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Perceptual Defect and Role Handicap: Missing Links in Explaining the Aetiology of Schizophrenia

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Introduction

The immense literature on schizophrenia abounds in discoveries of single associations between the symptoms of the disease and certain other factors. Braithwaite (1960) says in speaking about "science like psychology and the social sciences which make great use of tendency statements . . . that to assert an isolated tendency statement is to say very little". Such isolated statements in themselves are not explanations. A theory, on the other hand, interprets information; it explains.

In this paper a theory of the aetiology of schizophrenia is proposed. The aim is to link the main experimental data and observations concerning the disease, providing, in Beck's (1962) words "a single scheme of interpretation for a whole group of apparently disconnected facts".

The procedure will be, first of all, to assess the main types of investigation concerning schizophrenia. This assessment will be presented in tabular form, so that the data can be readily summarized and the relative strengths of the various associations be compared by the reader. Secondly, evidence will be cited in support of defective perception as a central factor in schizophrenia. Thirdly, taking defective perception as a starting-point, and with the aid of a diagram, a complex social interaction process will be outlined which organizes the previously summarized data into an explanatory pattern. Fourth, the proposed theory will be stated in summary form. Fifth, a preliminary test of the theory will be made by checking whether it can explain the specific symptoms and characteristics of the disease.

I. Assessing the Main Types of Investigations

In the accompanying table representative reports of the main types of investigations into the nature and aetiology of schizophrenia have been brought together. An attempt to assess them is made by listing the evidence for and against the association which a particular investigation claims to have discovered. Beyond this the investigator's report as a theory of aetiology is assessed by asking of each the following questions:

(1) Does the report demonstrate one or more factors with which the disease is typically associated?

(2) Does it postulate one or more mechanisms that can account for the observed association(s) and so begin to explain the formation of symptoms?

(3) Is this supposed mechanism sufficiently specific to make possible tests of its working?

II. Defective Perception and Schizophrenia

On the basis of the available evidence, then, the association between defective perception and schizophrenia emerges as the strongest, most consistent, most generally agreed-upon association. This is hardly surprising, since perceptual disorders have long been thought of as a symptom of the illness. Some of the investigators already cited have established connections with other variables, such as severity of thought disorder, Weckowicz and Blewett (1959), genetic factor and biochemical abnormality, Huxley *et al.* (1964), and anxiety, McReynolds (1960). McGhie and Chapman

TABLE I

Factor seen related to schizophrenia	Investigators	Evidence for relationship	Evidence against relationship	Assessment as theory		
				1	2	3
1. Genetic factor of some kind, unspecified.	Kallman (1946) Book (1960) (review article) Jackson (1960) (review article)	The closer the blood relationship to a schizophrenic, the more likely it is that a person will develop schizophrenia *Concordance*: Relationship: % concordant: Parents .. .. 10% Siblings .. .. 12% Children: One S. parent 16% Two S. parents 68% Twin studies: Dizygotic .. 13% Monozygotic .. 85% Not separated 91% Separated .. 77%	Kallman's methods are called into question—the problem of adequate diagnosis is crucial. Evidence from epidemiological studies tends to undermine genetic evidence. Association could be the result of environment.	Yes	No	No
2. Genetically determined error in biochemical function interferes with the mechanism of perception.	Huxley, Mayr, Osmond, and Hoffer (1964)	About 5% of people have this genetic factor, hence are "schizoprone". A quarter of these develop Schizophrenia in response to faulty perception plus stress; the nature of the stress is not specified.		Yes	No	Yes
Family dynamics, interaction, communication, psychological stress on child.	*Pro:* Lidz and Fleck (1960) (review article) Bowen *et al.* (1957) Boatman and Szurek (1960) *Con:* Clausen and Kohn (1960) Prout and White (1956) Schofield and Balian. (1959)	Schizophrenics are more likely than others to have weak fathers and/or "rejecting, inconsistent, over protective, domineering, overtly loving but covertly hostile" mothers. "Schizophrenogenic" mother who places child in "double bind" situation—not possible to respond correctly—either response punished	Type of maternal behaviour is related to *social class* more than to schizophrenia. 90 siblings of 60 schizophrenics experienced as much mental trauma but schizophrenics reacted differently. Parental deprivation as causal not substantiated. Histories of non-schizophrenic controls usually *were* disturbing—more trauma experienced than by schizophrenics.	?	No	?

