloud could be understood as an activation of the phase assemblies for words which are activated to just below threshold by the reticular system and fully activated by the central process. They would thus occur just before the commencement of the parallel process.

This change from apophany of external space to apophany of internal space could be regarded as the point at which X assemblies become independently active. Thought insertion, and thought deprivation can be explained as due to the parallel process.

The acute catatonic phenomenon can be explained in one of two ways. Firstly, if the parallel process became entirely autonomous and the normal central process disappeared, then gross behavioural disorder would occur, since behaviour would not be influenced by sensory input. Some acute catatonic illnesses and oneirophrenia could be accounted for in this way. Again rapid switching between two parallel processes, an autonomous central process and a sensorily dominated central process could cause stupor, since neither central process could find motor expression before it was interrupted by the other. Finally, in young people, especially children, disordered thinking expresses itself as disordered behaviour. There is no doubt that catatonia is a disorder of the young. Thus X and Z assemblies in young people are much more likely to have a strong motor element, and it is reasonable to assume that maturity is associated with an increase in X assemblies which are not directly expressed in motor activity. Thus a parallel central process in a young person would be likely to cause non-goal-directed motor activity.

The maintenance of this parallel central process poses a question. Chance factors might lead to the establishment of such a process, but without continuous reinforcement it would tend to link up with the sensorily directed central process. It is possible that a parallel process might be kept going by reverbatory circuits involving the so-called cortical association areas and the subcortical nuclei. It has been shown that in the monkey there are corticofugal projections to the arousal system which are only second in importance to the inflow from the peripheral receptors (French, Hernández-Peón and Livingston, 1955). Segundo, Naquet and Buser (1955) found the cortical areas which were specially effective in producing arousal were the cingulate, orbital and lateral frontal regions, the para-central and para-occipital areas and, I would like to stress this, particularly the superior gyrus and pole of the temporal lobe. Supposing that this is also true of the human brain, then a parallel process taking place in one of these areas might produce even more reticular overactivity, but this would have no reinforcing effect on the sensorily dominated

central process, but would reinforce the parallel central process. It could well be that the ability to form a stable parallel process is the decisive factor in true schizophrenia. All the previous disorders are potentially reversible, but once the parallel central process is firmly established, the further progress of the illness is no longer dependent on the original reticular overactivity, but on this vicious circle which is now firmly established.

I think that it is possible to account for the symptoms of the different varieties of paranoid schizophrenia by postulating that the parallel process interferes to a varying degree with the normal central process and the parallel process involves different cortical areas.

I hope to deal with this problem in more detail in a later paper, and I would like now to consider the chronic schizophrenic states. If the reticular overactivity disappears before a stable parallel process has been established, then it is obvious that complete recovery could occur. The chronic states can be accounted for in three ways:

(*a*) The reticular overactivity does not decline. This would account for those paranoid illnesses in which delusions remain affect laden for years and which Leonhard has called affect laden paraphrenia (Fish, 1958; Leonhard, 1959).

(*b*) The reticular overactivity subsides, but the cortical vicious circle remains. This would account for many paranoid and catatonic clinical pictures with marked affective blunting.

(*c*) The reticular overactivity subsides, but it has caused a cortical reorganization which is less efficient than the premorbid one. Hebb's theory is based on the postulate that some structural change occurs at synapses which are repeatedly active. In the sensorily dominated central process produced by reticular overactivity all those cell assemblies and phase sequences which were well linked to perception would be reinforced, while the ones mediating more abstract thought would not be. Thus if the reticular overactivity were severe enough or prolonged enough, a reorganization of phase sequences could occur which would make abstract thought more difficult. Thus a mild, prolonged trema could cause a marked defect. This would account for the insidious development of schizophrenic defect in the eccentric and silly hebephrenias (Leonhard, 1959).

The course of the illness would obviously be determined by the duration and severity of the primary reticular overactivity and also by establishment of a stable parallel process. This theory would explain the atypical or schizoaffective psychoses as being due to a reticular overactivity without the formation of a stable central parallel process. It would also explain that strange condition paranoid depression in which there are marked persecutory delusions which go far beyond what can be understood as due to projected guilt feelings. These depressions are always associated with marked anxiety (Bumke, 1948; Janzarik, 1959) and attempts have been made in the past to explain the paranoid delusions psychologically as partly due to the severe anxiety. The theory presented here would suggest that the severe anxiety is associated with marked overactivity of the reticular system. This would produce a sensorily dominated central process which would lead to a paranoid attitude.

