February 15, 1966

[Completed March 1967]

TO: D. T. Lykken

FROM: P. E. Meehl

RE: Problems of strategy in research on schizotaxia

[Pagination does not correspond to original memo]

CONTENTS

February 15, 1966

TO: D. T. Lykken

FROM: P. E .Meehl

RE: Problems of strategy in research on schizotaxia

1. I continue to postulate three constructs: 'schizotaxia,' 'schizotypy,' and 'schizophrenia.' Whatever may be the statistical relationship among these, they are distinguishable constructs, because they refer to different domains of data or to different levels of analysis or both. Even if all schizotaxics become schizotypic as a result of social learning, and if all schizotypic individuals become such on the basis of schizotaxia, so that there is a one-to-one correspondence over the domain of persons; nevertheless 'schizotypy' and 'schizotaxia' are not the same concept; since one is formulated in personality terms while the other, however vague and speculative, aims at an "open concept" in neurophysiology.

2. By 'schizotaxia' I mean a postulated "neural integrative defect" (Hoch) which is a constitutional characteristic of the schizotaxic individual's central nervous system. I am inclined at present to reject two of the three speculative notions in my APA presidential address, and to return to my original one (which is the second of three speculative ideas set forth there), which postulates that the neurological defect is specifically a reduced steepness of the gradient of differentiation in a synaptic signal space. You may remember three or four years ago I was talking about a neologism 'hypokrisia' meaning "insufficient discrimination." This differs from, say, Mednick in that he postulates a molar (autonomic?) defect and his theory is discorroborated whenever he fails to find one; whereas I do not postulate a molar defect but attribute hypokrisia at the level of the individual nervous element. Whether and to what extent this shows up at the molar level would depend upon other factors, including the relative importance of wiring versus learning, the possibility of self-correcting or compensatory learnings by a hypokrisic individual, and the like.

One might think of this postulated hypokrisia as a micro-duplication of the macro-phenomenon of cognitive slippage. Thus, I am doing precisely what Fred Skinner says is a sin, namely, attributing a molar aberration to a qualitatively similar aberration at the micro-level. That doesn't bother me because it has paid off in a lot of other scientific fields. Whereas many molar properties cannot be meaningfully attributed at the micro-level, others can. The schizotype is shy, but his neurons aren't shy. The schizotype has body image problems, his neurons do not. Eisenhower is Republican, but his (single) neurons are not. But I am quite willing to postulate that the "cognitive slippage" of the schizotype corresponds to, and literally arises from, a kind of "synaptic slippage," with the further hope that this postulated micro-level slippage can, together with the facts of social experience, enable us to understand how other molar features not obviously related to slippage come to pass.

Taking a particular micro-anatomy, that is, a neuron with a specified arrangement of pre-synaptic fibers terminating on its surface, if we associate each of the several pre-synaptic loci (even if they run into the hundreds) with a dimension in a synaptic signal space; and starting from some arbitrary zero time we represent the time of arrival of a

pre-synaptic spike at each locus by a coordinate. (By a locus I mean an end-foot or one of those bulges on axonal termini which collectively constitute the so-called "synaptic scale." I do not mean by 'locus' the entire synaptic juncture between Cell A and Cell B.) Then a given spatio-temporal configuration or pattern of pre-synaptic spike arrivals determines a unique point in the synaptic signal space. Corresponding to each of these points in the signal space there exists an "optional transmission probability" of the neuron firing. So far as I know up to this point we are operating with concepts that are pretty well established, that is, we know that the normal circumstance is for pre-synaptic impulses from different sources to converge on a post-synaptic cell and we know that if the timing of these is not suitable, what results is a short-lived local disturbance on the surface of the post-synaptic cell which then fades out so the cell doesn't get over the spiking threshold. We do not of course assume that these probabilities are all less than one for all points in the signal space, i.e., there are some patterns of inputs so timed and located (or sufficiently massive) that the post-synaptic cell is "certain" to discharge.