Factor seen related to schizophrenia	Investigators	Evidence for relationship	Evidence against relationship	Assessment as theory		
				1	2	3
Extreme physical and psychological trauma.	Eitinger (1961) Bettelheim (1960)	Concentration camp victims with no hereditary taint developed schizophrenia after starvation, depersonalization, loss of family, hopelessness.	Clinical study only—no information on victims *not* schizophrenic.	Yes	Yes	No
	Rogler and Hollingshead (1965)	Schizophrenics in Puerto Rican slums, in contrast to matched sample of non-schizophrenics, had experienced a "critical year" of personal trauma, physical and psychological, prior to onset of illness.	See above for more typical environment. These cases may be explained this way, but few schizophrenics have experienced such extremes of deprivation; death of loved ones a universal human experience.	Yes	Yes	No
Biochemical abnormality.	Weckowicz (1961) (review article) Kety (1960) (review article)	Hundreds of studies have shown differences in body chemistry between schizophrenics and others. Emotional reactions and chemical imbalance tend to perpetuate and exacerbate each other.	Many of these differences have been shown to be artifacts of hospitalization, and can be eliminated by changes in diet and/or activity level. Inconclusive findings with respect to metabolism of epinephrine, ceruloplasmin, serotonin, taraxein.	Yes	No	No
Prenatal environment factors—adverse intra-uterine conditions.	Pasamanick, Knobloch and Lilienfeld (1956) Sontag (1960) (review article)	The incidence of psychosis is higher among children whose births were difficult and/or when there were abnormalities during pregnancy. Evidence from epidemiological studies. Numerous animal studies relate enduring behaviour disorders to prenatal factors such as poor nutrition, altered oxygen supply, emotional disturbance of mother.		Yes	No	No
Prenatal environmental factors.	Boatman and Szurek (1960)	Strain for parents just before birth of child is related to onset and exacerbation of child's disorder—clinical study.	No information about strains experienced by parents of other children.	Yes	Yes	No

Factor seen related to schizophrenia	Investigators	Evidence for relationship	Evidence against relationship	Assessment as theory		
				1	2	3
Perceptual deficit, defect, disorder, distortion.	Weckowicz and Blewett (1959)	Degree of disturbance in size and distance constancy is correlated with severity of thought disorder in schizophrenics.		Yes	No	No
	Shakow (1963) Weckowicz, Sommer and Hall (1960)	Slowness of establishing set. Slowness of response. Slowness of learning. Unrealistic perception. Variability much greater.		Yes	No	No
	Buss and Lang (1965) Lang and Buss (1965) (review articles)	Underlying the multiple psychological deficits of schizophrenics is a basic defect in sensori-motor functioning.		Yes	Yes	No
	Lane and Albee (1965)	Pre-morbid group IQs of schizophrenics at grade 2, 6, and 8 level, when compared with those of siblings, show up as consistently lower, showing that intellectual deficit *precedes* onset of illness by *years*.		Yes	No	No
	Kephart (1960) Strauss and Lehtinen (1947) Bender (1956) Kirk— clinical observations	Symptoms and psychological test protocols of schizophrenics resemble those of children with learning disabilities due to perceptual defects. This is true of at least the following tests: Wechsler Intelligence Scales. Bender Visual-Motor Gestalt Test. Doman-Delacato Developmental Profile.		Yes	Not as yet	Not as yet

(1961), starting out from an investigation which was psychoanalytic in orientation, are led by their observations to reject the proposal that early schizophrenic symptoms are defensive activities related to unconscious conflicts and to suggest that the basic pathological breakdown is particularly to be found in the process of perception. Fish (1961) reaches the conclusion that early phases of acute schizophrenia are due to "overactivity of the reticular system producing an undue diversion and disruption of central processes by sensory input". Thus he sees anxiety as a result of disturbed perception and the resulting disruption of thought processes. Hoffer and Osmond (1963) report that certain schizophrenic symptoms are associated with measurable aspects of visual perception, specifically that schizophrenic patients were less able to decide whether or not an investigator was looking into their eyes than were non-schizophrenics. Most of these investigators have not, so far, been able to state the complex interrelated mechanisms which could explain the development of the complex symptomatology of the disease. Chapman (1966) gives evidence which relates disturbances in perception to the immobility of the catatonic, to cognitive disturbances, to visual imagery, and to the development of delusions and hallucinations, illustrating his ideas with material from interviews with acute schizophrenics. He suggests that the time is ripe for finding a way of integrating the vast number of disparate findings into a theory rather than looking for some major new aetiological factor.