We must now come to the evidence in favour of reticular overactivity as the cause of schizophrenia. The most important supporting evidence is the fact that amphetamine overdosage in normal individuals will produce a psychosis which is clinically indistinguishable from paranoid schizophrenia. It is, of

course, well known that amphetamine produces an arousal response in the EEG in animals which is similar to that produced by reticular stimulation. Bradley and his associates (1957) have produced evidence to suggest that in animals amphetamine stimulates activity in the reticular system below the level of the midbrain. Now both sodium amylobarbitone and chlorpromazine will produce marked improvement in patients suffering from acute schizophrenic shifts. There is evidence (Bradley, 1957) that chlorpromazine may block the receptors related to afferent collaterals entering the reticular formation at brain stem and mesencephalic levels. Thus it is fair to assume that such general blocking of the afferents to the reticular system would diminish reticular activity. There is evidence that pentobarbitone and other quick-acting barbiturates depress activity within the reticular system. This would account for the effectiveness of sodium amytal in acute schizophrenic clinical pictures. The fact that it often has no effect on chronic patients would again support the theory presented here. One final point about amphetamine is that it has been shown to cause worsening of symptoms in schizophrenia.

An interesting clinical condition which supports our present theory is the paranoid psychosis in clear consciousness which occurs in some epileptics. Some of these patients show a relatively normal electroencephalogram when psychotic and an epileptic electroencephalogram when normal. Landolt (1958, 1960) has suggested that the normal electroencephalogram is a "forced normalization" produced by overactivity of the arousal system. This worker has also claimed to have found "forced normalization" in thirty-two non-catatonic schizophrenics. The electroencephalographic investigation of a series of fifty adolescent schizophrenics by Lescable, Lelord and Fardeau gives some support to the theory presented here. There was an immature record in 18 per cent. of their patients and in only one patient with such a record was the outcome unfavourable. In 74 per cent. the record was normal, but eighteen of the records in this group were rich in fast rhythms and five were hypermature. The outcome in these five patients was unfavourable.

The overactivity of the reticular system which has been suggested as the immediate cause of schizophrenia is, of course, a final common path, and there are many possible morbid processes which could give rise to such overactivity. The following are obvious possibilities:

1. A cortical focus could produce reticular overactivity by stimulating corticofugal pathways to the reticular system. The electroencephalographic evidence of focal activity might then be masked by the effect of the reticular system on the cerebral cortex. This would be the "forced normalization" of Landolt, and could account for the schizophrenic clinical pictures seen in epilepsy, brain injury and organic hallucinosis.
2. An irritative lesion of the reticular due to physical trauma, toxins or infection could produce overactivity of the reticular system. This would account for the schizophrenic clinical pictures seen in post-encephalitic states and possibly some post-traumatic schizophrenias.
3. An inherited biochemical disorder of the reticular system, such as a lack of mono-amino oxidase or some other enzyme could cause reticular overactivity. Like other enzymatic disorders, e.g. adrenal virilism, this need not manifest itself until puberty and might come to light because of physiological overactivity caused by an adolescent crisis.
4. An excess of adrenaline, nor-adrenaline, or similar substances produced by adrenal disorder could affect the reticular system. Such adrenal dysfunction

could be due to primary disease of the adrenal or to a hypothalamic or pituitary disorder, which in turn might be made worse by the adrenal disorder. Thus schizophrenia could be due to a neuroendocrine vicious circle.

Many other combinations of these disorders and other disorders could be postulated as causing the overactivity of the reticular system.

Summary

It is suggested that the trema and early apophanous stage of acute schizophrenia can be explained as due to overactivity of the reticular system producing an undue diversion and disruption of the central process by the sensory input. Internal apophany and catatonic symptoms are explained as due to disruption of the central process with the formation of short-lived parallel central processes. Chronic schizophrenic process clinical pictures are considered to be due to a permanent parallel process which is dependent on a cortical reticular reverberatory system, while defect states without active symptoms are regarded as due to a reorganization of cell assemblies produced by the acute process. Pharmacological and electroencephalographic evidence in support of this theory is discussed. Possible causes of overactivity of the reticular system are briefly outlined.

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