

# SCHIZOTAXIA, SCHIZOTYPY, SCHIZOPHRENIA<sup>1</sup>

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IN the course of the last decade, while spending several thousand hours in the practice of intensive psychotherapy, I have treated—sometimes unknowingly except in retrospect—a considerable number of schizoid and schizophrenic patients. Like all clinicians, I have formed some theoretical opinions as a result of these experiences. While I have not until recently begun any systematic research efforts on this baffling disorder, I felt that to share with you some of my thoughts, based though they are upon clinical impressions in the context of selected research by others, might be an acceptable use of this occasion.

Let me begin by putting a question which I find is almost never answered correctly by our clinical students on PhD orals, and the answer to which they seem to dislike when it is offered. Suppose that you were required to write down a procedure for selecting an individual from the population who would be diagnosed as schizophrenic by a psychiatric staff; you have to wager \$1,000 on being right; you may not include in your selection procedure any behavioral fact, such as a symptom or trait, manifested by the individual. What would you write down? So far as I have been able to ascertain, there is only one thing you could write down that would give you a better than even chance of winning such a bet—namely, “Find an individual X who has a schizophrenic identical twin.” Admittedly, there are many other facts which would raise your odds somewhat above the low base rate of schizophrenia. You might, for example, identify X by first finding mothers who have certain unhealthy child-rearing attitudes; you might enter a subpopulation defined jointly by such demographic variables as age, size of community, religion, ethnic background, or social class. But these would leave you with a pretty unfair wager, as would the rule, “Find an X who has a fraternal twin, of the same sex, diagnosed as

schizophrenic” (Fuller & Thompson, 1960, pp. 272–283; Stern, 1960, pp. 581–584).

Now the twin studies leave a good deal to be desired methodologically (Rosenthal, in press); but there seems to be a kind of “double standard of methodological morals” in our profession, in that we place a good deal of faith in our knowledge of schizophrenic dynamics, and we make theoretical inferences about social learning factors from the establishment of group trends which may be statistically significant and replicable although of small or moderate size; but when we come to the genetic studies, our standards of rigor suddenly increase. I would argue that the concordance rates in the twin studies need not be accepted uncritically as highly precise parameter estimates in order for us to say that their magnitudes represent the most important piece of etiological information we possess about schizophrenia.

It is worthwhile, I think, to pause here over a question in the sociology of knowledge, namely, why do psychologists exhibit an aversive response to the twin data? I have no wish to argue *ad hominem* here—I raise this question in a constructive and irenic spirit, because I think that a substantive confusion often lies at the bottom of this resistance, and one which can be easily dispelled. Everybody readily assents to such vague dicta as “heredity and environment interact,” “there need be no conflict between organic and functional concepts,” “we always deal with the total organism,” etc. But it almost seems that clinicians do not fully believe these principles in any concrete sense, because they show signs of thinking that *if* a genetic basis were found for schizophrenia, the psychodynamics of the disorder (especially in relation to intrafamilial social learnings) would be somehow negated or, at least, greatly demoted in importance. To what extent, if at all, is this true?

Here we run into some widespread misconceptions as to what is meant by *specific etiology* in nonpsychiatric medicine. By postulating a “specific etiology” one does *not* imply any of the following:

<sup>1</sup> Address of the President to the seventieth Annual Convention of the American Psychological Association, St. Louis, September 2, 1962.

1. The etiological factor always, or even usually, produces clinical illness.

2. If illness occurs, the particular form and content of symptoms is derivable by reference to the specific etiology alone.

3. The course of the illness can be materially influenced only by procedures directed against the specific etiology.

4. All persons who share the specific etiology will have closely similar histories, symptoms, and course.

5. The largest single contributor to symptom variance is the specific etiology.

In medicine, not one of these is part of the concept of specific etiology, yet they are repeatedly invoked as arguments against a genetic interpretation of schizophrenia. I am not trying to impose the causal model of medicine by analogy; I merely wish to emphasize that *if* one postulates a genetic mutation as the specific etiology of schizophrenia, he is not thereby committed to any of the above as implications. Consequently such familiar objections as, "Schizophrenics differ widely from one another" or "Many schizophrenics can be helped by purely psychological methods" should not disturb one who opts for a genetic hypothesis. In medicine, the concept of specific etiology means the *sine qua non*—the causal condition which is necessary, but not sufficient, for the disorder to occur. A genetic theory of schizophrenia would, in this sense, be stronger than that of "one contributor to variance"; but weaker than that of "largest contributor to variance." In analysis of variance terms, it means an interaction effect such that no other variables can exert a main effect when the specific etiology is lacking.

Now it goes without saying that "clinical schizophrenia" as such cannot be inherited, because it has behavioral and phenomenal contents which are learned. As Bleuler says, in order to have a delusion involving Jesuits one must first have learned about Jesuits. It seems inappropriate to apply the geneticist's concept of "penetrance" to the crude statistics of formal diagnosis—if a specific genetic etiology exists, its phenotypic expression in *psychological* categories would be a quantitative aberration in some parameter of a behavioral acquisition function. What could possibly be a genetically determined functional parameter capable of generating such diverse behavioral outcomes,

including the preservation of normal function in certain domains?

The theoretical puzzle is exaggerated when we fail to conceptualize at different levels of molarity. For instance, there is a tendency among organically minded theorists to analogize between catatonic phenomena and various neurological or chemically induced states in animals. But Bleuler's masterly *Theory of Schizophrenic Negativism* (1912) shows how the whole range of catatonic behavior, including diametrically opposite modes of relating to the interpersonal environment, can be satisfactorily explained as instrumental acts; thus even a convinced organicist, postulating a biochemical defect as specific etiology, should recognize that the causal linkage between this etiology and catatonia is indirect, requiring for the latter's derivation a lengthy chain of statements which are not even formulable except in molar psychological language.