Fixing a particular micro-anatomic arrangement with k distinguishable synaptic loci we can conceive of a (k + 1)-space in which the transition-probability appears as a rather complicated hypersurface, and the steepness of its "hills and valleys" correspond to the cell's ability to discriminate or differentiate various synaptic input patterns. Whatever might be the most powerful mathematical function for putting these gradients together, a flattened or dedifferentiated topography of this hypersurface is essentially what I am postulating for the hypokrisic individual. That is, a combination of pre-synaptic inputs which is not optimally timed (for either him or the orthokrisic individual) will, at least on the average, correspond to a transmission probability less removed from that of the optimal or the several maxima (i.e., mountain peaks in this signal space) as would be true of the normal specimen. Thus, for instance, I think of the Bleuler "loosened associations" as arising at least in part from the fact that neural elements belonging to different but related cell assemblies (or cell-assembly-systems) tend gradually to accumulate Hebbian increments of passive control which a similarly located cell, on a similarly arranged regime, would not be accumulating or at least not nearly as rapidly, for a normal person. So that even though, for instance, the entire assembly-system, say, corresponding to a simple motor act or verbal phoneme does not get over its "molar threshold," its constituent elements are being gradually inter-knitted with certain adjacent or remote elements; so that on future occasions a proper combination of inputs from several different adjacent or remote systems will suffice to activate an assembly on the basis of this, so to speak, "latent accumulated control." I don't know how clear this is but it will have to do for now.

3. Not that it's of any importance at this stage of things, but if I really had to go out on a limb and guess I would guess that there is a very direct line between the mutated gene and hypokrisia. Since the passage of the nervous impulse and its arousal on a post-synaptic cell's surface consists essentially of a breakdown of the membrane (an actual disarrangement of the molecules making up the membrane) so that the potential difference set up by its differential permeability to various ions is momentarily destroyed, the essence of hypokrisia might be literally a reduced membrane stability, a greater readiness for a region of local disturbance to spread, or perhaps to maintain itself for a somewhat longer time-interval, than is normal. I assume that there is some aberrated molecule that doesn't quite have the structure it should have to do its job in helping to maintain the stability of the membrane. And I would guess that this substance is anabolized within the neurons

themselves. We don't have a situation like, say, Gellhorn's where some other bodily process in the vegetative system or the endocrine system or the carbohydrate metabolism system is indirectly responsible for the neural malfunction. The relevance this has for research strategy is that it would be a mistake to rely upon things like adult autonomic reactivity, inasmuch as on the present view they are several steps removed from the mutated gene in the causal chain. Whereas hypokrisia, or properly chosen CNS functions directly reflective of it at the neurological level, are as close as you can get unless you want to play the bio-chemists' game rather than the psychologists' or neurologists' game.

4. In passing I may say that I cannot believe that Hebb's (1949) postulate is correct. It seems to me that if it were true that every time a pre-synaptic cell participates in the firing of a post-synaptic cell, then at the molar level we would expect (at least on the average) that the Thorndikeian "law of exercise" was almost as powerful as the "law of effect," which is not the case. It may be that a pure law of exercise functions in the case of adjacent neurons or cell assemblies when there's actual neural anatomical closeness, combined with a massive and consistent kind of regime of sensory inputs. So I am prepared to go along with Hebb in assuming that a puppy or a human baby does not have to be reinforced (in either the Pavlovian or Skinnerian sense) in order to build up integrated perceptual systems in his brain. But for anatomically remote systems the law of exercise seems to have such a feeble power that one can still get an argument going among learning theorists as to whether any pure law of exercise holds at all. I don't think it's important here to decide whether learning can occur without reinforcement in the strict sense. My point is that the huge quantitative difference between pure law of exercise learning and rewarded learning leads me to think that Hebb cannot be right.

As a speculative alternative, my hunch is that in order for a pre-synaptic terminal fiber to gain a heavy increment of control over the post-synaptic cell at a given locus, the spatial-temporal condition is that the controllee cell's spike wave must be passing under the pre-synaptic locus, and hence must have been initiated by <u>other</u> inputs than the one now arriving, in order for the locus to acquire a Hebbian control increment. In other words, whether the activation is wholly by chemical transmitters or also partly by electrical effects, so far as an increment in magnitude of the control subsequently exercised at this particular locus is concerned, my guess is that this increment takes place provided the pre-synaptic spike arrival coincides with the presence of a broken-down membrane condition of the post-synaptic cell under that locus as the spike arrives.