III. Defective Perception and Social Interaction

The accompanying diagram, like the line of thought which is to follow, starts out with the postulation of an unrecognized perceptual defect in a child. It is likely that the defect is relatively slight, since gross defects would not remain unrecognized. The origin of this perceptual defect could be any one of the following:

(1) A genetic factor, possibly a biochemical abnormality.

(2) Adverse prenatal conditions.

(3) Brain injury at birth or at some later date —or some combination of two or more of these.

(1) If one assumes that perceptual defect in one or more sensory modalities exists, one can readily deduce some of the effects of such a defect on the growing child. The child can be affected by the fact that he has poor or distorted perception in several ways, namely:

(*a*) He will become aware that he cannot do something that others of his age and intelligence *can* do, such as read or spell or throw accurately or discriminate between similar sounds.

(*b*) When he listens to others talk of what they perceive, he will not entirely understand, because he does not perceive things as they do, his disability perhaps including poor perception of language itself.

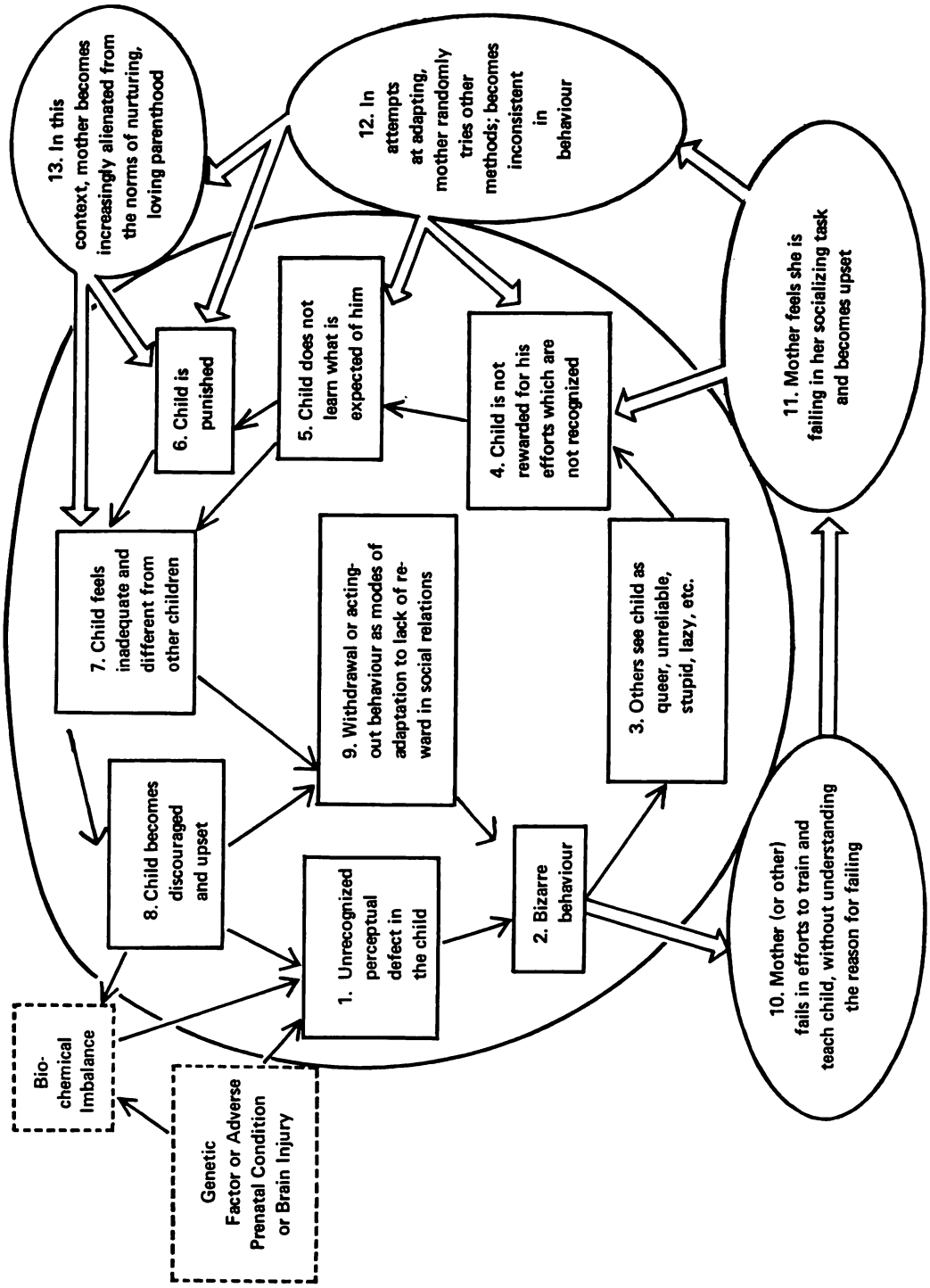
(*c*) When he acts on the basis of his deficient perceptions, he will not find consensus, i.e. his behaviour will not be understood by others, in short, will be seen as bizarre.