What kind of behavioral fact about the patient leads us to diagnose schizophrenia? There are a number of traits and symptoms which get a high weight, and the weights differ among clinicians. But thought disorder continues to hold its own in spite of today's greater clinical interest in motivational (especially interpersonal) variables. If you are inclined to doubt this for yourself, consider the following indicators: Patient experiences intense ambivalence, readily reports conscious hatred of family figures, is pananxious, subjects therapist to a long series of testing operations, is withdrawn, and says, "Naturally, I am growing my father's hair."

While all of these are schizophrenic indicators, the last one is the diagnostic bell ringer. In this respect we are still Bleulerians, although we know a lot more about the schizophrenic's psychodynamics than Bleuler did. The significance of thought disorder, associative dyscontrol (or, as I prefer to call it so as to include the very mildest forms it may take, "cognitive slippage"), in schizophrenia has been somewhat de-emphasized in recent years. Partly this is due to the greater interest in interpersonal dynamics, but partly also to the realization that much of our earlier psychometric assessment of the thought disorder was mainly reflecting the schizophrenic's tendency to underperform because uninterested, preoccupied, resentful, or frightened. I suggest that this realization has been overgeneralized and led us to swing too far the

other way, as if we had shown that there really *is* no cognitive slippage factor present. One rather common assumption seems to be that if one can demonstrate the potentiating effect of a motivational state upon cognitive slippage, light has thereby been shed upon the etiology of schizophrenia. Why are we entitled to think this? Clinically, we see a degree of cognitive slippage not found to a comparable degree among nonschizophrenic persons. Some patients (e.g., pseudoneurotics) are highly anxious and exhibit minimal slippage; others (e.g., burnt-out cases) are minimally anxious with marked slippage. The demonstration that we can intensify a particular patient's cognitive dysfunction by manipulating his affects is not really very illuminating. After all, even ordinary neurological diseases can often be tremendously influenced symptomatically via emotional stimuli; but if a psychologist demonstrates that the spasticity or tremor of a multiple sclerotic is affected by rage or fear, we would not thereby have learned anything about the etiology of multiple sclerosis.

Consequent upon our general assimilation of the insights given us by psychoanalysis, there is today a widespread and largely unquestioned assumption that when we can trace out the motivational forces linked to the content of aberrant behavior, then we understand why the person has fallen ill. There is no compelling reason to assume this, when the evidence is mainly our dynamic understanding of the patient, however valid that may be. The phrase "why the person has fallen ill" may, of course, be legitimately taken to include these things; an account of how and when he falls ill will certainly include them. But they may be quite inadequate to answer the question, "Why does X fall ill and not Y, granted that we can understand both of them?" I like the analogy of a color psychosis, which might be developed by certain individuals in a society entirely oriented around the making of fine color discriminations. Social, sexual, economic signals are color mediated; to misuse a color word is strictly taboo; compulsive mothers are horribly ashamed of a child who is retarded in color development, and so forth. Some color-blind individuals (not all, perhaps not most) develop a color psychosis in this culture; as adults, they are found on the couches of color therapists, where a great deal of *valid* understanding is achieved about color dynamics. Some of them make a

social recovery. Nonetheless, if we ask, "What was basically the matter with these patients?" meaning, "What is the specific etiology of the color psychosis?" the answer is that mutated gene on the X chromosome. This is why my own therapeutic experience with schizophrenic patients has not yet convinced me of the schizophrenogenic mother as a specific etiology, even though the picture I get of my patients' mothers is pretty much in accord with the familiar one. There is no question here of accepting the patient's account; my point is that *given* the account, and taking it quite at face value, does not tell me why the patient is a patient and not just a fellow who had a bad mother.

Another theoretical lead is the one given greatest current emphasis, namely, *interpersonal aversiveness*. The schizophrenic suffers a degree of social fear, distrust, expectation of rejection, and conviction of his own unlovability which cannot be matched in its depth, pervasiveness, and resistance to corrective experience by any other diagnostic group.

Then there is a quasi-pathognomonic sign, emphasized by Rado (1956; Rado & Daniels, 1956) but largely ignored in psychologists' diagnostic usage, namely, *anhedonia*—a marked, widespread, and refractory defect in pleasure capacity which, once you learn how to examine for it, is one of the most consistent and dramatic behavioral signs of the disease.

Finally, I include *ambivalence* from Bleuler's cardinal four (1950). His other two, "autism" and "derealism," I consider derivative from the combination of slippage, anhedonia, and aversiveness. Crudely put, if a person cannot think straight, gets little pleasure, and is afraid of everyone, he will of course learn to be autistic and dereistic.

If these clinical characterizations are correct, and we combine them with the hypothesis of a genetic specific etiology, do they give us any lead on theoretical possibilities?

Granting its initial vagueness as a construct, requiring to be filled in by neurophysiological research, I believe we should take seriously the old European notion of an "integrative neural defect" as the only direct phenotypic consequence produced by the genic mutation. This is an aberration in some parameter of single cell function, which may or may not be manifested in the functioning of more molar CNS systems, depending upon the organization of the mutual feedback controls and upon the stochastic parameters of the reinforce-

ment regime. This neural integrative defect, which I shall christen *schizotaxia*, is all that can properly be spoken of as inherited. The imposition of a social learning history upon schizotaxic individuals results in a personality organization which I shall call, following Rado, the *schizotype*. The four core behavior traits are obviously not innate; but I postulate that they are universally learned by schizotaxic individuals, given any of the actually existing social reinforcement regimes, from the best to the worst. If the interpersonal regime is favorable, and the schizotaxic person also has the good fortune to inherit a low anxiety readiness, physical vigor, general resistance to stress and the like, he will remain a well-compensated "normal" schizotype, never manifesting symptoms of mental disease. He will be like the gout-prone male whose genes determine him to have an elevated blood uric acid titer, but who never develops clinical gout.