5. I discuss these speculative suggestions here mainly to put a little flesh on the bones of my emphasis that 'hypokrisia' and 'schizotaxia' are <u>neurological</u> in their content. Hypokrisia, or some other defect in the parameters of cell control, are the first phenotypic link past the postulated biochemical aberration that is directly due to the mutated gene. It is of course technically "phenotypic," because anything outside the germ plasm is phenotypic, although I note that the geneticists are not always entirely consistent in their usage here. And, of course, ontologically speaking (rather than in terms of our knowledge at a given time), whether a postulated state or entity is phenotypic depends upon whether in the eyes of Omniscient Jones it belongs to the phenotype rather than to the chemical structure of the gene itself. And this point is relevant when we ask questions about expressivity and penetrance, because the schizogene might be completely penetrant for hypokrisia and yet there might be no molar neurological indicator which was infallible.

6. Given the schizotaxic defect determined directly by the genetic condition, the

parameters of various kinds of learning, especially social learning (because of the greater unpredictability or appetitive-aversive "mixing" that occurs in such regimes when contrasted with, say, learning to manipulate inanimate objects or abstract symbols), there develops a personality organization, the schizotypal organization. This organization is characterized by molar dispositions to cognitive slippage, anhedonia, inter-personal aversiveness ("social fear"), and ambivalence. Many other features of the schizotypal organization are described by Rado, as well as the classic descriptions found in Bleuler.

While it may be that there is an overlap between all individual molar dimensions involved in the schizotypal organizations' deviations from the norm (such that, for instance, a few non-schizotypal neurotics may have more intense or pervasive social fear than some schizotypes), I am betting that there exists a configural or even additive function of a set of indicators of schizotypal personality organization which would yield almost 100 percent separation between individuals carrying the schizogene and individuals lacking it. This prediction is not critical for what follows, but I merely record that guess here. As you know, I myself look upon the characteristic cognitive slippage as the most powerful differentiator, if we could only get adequate psychometric measures of it. And this should be expected on the above theory-sketch, since cognitive slippage is the most immediate and direct consequence of synaptic slippage, i.e., hypokrisia.

This amounts to saying that schizotaxia (a neural integrative defect) is a necessary and sufficient condition for the development of schizotypy (a personality organization) given the actual joint distribution of all other factors, genetic and environmental, that exist empirically today. I am saying that at the present time no child is exposed to an environment so pessimally "rigged" for schizotypy that he will in fact develop this personality organization if he lacks the schizogene; and similarly, that no environment or combination of other genes is so optimally "rigged" to avoid it that any child carrying the schizogene is in fact preserved from becoming schizotypic. Whether or not such non-occurrent "riggings" are conceivable is not, I think, very important at this stage of our knowledge. Needless to say, this kind of theorizing is quite unaffected by such familiar arguments as are advanced concerning single dimensions of clinical schizophrenia that may be "imitated," such as the disturbances in body-image that result in some isolation experiments, or the thought-disorder that one can develop from LSD (or even in a fever delirium). Obviously a fever delirium is not a phenocopy of clinical schizophrenia, let alone of non-disintegrated schizotypy. (I am at a loss to understand why psychiatrists and psychologists still advance such resemblances as evidence against a postulated specific etiology.)

7. Even though schizotaxia is intended to be a neurological construct and schizotypy a molar-behavioral construct, we are until further notice postulating a one-to-one correspondence in the domain of individuals between these two. However, when we pass from schizotypy to clinical schizophrenia the situation is different. Here we assume with Rado that all degrees of clinical and social health or "compensation" arise among schizotypic persons. Schizotaxia and schizotypy are thought of as genuinely taxonomic concepts, i.e., the use of a category language or of "Aristotleian thinking" is not merely an administrative or social approximation but is intended to refer to a genuine taxonomic situation in the state of Nature. But in the distinction between compensated and de-compensated schizotypy, no true objective taxonomy is demanded. (I do recall your arguments to the effect that, even here, there is some kind of "gear-shifting" or "black-and-white"

possibility, as we say the individual has shifted into a psychotic state, or has recovered from it, and I don't have any theoretical investment in denying such. I only mean to say that I do not at the moment include a positive requirement of genuine taxonomy with regard to the degree of clinical compensation.)