(2) The perceptual defect, then, results in bizarre behaviour from the point of view of adults and of other children, who are not aware that the child is not perceiving things as they do.

(3) Because he is behaving in a bizarre manner and not learning normally, others tend to regard the child as queer and unreliable. When he talks of what he perceives, people will tell him he is mistaken, or, more bluntly, stupid. If he is unable to judge distances, so that he trips over and bumps into obstacles, he will be thought of as clumsy. When he is unable to keep his attention focused long enough to complete a task, people will be likely to interpret this as laziness.

(4) Because his efforts are not recognized as such, the child is not rewarded for them, nor is he rewarded by his own feelings of accomplishment, since many of his attempts are unsuccessful ones.

(5) The net result of the perceptual handicap, then, is that the child does not learn the things he is expected to learn, both because the handicap in itself makes learning difficult (and the learning of some of the more complex



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skills, such as reading, extremely taxing), but also the child's best efforts may often go unrecognized and unrewarded.

(6) In addition, if he and others have no knowledge of his perceptual defect, he will, in effect, be punished for things he cannot understand nor avoid doing. Also, the child will be punished for failing at tasks he cannot master despite consistent effort.

(7) Even if he is not punished directly, he will be likely to feel himself to be somehow different from other people, and less adequate than other children.

(8) The child's conscious and/or unconscious reaction of discouragement and emotional upset to the unrecognized fact of his perceptual disability and his own sense of his difference from others can take various forms, but it is bound to be present.

(9) Because social relations are unrewarding to him, the child may withdraw from others and live chiefly in a fantasy world in which his own perceptions are not tested against the perceptions of others and hence would be likely to be less disturbing, or not disturbing at all. This is the autistic reaction. Alternatively, the child's emotional reaction to his handicap may be one of violent aggressive, uncontrolled behaviour, that of a person who tries to force his own perceptions on other people. This is the acting-out reaction.

This inevitable emotional reaction to the experience of impaired perception is likely to compound the feeling of difference in at least two ways:

(*a*) In children with perceptual defects, these defects are much more pronounced when the child is upset, angry, or tired than when he is rested, happy and relaxed. Hence, the emotional reaction will tend to increase the perceptual handicap.

(*b*) The emotional reactions of withdrawal, aggression, or lack of control will often be construed as infantile or bizarre behaviour unacceptable to others, possibly leading to rejection of the child or to punishment which he may feel as rejection.

To sum up the argument so far: the perceptual defects, the reactions of other people, and the child's emotional reactions to both (including, of course, his physiological reactions) appear to constitute a vicious circle which will tend to spiral downwards unless the defect is somehow corrected or compensated for and the emotional reactions are neutralized or reversed. Chapman (1966) has described this process to some extent, and illustrates it with quotations from interviews with young schizophrenics.