Only a subset of schizotypic personalities decompensate into clinical schizophrenia. It seems likely that the most important causal influence pushing the schizotype toward schizophrenic decompensation is the schizophrenogenic mother.

I hope it is clear that this view does not conflict with what has been established about the mother-child interaction. If this interaction were totally free of maternal ambivalence and aversive inputs to the schizotaxic child, even compensated schizotypy might be avoided; at most, we might expect to find only the faintest signs of cognitive slippage and other minimal neurological aberrations, possibly including body image and other proprioceptive deviations, but not the interpersonal aversiveness which is central to the clinical picture.

Nevertheless, while assuming the etiological importance of mother in determining the course of aversive social learnings, it is worthwhile to speculate about the modification our genetic equations might take on this hypothesis. Many schizophrenogenic mothers are themselves schizotypes in varying degrees of compensation. Their etiological contribution then consists jointly in their passing on the gene, *and* in the fact that being schizotypic, they provide the kind of ambivalent regime which potentiates the schizotypy of the child and raises the odds of his decompensating. Hence the incidence of the several parental genotypes among parent pairs of diagnosed proband cases is not calculable from the usual genetic formulas. For example, given a schizophrenic proband, the odds

that mother is homozygous (or, if the gene were dominant, that it is mother who carries it) are different from those for father; since we have begun by selecting a decompensated case, and formal diagnosis as the phenotype involves a potentiating factor for mother which is psychodynamically greater than that for a schizotypic father. Another important influence would be the likelihood that the lower fertility of schizophrenics is also present, but to an unknown degree, among compensated schizotypes. Clinical experience suggests that in the semicompensated range, this lowering of fertility is greater among males, since many schizotypic women relate to men in an exploited or exploitive sexual way, whereas the male schizotype usually displays a marked deficit in heterosexual aggressiveness. Such a sex difference in fertility among decompensated cases has been reported by Meyers and Goldfarb (1962).

Since the extent of aversive learnings is a critical factor in decompensation, the inherited anxiety readiness is presumably greater among diagnosed cases. Since the more fertile mothers are likely to be compensated, hence themselves to be relatively low anxiety if schizotaxic, a frequent parent pattern should be a compensated schizotypic mother married to a neurotic father, the latter being the source of the proband's high-anxiety genes (plus providing a poor paternal model for identification in male patients, and a weak defender of the child against mother's schizotypic hostility).

These considerations make ordinary family concordance studies, based upon formal diagnoses, impossible to interpret. The most important research need here is development of high-validity indicators for compensated schizotypy. I see some evidence for these conceptions in the report of Lidz and co-workers, who in studying intensively the parents of 15 schizophrenic patients were surprised to find that "minimally, 9 of the 15 patients had at least one parent who could be called schizophrenic, or ambulatory schizophrenic, or clearly paranoid in behavior and attitudes" (Lidz, Cornelison, Terry, & Fleck, 1958, p. 308). As I read the brief personality sketches presented, I would judge that all but two of the probands had a clearly schizotypic parent. These authors, while favoring a "learned irrationality" interpretation of their data, also recognize the alternative genetic interpretation. Such facts do not permit a decision, obviously; my main point is the striking difference

between the high incidence of parental schizotypes, mostly quite decompensated (some to the point of diagnosable psychosis), and the zero incidence which a conventional family concordance study would have yielded for this group.

Another line of evidence, based upon a very small sample but exciting because of its uniformity, is McConaghy's report (1959) that among non-diagnosed parent pairs of 10 schizophrenics, sub-clinical thought disorder was psychometrically detectable in at least one parent of every pair. Rosenthal (in press) reports that he can add five tallies to this parent-pair count, and suggests that such results might indicate that the specific heredity is dominant, and completely penetrant, rather than recessive. The attempt to replicate these findings, and other psychometric efforts to tap subclinical cognitive slippage in the "normal" relatives of schizophrenics, should receive top priority in our research efforts.

Summarizing, I hypothesize that the statistical relation between schizotaxia, schizotypy, and schizophrenia is class inclusion: All schizotaxics become, *on all actually existing social learning regimes*, schizotypic in personality organization; but most of these remain compensated. A minority, disadvantaged by other (largely polygenically determined) constitutional weaknesses, and put on a bad regime by schizophrenogenic mothers (most of whom are themselves schizotypes) are thereby potentiated into clinical schizophrenia. What makes schizotaxia etiologically specific is its role as a *necessary* condition. I postulate that a non-schizotaxic individual, whatever his other genetic makeup and whatever his learning history, would at most develop a character disorder or a psychoneurosis; but he would not become a schizotype and therefore could never manifest its decompensated form, schizophrenia.

What sort of quantitative aberration in the structural or functional parameters of the nervous system can we conceive to be directly determined by a mutated gene, and to so alter initial dispositions that affected individuals will, in the course of their childhood learning history, develop the four schizotypal source traits: cognitive slippage, anhedonia, ambivalence, and interpersonal aversiveness? To me, the most baffling thing about the disorder is the phenotypic heterogeneity of this tetrad. If one sets himself to the task of doing a theoretical Vigotsky job on this list of psychological

dispositions, he may manage part of it by invoking a sufficiently vague kind of descriptive unity between ambivalence and interpersonal aversiveness; and perhaps even anhedonia could be somehow subsumed. But the cognitive slippage presents a real roadblock. Since I consider cognitive slippage to be a core element in schizophrenia, any characterization of schizophrenic or schizotypic behavior which purports to abstract its essence but does not include the cognitive slippage must be deemed unsatisfactory. I believe that an adequate theoretical account will necessitate moving downward in the pyramid of the sciences to invoke explanatory constructs not found in social, psychodynamic, or even learning theory language, but instead at the neurophysiological level.