When a schizotype becomes decompensated to the extent that he is psychiatrically symptomatic but is free of either the accessory symptoms (delusions, hallucinations, catatonic posturing) or of a clinically obvious and marked degree of either inappropriate affect or thought disorder; then his symptoms are "neurotic" ("pseudo-neurotic schizophrenia," "borderline state," "ambulatory schizophrenia") or less clearly, but I think plausibly, psychopathic-like ("pseudo-psychopathic schizophrenia"). I do not share Hathaway's objections to these terms. In my opinion, Hoch has been quite clear in the matter. In a nutshell, pseudo-neurotic schizophrenia is simply schizophrenia presenting neurotic symptoms rather than obvious and continuous psychotic ones and therefore having a sizeable liability to being misdiagnosed. It's schizophrenia without the accessory symptoms, and with quantitatively reduced amount of affective inappropriateness or associative loosening; if loosening and affective inappropriateness are present, we ordinarily would make the diagnosis of "schizophrenia simplex."

8. By analogy with cardiac decompensation (which is of course where Rado got the term) we are not committed to any particular view as to reversibility, social or clinical recovery, and so forth. Bleuler says that after studying thousands of schizophrenics (and hundreds of them over periods of several decades) he does not believe he ever saw a single patient that he clearly recognized as schizophrenic when first diagnosed who subsequently remitted with a complete restitutio ad integrum. This is my hunch too, but, again, I don't have any theoretical investment in maintaining it. All I do maintain is that a clinical restitutio ad integrum would still leave the patient psychologically schizotypic in personality make-up.

9. The degrees and the directions of clinical decompensation, while of great administrative importance, and also interesting psychodynamically, are not of concern here. I agree with Bleuler that the heterogeneity of the symptom picture tells us little or nothing as to the existence of a specific etiology, or even, for that matter, of a "disease entity," and it is surprising that physicians (who, after all, should be familiar with numerous analogous examples in other branches of medicine) have continued to advance this fact of clinical heterogeneity as strong evidence against a unitary conception of the disease. The classic example is syphilis, but there are many others. As to the degree of decompensation, if the primary symptoms are quantitatively great enough (e. g., grossly flattened affect or complete withdrawal into fantasy or marked schizophasic speech) we consider the patient psychotic and drop the "pseudo-neurotic" from the diagnostic label. Decompensation to a psychotic degree is what Rado calls disintegrated schizotypy. Finally, long-term continuation in a disintegrated decompensated state with certain associated characteristic features such as carelessness about the person, open masturbation and the like (the so-called 'chronic-burnt-out' back ward state hospital syndrome) we call "deteriorated schizotype." But for my purposes none of these distinctions is of theoretical importance. Yet for research purposes they have to be worried about, because the various degrees and durations of decompensation may very well influence some of the nuisance variables such as test cooperativeness or even arousal (if arousal is a nuisance variable rather than the essence of schizotaxia itself!) and

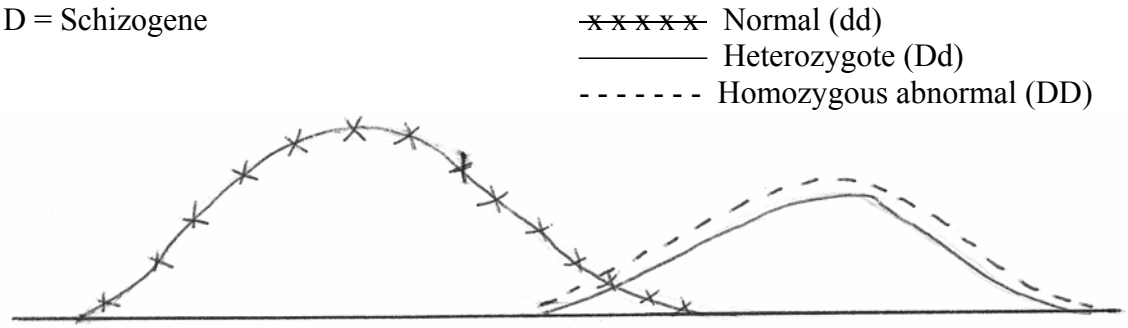
consequently methodologically they must be taken into account.

10. I am therefore postulating that clinical schizophrenia (under which I include pseudo-neurotic as well as disintegrated degrees of decompensation) has a specific etiology, a sine qua non, a condition which is necessary but not sufficient for the production of the disease. This specific etiology I postulate to be a dominant gene, the 'schizogene.' Nobody can get schizophrenia if he lacks the schizogene, nobody in fact avoids schizotypy if he carries the schizogene, but many people — I postulate most, well over half — who carry the schizogene avoid schizophrenia, i.e., they remain compensated or at lease semi-compensated and at no point become psychiatrically diagnosable even by the currently enlarged standards of American psychiatry for the use of the schizophrenia label.