The child whose perceptions are thus not only deficient or distorted, but are made unstable by his emotional reactions, will have difficulty in identifying himself with anyone, since he can have no stable models. This instability of models is, of course, much greater in the case of the child with a "schizophrenogenic" mother. This is true because her behaviour toward the child is actually unstable and unreliable. According to the proposed theory, a child with such a mother, if his own perceptions were normal, might become neurotic, but not schizophrenic. He would, in time, find other models with whom to identify, and could come to realize that his mother, not he, is "different". But a child with faulty perception, compounded by his own and others' reactions to it—for whom nothing and no one in the external world of reality is stable—will have no basis for forming a sturdy ego. His natural tendency would be to regard his own inner bodily needs and his fantasies built around these needs as the only stable realities of his life. Hence, he would, in schizophrenic fashion, tend to respond mainly to these inner needs and fantasies, rather than to external reality.

The next link in the chain can be best understood in terms of Kirk's (1964*a*, *b*) concept of "role handicap".

The notion of "role handicap" helps to clarify how the kind of interpersonal communication, or more accurately, miscommunication which is characteristic of schizophrenics and their families, develops. Roles are learned patterns of interpersonal conduct governed by particular norms. In that sense they involve our learning that certain responses are generally to be expected in particular social situations. Let us now think of an individual who has learned the proper role for a particular situation; but when

he finds himself in the actual situation it is not at all what his role training has led him to expect. The behaviour of certain others in that situation is sharply different from his anticipations of them. Such a discrepancy between the learned and thus expected situation and the encounter with the actually experienced one constitutes a constraint for normative role performance and such a structural constraint has been called "role handicap".

If the role-handicapped person can gain the key to the meaning of the actual behaviour of others, he may be able to adjust his behaviour accordingly, and thus overcome his role handicap and carry out his relations in an altered but effective manner. But if the role-handicapped person does not gain understanding of the nature of the changed situation, he is likely to act in ways that are inappropriate and that are likely to alienate him from the relational norms that were supposed to govern his role. This concept of role handicap can, when considered in combination with a learning handicap in the child, explain the genesis of the syndrome of behaviour on the part of the mother which has been called "schizophrenogenic".

(10) To see how this kind of behaviour on the part of the mother could be generated toward a particular child, a perceptually handicapped child, let us again follow the sequence illustrated in our diagram. Since adequate role performance depends in part on getting adequate responses from significant others, not only does the child require adequate responses from the mother; the mother, if she is to feel adequate in her role as a nurturing, loving parent and teacher, requires adequate responses from the child. But the bizarre behaviour of the child means to the mother that she is failing in her efforts to teach and train the child without understanding why she is failing.

(11) The mother of such a child, feeling that she is failing in her task, becomes upset; she feels she is failing, not only because of the child's bizarre behaviour, but because she becomes aware that others see the child as queer, stupid, lazy, etc.

(12) She thus feels handicapped in her role as mother, and will be likely to try to adapt herself in at least two ways. Because her child responds differently from others to her usual methods of discipline and training, her efforts at using these methods are likely to give way to a random succession of methods, in the hope of finding a successful one. Since she does not have the key to understanding what is wrong, these random attempts at adaptation compound the child's difficulty by forcing him to cope not only with his own perceptual defect and hence learning handicap, but also with the inconsistency of his mother's approach, which fails to be rewarding, and may be actually punishing.

(13) As each new method tried falls short of success, the mother, feeling more and more inadequate, is likely to begin blaming the child, if she hasn't already done so at an earlier stage. Her hostility, coupled with her feeling of guilt (because of both her failure and her hostility) would be likely to make the child feel even less secure and his performance even less adequate than it would be without this emotional strain, so that again a vicious cycle is in operation. In this cycle, the child's withdrawn and/or hostile behaviour serves to perpetuate the mother's inconsistent, rejecting, "schizophrenogenic" behaviour, which in turn exacerbates the child's tendency to bizarre behaviour, and tends to alienate the mother from the norms of nurturing, loving, parental behaviour toward this child who is so unrewarding to her. Thus the mother develops an ambivalent attitude toward the child, with love and rejection felt simultaneously.

The adult who is closest to the child (usually the mother) will suffer more of a handicap in her role toward him, but such significant others as fathers, teachers, and siblings will also be likely to meet with responses based on the child's faulty perception, and they and the child be prone to reduce their interaction with each other, thus reducing their mutual frustration. Although the other people will not suffer much from this reduced contact because they experience other, more normal relationships, the child himself will become increasingly isolated and withdrawn as social interaction tends to be continually frustrating for him.