Perhaps we don't know enough about "how the brain works" to theorize profitably at that level; and I daresay that the more a psychologist knows about the latest research on brain function, the more reluctant he would be to engage in etiological speculation. Let me entreat my physiologically expert listeners to be charitable toward this clinician's premature speculations about how the schizotaxic brain might work. I feel partially justified in such speculating because there are some well-attested general truths about mammalian learned behavior which could almost have been set down from the armchair, in the way engineers draw block diagrams indicating what kinds of parts or subsystems a physical system *must* have, and what their interconnections *must* be, in order to function "appropriately." Brain research of the last decade provides a direct neurophysiological substrate for such cardinal behavior requirements as avoidance, escape, reward, drive differentiation, general and specific arousal or activation, and the like (see Delafresnaye, 1961; Ramey & O'Doherty, 1960). The discovery in the limbic system of specific positive reinforcement centers by Olds and Milner in 1954, and of aversive centers in the same year by Delgado, Roberts, and Miller (1954), seems to me to have an importance that can scarcely be exaggerated; and while the ensuing lines of research on the laws of intracranial stimulation as a mode of behavior control present some puzzles and paradoxes, what *has* been shown up to now may already suffice to provide a theoretical framework. As a general kind of brain model let us take a broadly Hebbian conception in combination with the findings on intracranial stimulation.

To avoid repetition I shall list some basic assumptions first but introduce others in context and only implicitly when the implication is obvious. I shall assume that:

When a presynaptic cell participates in firing a postsynaptic cell, the former gains an increment in firing control over the latter. Coactivation of anatomically connected cell assemblies or assembly systems therefore increases their stochastic control linkage, and the frequency of discharges by neurons of a system may be taken as an intensity variable influencing the growth rate of intersystem control linkage as well as the momentary activity level induced in the other systems. (I shall dichotomize acquired cortical systems into "perceptual-cognitive," including central representations of goal objects; and "instrumental," including overarching monitor systems which select and guide specific effector patterns.)

Most learning in mature organisms involves altering control linkages between systems which themselves have been consolidated by previous learnings, sometimes requiring thousands of activations and not necessarily related to the reinforcement operation to the extent that perceptual-to-instrumental linkage growth functions are.

Control linkage increments from coactivation depend heavily, if not entirely, upon a period of reverberatory activity facilitating consolidation.

Feedback from positive limbic centers is facilitative to concurrent perceptual-cognitive or instrumental sequences, whereas negative center feedback exerts an inhibitory influence. (These statements refer to initial features of the direct wiring diagram, not to all long-term results of learning.) Aversive input also has excitatory effects via the arousal system, which maintain activity permitting escape learning to occur because the organism is alerted and keeps doing things. But I postulate that this overall influence is working along with an opposite effect, quite clear from both molar and intracranial experiments, that a major biological function of aversive-center activation is to produce "stoppage" of whatever the organism is currently doing.

Perceptual-cognitive systems and limbic motivational control centers develop two-way mutual controls (e.g., discriminative stimuli acquire the reinforcing property; "thoughts" become pleasantly toned; drive-relevant perceptual components are "souped-up.")

What kind of heritable parametric aberration could underlie the schizotaxic's readiness to acquire the schizotypic tetrad? It would seem, first of all, that the defect is much more likely to reside in the neurone's synaptic control function than in its storage function. It is hard to conceive of a general defect in storage which would on the one hand permit so many perceptual-cognitive functions, such as tapped by intelligence tests, school learning, or the high order cognitive powers displayed by some schizotypes, and yet have the diffuse motivational and emotional effects found in these same individuals. I am not saying that a storage deficit is clearly excludable, but it hardly seems the best place to look. So we direct our attention to parameters of control.

One possibility is to take the anhedonia as fundamental. What is *phenomenologically* a radical pleasure deficiency may be roughly identified *behaviorally* with a quantitative deficit in the positive reinforcement growth constant, and each of these—the "inner" and "outer" aspects of the organism's appetitive control system—reflect a quantitative deficit in the limbic "positive" centers. The anhedonia would then be a direct consequence of the genetic defect in wiring. Ambivalence and interpersonal aversiveness would be quantitative deviations in the balance of appetitive-aversive controls. Most perceptual-cognitive and instrumental learnings occur under mixed positive and negative schedules, so the normal consequence is a collection of habits and expectancies varying widely in the intensity of their positive and negative components, but mostly "mixed" in character. Crudely put, everybody has *some* ambivalence about almost everything, and everybody has *some* capacity for "social fear." Now if the brain centers which mediate phenomenal pleasure and behavioral reward are numerically sparse or functionally feeble, the aversive centers meanwhile functioning normally, the long-term result would be a general shift toward the aversive end, appearing clinically as ambivalence and exaggerated interpersonal fear. If, as Brady believes, there is a wired-in reciprocal inhibiting relation between positive and negative centers, the long-term aversive drift would be further potentiated (i.e., what we see at the molar level as a sort of "softening" or "soothing" effect of feeding or petting upon anxiety elicitors would be reduced).