11. As I pointed out to Auke in a recent memo, after failing to get clear in the head about penetrance and expressivity from genetics texts or conversations with our local geneticists, I decided to revamp these concepts along lines that make sense to me and which I believe are methodologically more precise. I take expressivity as being the more fundamental concept and one which is non-arbitrary within a given population and environment; whereas I view penetrance as having an arbitrary character.

Consider a population with a fixed joint distribution of environmental parameters (not a constant environment, which is useless for our purposes, but one in which the joint distribution of all relevant environmental parameters is taken as fixed). We also assume a fixed joint distribution of genes other than the gene of interest, i.e., a certain joint distribution of values at all genic loci (the so-called "genetic background").

Let x be an indicator-variable regardless of where it is in the organism or where it occurs in the phenotypic causal chain. If it is to function as an indicator-variable in the specified population and environment, then its heterozygote distribution is like that of individuals homozygous for the mutation. The situation for a complete dominance affair is then like this:



That is, the concept 'dominance' requires for its explanation to be tied to a particular indicator. Since most, if not all, genes are pleiotropic, the dominance concept would seem to have to be refurbished so that a given mutated gene can be dominant with respect to one indicator, incompletely dominant with respect to another indicator, and so forth. As I am here interpreting complete dominance, what it means is that the frequency distribution of indicator x for the population of homozygously dominant individuals is indistinguishable from the frequency distribution of the indicator x for heterozygous individuals

whereas both of these curves differ from the frequency distribution of x for the population of homozygous recessive individuals.

The indicator distribution arises (i.e., the variable shows variance) because of the fact that neither the environmental parameters nor the genic background are taken as fixed, but rather their distribution is taken as fixed. Because, after all, if the genic background of all homozygous individuals were identical and the environmental variables were literally identical then there wouldn't be any source of variation left and the distributions shown on the graph would consist of two vertical lines.

These frequency functions I refer to simply as 'indicator-functions.'

Now, since the schizogene (rather than its normal allele) is the condition of interest, its indicator-function I call an 'expressivity-function.' It seems to me that the core idea of expressivity in genetics lies here. For a fixed distribution of genic background and environmental variables, the two or more indicator-functions for a given indicator are determined as a question of fact and there is nothing arbitrary about the indicator-functions or the expressivity function in particular. That is to say, they are what they in fact are, regardless of any convention we may adopt. However, when a geneticist speaks of 'penetrance' and defines it in terms of the proportion of individuals displaying the genotype in their phenotype, this seems to me to have a basic element of arbitrariness. When a geneticist speaks of the abnormal phenotypic trait "manifesting itself," he obviously has in mind that something manifests itself that does not manifest itself in the normal specimen. Strictly speaking, that ought to mean that in the case of continuous indicators (and almost all indicators are continuous if you bother sufficiently to refine the measurements!) we have to establish a cutting score which lies above the highest empirically occurring instance among the genetically normal individuals. That is to say, the penetrance coefficient with regard to a given indicator is the proportion of the area under the expressivity function above the highest occurring value of the homozygously normal indicator-function.

So what I'm saying is that there is a problem of how many (if any) false positives are permitted, which must be settled by some kind of a convention before the penetrance of the gene with regard to indicator x has an empirically definite answer. Of course, there is one non-arbitrary circumstance, namely, when there is zero overlap between the two indicator-functions, in which case we get 100% penetrance by locating the cut anywhere in the unoccupied gap between the two distributions.

12. If several indicators are available, some further convention becomes necessary to decide on the meaning of 'penetrance,' because we have to decide whether we are going to construct a composite expressivity function, a kind of joint indicator (including one that allows for configural possibilities) and then determine a cutting score on it which yields zero incidence of false positives among the homozygous normals; or whether we instead adopt some other kind of model, such as a disjunctive model, in which we say that the penetrance coefficient is the proportion of individuals who fall above the cut on any of a set of indicators when the cut on each has been determined so as to yield zero false-positives. I have not been able to find out whether geneticists think along these lines or not, but I am inclined to think not, and to advocate that they should.

13. So that what my former statement that the schizogene is dominant seems to come to is simply that the indicator variables affected by it are not significantly affected by

whether the gene is present homozygously or heteozygously. I don't really know that I need this strong a demand for present purposes. What would seem to be more important is to say that it is not recessive, that is, to say that in order for the gene to manifest itself, it is not necessary for the individual to be homozygously schizogenic.