In recent years, the author has had occasion to take several histories in which this kind of complex vicious circle process was clearly visible.

Even more significant, a reversal of the vicious circle has been observed when perceptual defect was diagnosed and appropriate perceptual training begun. Parents of perceptually handicapped children experience a profound relief and a corresponding shift toward a positive desire to help their child overcome his handicap, once they understand what the child's problem is. In such cases, the key to overcoming the role handicap lies in the parents' knowledge of the underlying defect, so that the social milieu, instead of being rejecting and hostile, becomes positive, accepting and loving, working toward the child's better functioning rather than exacerbating the difficulty.

A case history may serve to illustrate this reversal of the vicious cycle of parental and child behaviour.

J., an 8-year-old boy, the son of a university teacher, was the younger of two children in the family. For two or three years, he had been gradually withdrawing from contact with family members, and within the six months prior to testing, the withdrawal had extended to withdrawal from other children, both at school and in the neighbourhood. Despite a bright–normal IQ, J.'s school work was poor, and he spent most of his time in his room with the door closed. His parents were having marital problems, and were told by their doctor that they were rejecting their son emotionally and that this withdrawal was a reaction to his knowledge of their rejection of him.

Psychological testing showed that J. had many signs of minimal cerebral dysfunction, including deficiencies in both auditory and visual perception, as well as fluctuating attention, hyperactivity, and inadequate language development. (His Verbal IQ was 22 points lower than his Performance IQ.) When these difficulties and the handicap they meant in the school situation were explained to his parents, their previous ambivalent attitudes toward him changed to loving concern. They undertook to launch J. on a vigorous programme of exercises and training designed to decrease these handicaps (Delacato, 1963).

Over a period of ten months, J. became actively involved in both family and school activities, and developed a lively, friendly personality. His Verbal IQ increased by 15 points, and his test protocol no longer showed the evidences of perceptual handicap. These improvements took place and the gains have been maintained after treatment was stopped, in spite of the fact that the emotional stress in the home became worse, resulting in the separation of J.'s parents.

The downward spiral of reactions to perceptual defect on the part of the child and others is not likely to occur to any great extent until after the child is no longer an infant. One reason for this is that satisfactory performance by a baby is relatively simple and does not require as accurate and stable perception as is necessary for the more skilled performances required of the older child or adolescent. Also, everyone expects that infants will be infantile in their emotional responses. But by the time the child is 3 or 4 years old and has learned to walk and talk, he is expected to begin behaving as others do, and is punished in subtle or not-so-subtle ways if he does not conform to these expectations.

In order to construct a theory of the aetiology of schizophrenia, it was only necessary to arrange into a new pattern the data summarized previously in this paper. The first step in forming the new pattern was to think of the perceptual deficiency associated with schizophrenia as a predisposing factor, rather than simply as a symptom of the disease. It predisposes in the sense that it constitutes a learning handicap for the child, and that learning handicap leads to a role handicap for the mother. The interaction of these two handicap patterns was shown to lead to alienation on the part of the mother and to withdrawal or acting-out behaviour on the part of the child. The theory of the aetiology of schizophrenia can now be formulated.

IV. Statement of Theory

A deficiency in the processes of perception resulting from a central nervous system malfunction is necessary to produce schizophrenia. The origin of the defect may be either genetic or the result of injury to the brain, and the cause of the malfunction may be either physical or chemical. Given such a perceptual deficiency, the nature of the individual's social environment and his interaction with it determines whether or not schizophrenic symptomatology will develop.

V. Preliminary Test of Theory

The theory will now be tested in a preliminary way by examining it in the light of the specific symptoms and characteristics of the disease, which a theory of aetiology should be capable of explaining.

Hallucinations

An hallucination, by definition, is a perception (in one or more of the sense modalities) which would not be perceived by other people similarly situated. There is a type of hallucination which is common in very young children, who have not yet learned to distinguish between external reality and their own fantasies. In testing his fantasies against external reality as perceived by himself and by others, the child with normal perception finds that these fantasies are the products of his imagination; thus he can learn to distinguish between them and external reality. But a child who is perceptually handicapped regularly sees (or hears, tastes, smells, touches) things and perceives them in ways that differ from the perceptions of others, and differ in unpredictable ways. Such a child has the basis of hallucination, as do all young children, but without the normal child's adequate equipment for reality testing. Because of his perceptual handicap, he is unable to perceive external reality as others do; thus he has no built-in basis for learning the difference between this reality and his own fantasies. Thus, he would be likely to more frequently confuse his fantasies with reality, i.e. hallucinate. (See Chapman (1966) for observations of this kind of process in schizophrenic patients.)