Cognitive slippage is not as easy to fit in, but if

we assume that normal ego function is acquired by a combination of social reinforcements and the self-reinforcements which become available to the child via identification; then we might say roughly that "everybody has to learn *how* to think straight." Rationality is socially acquired; the secondary process and the reality principle are slowly and imperfectly learned, by even the most clear headed. Insofar as slippage is manifested in the social sphere, such an explanation has some plausibility. An overall aversive drift would account for the paradoxical schizotypic combination of interpersonal distortions and acute perceptiveness of others' unconscious, since the latter is really a hypersensitivity to aversive signals rather than an overall superiority in realistically discriminating social cues. On the output side, we might view the cognitive slippage of mildly schizoid speech as originating from poorly consolidated second-order "monitor" assembly systems which function in an editing role, their momentary regnancy constituting the "set to communicate." At this level, selection among competing verbal operants involves slight differences in appropriateness for which a washed-out social reinforcement history provides an insufficiently refined monitor system. However, if one is impressed with the presence of a pervasive and primary slippage, showing up in a diversity of tests (cf. Payne, 1961) and also on occasions when the patient is desperately trying to communicate, an explanation on the basis of deficient positive center activity is not too convincing.

This hypothesis has some other troubles which I shall merely indicate. Schizoid anhedonia is mainly interpersonal, i.e., schizotypes seem to derive adequate pleasure from esthetic and cognitive rewards. Secondly, some successful psychotherapeutic results include what appears to be a genuine normality of hedonic capacity. Thirdly, regressive electroshock sometimes has the same effect, and the animal evidence suggests that shock works by knocking out the aversive control system rather than by souping up appetitive centers. Finally, if the anhedonia is really general in extent, it is hard to conceive of any simple genetic basis for weakening the different positive centers, whose reactivity has been shown by Olds and others to be chemically drive specific.

A second neurological hypothesis takes the slippage factor as primary. Suppose that the immediate consequence of whatever biochemical aber-

ration the gene directly controls were a specific alteration in the neurone's membrane stability, such that the distribution of optional transmission probabilities is more widely dispersed over the synaptic signal space than in normals. That is, presynaptic input signals whose spatio-temporal configuration locates them peripherally in the neurone's signal space yield transmission probabilities which are relatively closer to those at the maximum point, thereby producing a kind of de-differentiation or flattening of the cell's selectivity. Under suitable parametric assumptions, this synaptic slippage would lead to a corresponding dedifferentiation of competing interassembly controls, because the elements in the less frequently or intensely coactivated control assembly would be accumulating control increments more rapidly than normal. Consider a perceptual-cognitive system whose regnancy is preponderantly associated with positive-center coactivation but sometimes with aversive. The cumulation of control increments will draw these apart; but if synaptic slippage exists, their difference, at least during intermediate stages of control development, will be attenuated. The intensity of aversive-center activation by a given level of perceptual-cognitive system activity will be exaggerated relative to that induced in the positive centers. For a preponderantly aversive control this will be reversed. But now the different algebraic sign of the feedbacks introduces an important asymmetry. Exaggerated negative feedback will tend to lower activity level in the predominantly appetitive case, retarding the growth of the control linkage; whereas exaggerated positive feedback in the predominantly aversive case will tend to heighten activity levels, accelerating the linkage growth. The long-term tendency will be that movement in the negative direction which I call *aversive drift*. In addition to the asymmetry generated by the difference in feedback signs, certain other features in the mixed-regime setup contribute to aversive drift. One factor is the characteristic difference between positive and negative reinforcers in their role as strengtheners. It seems a fairly safe generalization to say that positive centers function only weakly as strengtheners when "on" continuously, and mainly when they are turned on as terminators of a cognitive or instrumental sequence; by contrast, negative centers work mainly as "off" signals, tending to inhibit elements while steadily "on." We may suppose that

the former strengthen mainly by facilitating post-activity reverberation (and hence consolidation) in successful systems, the latter mainly by holding down such reverberation in unsuccessful ones. Now a slippage-heightened aversive steady state during predominantly appetitive control sequences reduces their activity level, leaves fewer recently active elements available for a subsequent Olds-plus "on" signal to consolidate. Whereas a slippage-heightened Olds-plus steady state during predominantly aversive control sequences (a) increases their negative control *during* the "on" period and (b) leaves relatively more of their elements recently active and hence further consolidated by the negative "off" signal when it occurs. Another factor is exaggerated competition by aversively controlled sequences, whereby the appetitive chains do not continue to the stage of receiving socially mediated positive reinforcement, because avoidant chains (e.g., phobic behavior, withdrawal, intellectualization) are getting in the way. It is worth mentioning that the schizophrenogenic mother's regime is presumably "mixed" not only in the sense of the frequent and unpredictable aversive inputs she provides in response to the child's need signals, but also in her greater tendency to present such aversive inputs *concurrently* with drive reducers—thereby facilitating the "scrambling" of appetitive-and-aversive controls so typical of schizophrenia.

The schizotype's dependency guilt and aversive overreaction to offers of help are here seen as residues of the early knitting together of his cortical representations of appetitive goals with punishment-expectancy assembly systems. Roughly speaking, he has learned that to want anything interpersonally provided is to be endangered.

The cognitive slippage is here conceived as a direct molar consequence of synaptic slippage, potentiated by the disruptive effects of aversive control and inadequate development of interpersonal communication sets. Cognitive and instrumental linkages based upon sufficiently massive and consistent regimes, such as reaching for a seen pencil, will converge to asymptotes hardly distinguishable from the normal. But systems involving closely competing strengths and automatized selection among alternatives, especially when the main basis of acquisition and control is social reward, will exhibit evidences of malfunction.