14. Another mix-up which I am not clear about in genetics is the extent to which concepts like penetrance are intended as epistemic or ontological? Sometimes it sounds as if the geneticist wants to talk about the present state of his knowledge, other times it sounds as though he wants to talk about the state of nature, including those aspects of it which he doesn't happen to know much about at present. Personally I would think it desirable to use both the concepts of expressivity and penetrance in an ontological rather than in an epistemological sense. If I find a perfect indicator of the gout-predisposing gene, do I want to assert that "50 years ago this gene had low penetrance"? That's a very inconvenient convention to adopt, especially in the context of discovery. Normal scientific usage would be to say "50 years ago the gene was completely penetrant for uric acid titer, as it is today; but no one had thought of using that indicator at that time."

15. What does it mean to say that all schizotaxic individuals become schizotypic? The best I can do by way of spelling this out is to say that I am guessing that there is a set of dimensions of the adult acculturated personality which, while they may singly show some overlap with the distribution of non-schizotaxic individuals, collectively define a region in the personality trait space, a "blob" or "swarm" of individuals who are completely separable (by drawing a surface through that hyperspace) from the entire swarm of individuals who are not schizotaxic. I don't pre-judge the possibility that there may even be some single personality or neurological or cognitive variables on which schizotaxic and non-schizotaxic are completely separated; but I do not want to require that, preferring a weaker postulate that the "type" in the sense of the joint distribution of the variables be distinctly separated in the space.

16. Since the word 'modifier' is used by geneticists to refer to a gene that alters the expression of the gene of interest, I need some term to refer to genes that do not directly do this, but which do alter the probability that an individual who is schizotypic decompensates to the point that he would be, if subjected to ordinary clinical psychiatric study, diagnosable as a schizophrenic (pseudo-neurotic or psychotic). For this I use the word 'potentiator.' 'Potentiator' refers to a gene influencing clinical status.

17. I can readily think, without any trouble at all (so I am sure the list is incomplete) of nine classes of potentiating factors which should theoretically be expected to alter the probabilities of decompensation in schizotypes. For all of these we have at least some evidence (in humans, or animals, or both) that they are considerably influenced by genes. Indicating each of them together with the direction of deviation on each of which would be expected to raise the odds of decompensating (or the direction, severity, and reversibility of decompensation!), I list:

a. anxiety-parameter (high)

b. rage-parameter (high, low?)

c. arousal-parameter (high, low?)

d. sex drive (high, low?)

e. hedonic potential [Olds(+) center density?] (low)

f. energy level (low)

g. polymorph-perverse erotic parameters (high)

h. primary introversion (high)

i. mesomorphic toughness (low)

j. ability variables (e.g., intelligence) (low)

As you see, I suggest that on some of these variables an extreme variation either at the high or the low end seems likely to be a potentiator in the direction of decompensation. One might also raise the question of whether there is some kind of inter-personal dominance factor (such as Lindzey finds in the mouse) that is distinguishable from either rage-parameter or energy level or mesomorphic toughness or all these taken collectively. I suspect not, but if there is some kind of "dominance of other organisms" that exists over-and-above rage + energy + mesamorphy, it would have to be included in the list because low value of it is presumably a potentiator of decompensation. Another potentiator might be n Affiliation, if it is different (genetically) from primary social introversion. I believe there do exist, phenotypically, as adults, cases with high affiliative need plus high social fear who are not schizotypic. If true, these are distinguishable dimensions, and could function as potentiators oppositely among schizotaxics. The "crusty schizoid" would have a low n Affiliation, so that his acquired interpersonal aversiveness would result in less frustration — a "contented loner" syndrome. Flanagan's (1935) factor analysis suggested a separation of "introversion" into orthogonal components of (low) Sociability and (low) Self-confidence.

18. Environmental potentiators I consider of two broad classes, first the early life history (especially in its social aspects), and the current stress factors which perhaps, rather than being called potentiators, should be called 'precipitators.'