Withdrawal

In the kind of situation just described, the reality sense of other people will be likely to disturb, for the child with faulty perception, the reality of his own perceptions, thus leading to withdrawal from others as the best defence available to him. To illustrate this idea, consider the situation of a dyslexic child who has a confusion in laterality. He may see the word "on" and perceive it as "no" sometimes and "on" at other times. He will be told that it is "on" no matter how he happens to be perceiving it at the time. Also, he may see "no" and perceive it as "on" in which case he will also be told he is wrong. In this situation, his only escape from his confusion and from the ridicule of others would be to retreat, and withdraw entirely from the reading situation.

The idea of withdrawal as a defence reaction to impaired perception is supported by evidence from a recent experiment performed by Lawes (1963). This experiment demonstrated that in normal adults under the sensory and social stimulation of an ordinary conversation the symptoms produced by phencyclidine are similar to those of early schizophrenia—such symptoms as thought disorder, vertigo, disorders of perception and attention, affective reactions, and body image disturbances being common. But under conditions of social isolation and sensory deprivation, the administration of the drug to the same normal subjects produced either no such symptoms, or the symptoms were much reduced in intensity. This finding lends support to the view that withdrawal from other people can reduce or eliminate the disturbance experienced by a person with defective perception. Chapman (1966) describes the function of withdrawal as a defence against disturbed perception, quoting from recordings of interviews with young schizophrenics.

Thought Disorder

Perceptual defect readily explains the source of this most definitive of the symptoms of schizophrenia, since the ability to form associations and concepts, to abstract, and to think logically are each based on accurate and stable perception. For example, if there is a disturbance in the perception of size and distance, how can one firmly understand such abstract ideas as "tall", "far", or "little"? Or how can one be sure that 50 feet is one-half of 100 feet? Bizarre associations and misinterpretations can easily be explained by poor auditory perception. There are many words which differ from each other only in that they have a single similar sound that differs, while the rest of the word is the same. Suppose that a person cannot readily distinguish that slightly different sound. He may understand "sought" or "fought", when someone says "thought", or "fence" when what is meant is "sense". One can readily understand how confusing the most ordinary statements could become to a person so handicapped and how difficult he would find it to follow just a social conversation, let along a logical sequence of thought. Lawson, McGhie and Chapman

(1964) have collected both clinical and experimental data which suggests that faulty perception of speech by schizophrenic patients may be due to their relative inability to perceive a sentence as an organized meaningful whole, even when the individual words are perceived accurately.

Paranoid Delusions

If a person perceives things differently from other people he will, as was shown earlier, be punished through ridicule and in other ways for errors of which he cannot be aware. This would be likely to lead him to believe that other people are against him and/or to believe that he is a chosen emissary of God or some supernatural power and thus that he is different from and superior to the ordinary people with whom he lives. In either case, he would be protected by his delusions from the reality sense of other people, with the net result that his deficient or distorted perceptions would be less disturbing to him.

Catatonia

Various investigators, e.g. Kephart (1960), have demonstrated that visual and tactual perception depends upon movement. Therefore it seems reasonable to suppose that the immobility of the catatonic patient serves as a defence against distorted perception. This immobility can be thought of as a kind of self-induced sensory and social deprivation which can make perceptual and body-image distortions less disturbing. Chapman (1967) not only describes this process, but quotes young schizophrenics who have experienced it and tell precisely what it feels like.

Affective Flattening

The flattening of affect characteristic of schizophrenia can be understood as a conditioned reaction. Since emotional arousal increases perceptual distortion, this increased distortion and its concomitant disturbance would be avoided if emotional arousal did not occur. Therefore, a person who has distorted perception would be likely to become conditioned to avoid becoming emotionally aroused. This avoidance behaviour would be powerfully reinforced by the fact that the person would thereby avoid the disturbance which increased distortion precipitates. The withdrawal behaviour described above would go along with and reciprocally reinforce avoidance of emotional arousal, with the end result of a progressive flattening of affect.