My third speculative model revives a notion with a long history, namely, that the primary schizotaxic

defect is a quantitative deficiency of inhibition. (In the light of Milner's revision of Hebb, in which the inhibitory action of Golgi Type II cells is crucial even for the formation of functionally differentiated cell assemblies, a defective inhibitory parameter could be an alternative basis for a kind of slippage similar in its consequences to the one we have just finished discussing.) There are two things about this somewhat moth-eaten "defective inhibition" idea which I find appealing. First, it is the most direct and uncomplicated neurologizing of the schizoid cognitive slippage. Schizoid cognitive slippage is neither an incapacity to link, nor is it an unhealthy overcapacity to link; rather it seems to be a defective *control* over associations which are also accessible to the healthy (as in dreams, wit, psychoanalytic free association, and certain types of creative work) but are normally "edited out" or "automatically suppressed" by those superordinate monitoring assembly systems we lump together under the term "set." Secondly, in working with pseudoneurotic cases one sees a phenomenon to which insufficient theoretical attention has been paid: Namely, these patients cannot turn off painful thoughts. They suffer constantly and intensely from painful thoughts about themselves, about possible adverse outcomes, about the past, about the attitudes and intentions of others. The "weak ego" of schizophrenia means a number of things, one of which is failure of defense; the schizophrenic has too ready access to his own id, and is too perceptive of the unconscious of others. It is tempting to read "failure of defense" as "quantitatively deficient inhibitory feedback." As mentioned earlier, aversive signals (whether exteroceptive or internally originated) must exert both an exciting effect via the arousal system and a quick-stoppage effect upon cortical sequences which fail to terminate the ongoing aversive signal, leading the organism to shift to another. Suppose the gene resulted in an insufficient production (or too rapid inactivation) of the specific inhibitory transmitter substance, rendering all inhibitory neurones quantitatively weaker than normal. When aversively linked cognitive sequences activate negative limbic centers, these in turn soup up the arousal system normally but provide a subnormal inhibitory feedback, thereby permitting their elicitor to persist for a longer time and at higher intensity than normal. This further activates the negative control center, and so on, until an equilibrium level is reached



which is above normal in intensity all around, and which meanwhile permits an excessive linkage growth in the aversive chain. (In this respect the semicompenated case would differ from the late-stage deteriorated schizophrenic, whose aversive drift has gradually proliferated so widely that almost any cognitive or instrumental chain elicits an overlearned defensive "stoppage," whereby even the inner life undergoes a profound and diffuse impoverishment.)

The mammalian brain is so wired that aversive signals tend to produce stoppage of regnant cognitive or instrumental sequences without the aversive signal having been specifically connected to their controlling cues or motivational systems. E.g., lever pressing under thirst or hunger can be inhibited by shock-associated buzzer, even though the latter has not been previously connected with hunger, paired with the discriminative stimulus, nor presented as punishment for the operant. A deficient capacity to inhibit concurrent activity of fringe elements (aversively connected to ambiguous social inputs from ambivalent mother) would accelerate the growth of linkages between them and appetitive systems not hitherto punished. Sequential effects are here especially important, and combine with the schizophrenogenic mother's tendency not to provide differential cues of high consistency as predictors of whether aversive or appetitive consequences will follow upon the child's indications of demand.

Consider two cortical systems having shared "fringe" subsystems (e.g., part percepts of mother's face). When exteroceptive inputs are the elicitors, negative feedback from aversive centers cannot usually produce stoppage; in the absence of such overdetermining external controls, the relative activity levels are determined by the balance of facilitative and inhibitory feedbacks. "Fringe" assemblies which have already acquired more aversive control, if they begin to be activated by regnant perceptual-cognitive sequences, will increase inhibitory feedback; and being "fringe" they can thereby be held down. The schizotaxic, whose aversive-feedback stoppage of fringe-element activity is weakened, accumulates excessive intertrial Hebbian increments toward the aversive side, the predominantly aversive fringe elements being more active and becoming more knit into the system than normally. On subsequent exteroceptively controlled trials, whenever the overdetermining stimulus input

activates predominantly aversive perceptual-cognitive assemblies, their driving of the negative centers will be heightened. The resulting negative feedback may now be strong enough that, when imposed upon "fringe" assemblies weakly activated and toward the appetitive side, it can produce stoppage. On such occasions the more appetitive fringe elements will be retarded in their linkage growth, receiving fewer Hebbian increments. And those which do get over threshold will become further linked during such trials to the concurrent negative center activity. The result is twofold: a retarded growth of appetitive perceptual-cognitive linkages; and a progressive drawing of fringe elements into the aversive ambit.

"Ambiguous regimes," where the pairing of S<sup>+</sup> and S<sup>-</sup> inputs occurs very unpredictably, will have a larger number of fringe elements. Also, if the external schedule is dependent upon regnant appetitive drive states as manifested in the child's instrumental social acts, so that these are often met with mixed S<sup>+</sup> (drive-relevant) and S<sup>-</sup> (anxiety-eliciting) inputs, the appetitive and aversive assemblies will tend to become linked, and to activate positive and negative centers concurrently. The anhedonia and ambivalence would be consequences of this plus-minus "scrambling," especially if the positive and negative limbic centers are mutually inhibitory but here deficiently so. We would then expect schizotypic anhedonia to be basically interpersonal, and only derivatively present, if at all, in other contexts. This would in part explain the schizotype's preservation of relatively normal function in a large body of instrumental domains. For example, the acquisition of basic motor and cognitive skills would be relatively less geared to a mixed input, since "successful" mastery is both mechanically rewarded (e.g., how to open a door) and also interpersonally rewarded as "school success," etc. The hypercathexis of intellect, often found even among nonbright schizotypes, might arise from the fact that these performances are rewarded rather "impersonally" and make minimal demands on the reinforcing others. Also, the same cognitive and mechanical instrumental acts can often be employed both to turn on positive center feedback and to turn off negative, an equivalence much less true of purely social signals linked to interpersonal needs.