Age of Onset

The fact that the onset of schizophrenia typically occurs in late adolescence or early adulthood is congruent with the hypothesis that perceptual defect is the crucial factor in its aetiology. For it is only as the individual is leaving childhood and entering adulthood that society demands insistently that he leave the world of fantasy behind him and conform consistently to the demands of external reality. A person who has been handicapped by defective perception and his own emotional reactions to it may get along well enough until the demands of external reality as they exist in his social group become insistent. At that point, the weak ego becomes overwhelmed, and a catastrophic downward spiral of emotional reaction, disturbed body chemistry, further impaired perception, thought disorder, and exacerbated emotional reaction takes place. Such a downward spiral corresponds to the onset of schizophrenia as a recognizable disorder; in acute cases, it corresponds to "decompensation".

Acute v. Process Schizophrenia

Acute schizophrenia, with sudden onset and more hopeful prognosis, could be explained on the basis of perceptual defect occurring in adulthood as a result of either a delayed-action gene (as in Huntington's chorea) or of organic damage as a result of brain injury, or as a result of biochemical imbalance due to severe malnutrition as in slums and concentration camps. In this case the individual may have had normal perception during his childhood and hence have been able to form a relatively sturdy ego and a basically sound reality sense. Then the relatively sudden disruption of perception would be frightening and upsetting to him and lead to acute and sudden symptom formation. But with a basically sound ego structure, he

would be able to compensate and overcome his symptoms faster and more completely than would be possible in the case of a person whose whole ego structure is weak because he has never *had* adequate perception. This latter kind of individual would develop process schizophrenia, which is slow and insidious in onset. His reality sense would be grossly underdeveloped and his ego without any stable foundation. He would, therefore, have little chance of recovery, though his symptoms might appear to be milder. In this connection, we note that psychiatrists and others who have experimented with LSD and other psychotomimetic drugs report experiencing both perceptual disturbances and psychotic symptoms such as acute anxiety and disrupted thought processes. This has enabled them to understand the world of the psychotic better than before, but has not permanently disrupted their normal ego functioning.

Schizotype

The hypothesis of perceptual defect interacting with social environment also explains Meehl's (1962) "Schizotype", or schizoid personality. A person who has a perceptual defect in one modality could be sufficiently handicapped by it to develop a schizoid personality, yet might be able to compensate with accurate perceptions in the other modalities to the extent that reality testing is impaired, but not destroyed. Also, a child whose mother is able to remain consistent, loving, and stable in her behaviour toward the child in spite of the fact that she is frequently punished by his lack of adequate response, would have a better chance of compensating for a perceptual defect than one with an unstable, inconsistent, rejecting, "schizophrenogenic" kind of mother. Finally, if the modality in which the perceptual defect occurs is not crucial to adequacy in the culture to which the person belongs, it will handicap him less than if it *is* crucial in his culture. For example, accurate perception of distance would be crucial in a hunting culture, but not in ours, where the perception of laterality is crucial because it is basic to reading.

The McGill cross-cultural study on the symptomatology of schizophrenia (Murphy, Wittkower, Fried, and Ellenberger, 1963) has gathered data which suggest that, while the visual and auditory modalities are important in all cultures, the tactile and olfactory modalities may play an important role in schizophrenia in some of the non-literature cultures, notably in Africa. In these cultures, tactile and olfactory hallucinations are frequent symptoms in schizophrenia, while such symptoms are rarely reported in our culture.

VI. Summary

This theory postulates that a child's unrecognized perceptual defect leads to a learning handicap for the child, and to a role handicap for the child's parent(s). A complex interaction between the two handicaps has been shown to create a vicious circle which tends to perpetuate and exacerbate the problems of both child and parent and, in extreme cases, leads to the development of schizophrenic symptoms on the part of the child and to alienation on the part of the parent(s). Ways in which this process can explain the development of the specific symptoms and characteristics of the disease have been outlined and described.

The theory is congruent with the bulk of the separate findings of many investigators. It postulates mechanisms which account for the observed associations and explain symptom formation. The postulated defect is measurable and its working can be observed, so that tests of the theory are possible. Specific researches designed to test the theory are now in the planning stage.

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