Having briefly sketched three neurological possibilities for the postulated schizotaxic aberration,

let me emphasize that while each has sufficient merit to be worth pursuing, they are mainly meant to be illustrative of the vague concept "integrative neural defect." I shall myself not be surprised if all three are refuted, whereas I shall be astounded if future research shows no fundamental aberration in nerve-cell function in the schizotype. Postulating schizotaxia as an open concept seems at first to pose a search problem of needle-in-haystack proportions, but I suggest that the plausible alternatives are really somewhat limited. After all, what does a neuron do to another neuron? It excites, or it inhibits! The schizotypic preservation of relatively normal function in selected domains directs our search toward some minimal deviation in a synaptic control parameter, as opposed to, say, a gross defect in cell distribution or structure, or the kind of biochemical anomaly that yields mental deficiency. Anything which would give rise to defective storage, grossly impaired transmission, or sizable limitations on functional complexity can be pretty well excluded on present evidence. What we are looking for is a quantitative aberration in synaptic control—a deviation in amount or patterning of excitatory or inhibitory action—capable of yielding cumulative departures from normal control linkages under mixed appetitive-aversive regimes; but slight enough to permit convergence to quasi-normal asymptotes under more consistent schedules (or when massive repetition with motive-incentive factors unimportant is the chief basis for consolidation). The defect must generate aversive drift on mixed social reinforcement regimes, and must yield a primary cognitive slippage which, however, may be extremely small in magnitude except as potentiated by the cumulative effects of aversive drift. Taken together these molar constraints limit our degrees of freedom considerably when it comes to filling in the neurophysiology of schizotaxia.

Leaving aside the specific nature of schizotaxia, we must now raise the familiar question whether such a basic neurological defect, however subtle and nonstructural it might be, should not have been demonstrated hitherto? In reply to this objection I shall content myself with pointing out that there are several lines of evidence which, while not strongly arguing *for* a neurological theory, are rebuttals of an argument presupposing clear and consistent *negative* findings. For example: Ignoring

several early European reports with inadequate controls, the literature contains a half-dozen quantitative studies showing marked vestibular system dysfunction in schizophrenics (Angyal & Blackman, 1940, 1941; Angyal & Sherman, 1942; Colbert & Koegler, 1959; Freeman & Rodnick, 1942; Leach, 1960; Payne & Hewlett, 1960; Pollock & Krieger, 1958). Hoskins (1946) concluded that a neurological defect in the vestibular system was one of the few clear-cut biological findings in the Worcester studies. It is of prime importance to replicate these findings among compensated and pseudoneurotic cases, where the diffuse withdrawal and deactivation factor would not provide the explanation it does in the chronic, burnt-out case (cf. Collins, Crampton, & Posner, 1961). Another line of evidence is in the work of King (1954) on psychomotor deficit, noteworthy for its careful use of task simplicity, asymptote performance, concern for patient cooperation, and inclusion of an outpatient pseudoneurotic sample. King himself regards his data as indicative of a rather basic behavior defect, although he does not hold it to be schizophrenia-specific. Then we have such research as that of Barbara Fish (1961) indicating the occurrence of varying signs of perceptual-motor maldevelopment among infants and children who subsequently manifest clinical schizophrenia. The earlier work of Schilder and Bender along these lines is of course well known, and there has always been a strong minority report in clinical psychiatry that many schizophrenics provide subtle and fluctuating neurological signs of the "soft" variety, if one keeps alert to notice or elicit them. I have myself been struck by the frequent occurrence, even among pseudoneurotic patients, of transitory neurologic-like complaints (e.g., diplopia, localized weakness, one-sided tremor, temperature dyscontrol, dizziness, disorientation) which seem to lack dynamic meaning or secondary gain and whose main effect upon the patient is to produce bafflement and anxiety. I have seen preliminary findings by J. McVicker Hunt and his students in which a rather dramatic quantitative deficiency in spatial cognizing is detectable in schizophrenics of above-normal verbal intelligence. Research by Cleveland (1960; Cleveland, Fisher, Reitman, & Rothaus, 1962) and by Arnhoff and Damianopoulos (in press) on the clinically well-known body-image anomalies in schizophrenia suggests that this domain yields

quantitative departures from the norm of such magnitude that with further instrumental and statistical refinement it might be used as a quasi-pathognomonic sign of the disease. It is interesting to note a certain thread of unity running through this evidence, which perhaps lends support to Rado's hypothesis that a kinesthetic integrative defect is even more characteristic of schizotypy than is the radical anhedonia.

All these kinds of data are capable of a psychodynamic interpretation. "Soft" neurological signs are admittedly ambiguous, especially when found in the severely decompensated case. The only point I wish to make here is that *since* they exist and are at present unclear in etiology, an otherwise plausible neurological view cannot be refuted on the ground that there is a *lack* of any sign of neurological dysfunction in schizophrenia; there is no such lack.

Time forces me to leave detailed research strategy for another place, but the main directions are obvious and may be stated briefly: The clinician's Mental Status ratings on anhedonia, ambivalence, and interpersonal aversiveness should be objectified and preferably replaced by psychometric measures. The research findings on cognitive slippage, psychomotor dyscontrol, vestibular malfunction, body image, and other spatial aberrations should be thoroughly replicated and extended into the pseudo-neurotic and semicomensated ranges. If these efforts succeed, it will be possible to set up a multiple sign pattern, using optimal cuts on phenotypically diverse indicators, for identifying compensated schizotypes in the nonclinical population. Statistics used must be appropriate to the theoretical model of a dichotomous latent taxonomy reflecting itself in otherwise independent quantitative indicators. Family concordance studies should then be run relating proband schizophrenia to schizotypy as identified by this multiple indicator pattern. Meanwhile we should carry on an active and varied search for more direct neurological signs of schizotaxia, concentrating our hunches on novel stimulus inputs (e.g., the stabilized retinal image situation) which may provide a better context for basic neural dysfunction to show up instead of being masked by learned compensations or imitated by psychopathology.

In closing, I should like to take this unusual propaganda opportunity to play the prophet. It is

my strong personal conviction that such a research strategy will enable psychologists to make a unique contribution in the near future, using psychological techniques to establish that schizophrenia, while its content is learned, is fundamentally a neurological disease of genetic origin.

## REFERENCES

- ANGYAL, A., & BLACKMAN, N. Vestibular reactivity in schizophrenia. *Arch. Neurol. Psychiat.*, 1940, **44**, 611-620.
- ANGYAL, A., & BLACKMAN, N. Paradoxical reactions in schizophrenia under the influence of alcohol, hyperpnea, and CO<sub>2</sub> inhalation. *Amer. J. Psychiat.*, 1941, **97**, 893-903.
- ANGYAL, A., & SHERMAN, N. Postural reactions to vestibular stimulation in schizophrenic and normal subjects. *Amer. J. Psychiat.*, 1942, **98**, 857-862.
- ARNHOFF, F., & DAMIANOPOULOS, E. Self-body recognition and schizophrenia: An exploratory study. *J. abnorm. soc. Psychol.*, in press.
- BLEULER, E. *Theory of schizophrenic negativism*. New York: Nervous and Mental Disease Publishing, 1912.
- BLEULER, E. *Dementia praecox*. New York: International Universities Press, 1950.
- CLEVELAND, S. E. Judgment of body size in a schizophrenic and a control group. *Psychol. Rep.*, 1960, **7**, 304.
- CLEVELAND, S. E., FISHER, S., REITMAN, E. E., & ROTHHAUS, P. Perception of body size in schizophrenia. *Arch. gen. Psychiat.*, 1962, **7**, 277-285.
- COLBERT, G., & KOEGLER, R. Vestibular dysfunction in childhood schizophrenia. *AMA Arch. gen. Psychiat.*, 1959, **1**, 600-617.
- COLLINS, W. E., CRAMPTON, G. H., & POSNER, J. B. The effect of mental set upon vestibular nystagmus and the EEG. *USA Med. Res. Lab. Rep.*, 1961, No. 439.
- DELAFFRESNAYE, J. F. (Ed.) *Brain mechanisms and learning*. Springfield, Ill.: Charles C Thomas, 1961.
- DELGADO, J. M. R., ROBERTS, W. W., & MILLER, N. E. Learning motivated by electrical stimulation of the brain. *Amer. J. Physiol.*, 1954, **179**, 587-593.
- FISH, BARBARA. The study of motor development in infancy and its relationship to psychological functioning. *Amer. J. Psychiat.*, 1961, **117**, 1113-1118.
- FREEMAN, H., & RODNICK, E. H. Effect of rotation on postural steadiness in normal and schizophrenic subjects. *Arch. Neurol. Psychiat.*, 1942, **48**, 47-53.
- FULLER, J. L., & THOMPSON, W. R. *Behavior genetics*. New York: Wiley, 1960. Pp. 272-283.
- HOSKINS, R. G. *The biology of schizophrenia*. New York: Norton, 1946.
- KING, H. E. *Psychomotor aspects of mental disease*. Cambridge: Harvard Univer. Press, 1954.
- LEACH, W. W. Nystagmus: An integrative neural deficit in schizophrenia. *J. abnorm. soc. Psychol.*, 1960, **60**, 305-309.
- LIDZ, T., CORNELISON, A., TERRY, D., & FLECK, S. Intra-familial environment of the schizophrenic patient: VI.

- The transmission of irrationality. *AMA Arch. Neurol. Psychiat.*, 1958, **79**, 305-316.
- MCCONAGHY, N. The use of an object sorting test in elucidating the hereditary factor in schizophrenia. *J. Neurol. Neurosurg. Psychiat.*, 1959, **22**, 243-246.
- MEYERS, D., & GOLDFARB, W. Psychiatric appraisals of parents and siblings of schizophrenic children. *Amer. J. Psychiat.*, 1962, **118**, 902-908.
- OLDS, J., & MILNER, P. Positive reinforcement produced by electrical stimulation of septal area and other regions of rat brain. *J. comp. physiol. Psychol.*, 1954, **47**, 419-427.
- PAYNE, R. W. Cognitive abnormalities. In H. J. Eysenck (Ed.), *Handbook of abnormal psychology*. New York: Basic Books, 1961. Pp. 248-250.
- PAYNE, R. S., & HEWLETT, J. H. G. Thought disorder in psychotic patients. In H. J. Eysenck (Ed.), *Experiments in personality*. Vol. 2. London: Routledge, Kegan, Paul, 1960. Pp. 3-106.
- POLLACK, M., & KRIEGER, H. P. Oculomotor and postural patterns in schizophrenic children. *AMA Arch. Neurol. Psychiat.*, 1958, **79**, 720-726.
- RADO, S. *Psychoanalysis of behavior*. New York: Grune & Stratton, 1956.
- RADO, S., & DANIELS, G. *Changing concepts of psychoanalytic medicine*. New York: Grune & Stratton, 1956.
- RAMEY, E. R., & O'DOHERTY, D. S. (Ed.) *Electrical studies on the unanesthetized brain*. New York: Hoeber, 1960.
- ROSENTHAL, D. Problems of sampling and diagnosis in the major twin studies of schizophrenia. *J. psychiat. Res.*, in press.
- STERN, K. *Principles of human genetics*. San Francisco: Freeman, 1960. Pp. 581-584.