19. As in my memo[1] to Garmezy of last spring (Meehl: March 10, 1965 — I believe you have a copy), I further assume that when we start with a decompensated proband and work backward to the parents, we are likely to be dealing with an individual who received the schizogene from one parent but his unfortunate adverse loading of genic potentiators from the other parent. The reasoning behind this was that if either parent had carried both the schizogene and a heavy loading of the potentiators, such a parent would be less adequately compensated (and in fact would be much more likely to have himself become clinically schizophrenic), and therefore in turn, from statistics we already have in the literature, not likely to have become a parent at all. Hence, the main source of the schizogene in the preceding generation when we start with schizophrenic probands would be those persons in the previous generation who remained sufficiently compensated to mate and having mated to produce offspring. (You may recall the figures which show that schizophrenics, even if they do marry, show a very material reduction in the number of children they tend to have.) And yet the proband, whom we have identified by the fact of his having decompensated, presumably carries a number of potentiators. Therefore, we infer that these potentiators will, on the average, tend to have come from the non-schizotypal parent. I will not elaborate on this argument further but refer you to the earlier memo, where I also tried to show the plausibility of assuming that there is an asymmetry between fathers and mothers in the sense that I would expect the usual pattern

[1] Posted on Meehl's web site in Unpublished Materials.

to be that of the schizo-typal mother married to the neurotic father so that the schizophrenic proband receives the schizogene from mother and the potentiators (such as an excessive anxiety-readiness and the like) from father. This is further plausible, from a social learning viewpoint, because the schizotypic mother is more likely to be malignant as a mother; and the additional likelihood that the influence of schizotypy on heterosexual aggressiveness is almost certainly considerably greater in the male than in the female (data exist on this also), so that there would be more parent-pairs in which the father is not schizoid than the other way around.

20. In my Sc^3 [shorthand "Sc-three" for Meehl's, "Schizotaxia, schizotypy, schizophrenia." *American Psychologist*, 1962, *17*, 827-838] I said that it would be necessary in order to check genetic models to have indicators that were of very high validity, and hopefully a set of indicators which would be (jointly) practically infallible. I believe now that this was a mistake on my part, although a perhaps forgivable one as being a reaction to the unsatisfactory state of affairs where we have to rely upon formal diagnoses that got recorded on some publicly available chart. It seems to me now that, while it would certainly be nice to find a couple of quasi-pathognomic indicators (and I have certainly not abandoned the idea of continuing to search for such), it should be possible to test at least the simpler genetic models by means of signs whose validity is not appreciably greater than we already can get with existing psychometrics such as MMPI. The succeeding sections of the memo deal with some of these considerations about which I have not talked to you.

21. Begin with clearcut cases of schizophrenia. A "solid-gold" schizophrenic would be, for example, a patient who had

a. History of long, continuous hospitalization with consistent diagnosis

b. Schizophrenic MMPI

c. Gross thought-disorder, evidenced by either

(1) accessory symptoms of reality-distortion (delusions, hallucinations)

(2) schizophasic speech

d. Inappropriate affect, by coarse clinical criteria. Doubtful sign implies unsuitable case.

22. Suppose we have a quantitative indicator $\underline{x}$ which we know has moderate-to-high concurrent validity against formal diagnosis as criterion. It is not required to have an accurate estimate of this concurrent validity, let alone of the sign's construct validity. All we need to know at this stage is that the concurrent validity is respectable. I would take MMPI Scale 8, or one of the currently used cognitive slippage tests, as meeting this requirement. Thus, an MMPI scale for which the average schizophrenic is at $T = 70$ is separating (equal-N) groups at a hitmax cut of +10 on controls and –10 on schizophrenics, yielding a concurrent validity hit-rate of around 84%. This is better than one needs, although not as good as one would like. [In the K-scale article, McKinley-Hathaway-Meehl report cross-validation K-corrected Scale 8 as identifying 75% of schizophrenic patients at expense of 10% false positives, a cut which would yield 82% "hits" in a population where $P = Q = 1/2$. Or we can get (cutting at $T = 70$) 59% of schizophrenics at expense of 2% false positives, a hit-rate of 78%, with equal base-rates. These data are using pretty punk diagnoses, and it would be easy to do better in any decent study today.]

23. Suppose we locate an arbitrary x-cut on the schizophrenic proband distribution. To avoid getting unreliably small tail-areas, this arbitrary cut should be between Q_1 and Q_2, closer to Q_1 because we don't want to be too high up on the non-schizotaxic curve. (Thus, if symmetry and equal variance obtained, a cut at Q_1 on the schizotaxic curve will misidentify upper 25% of normals.)

24. Suppose we assume that the indicator reflects schizotaxia rather than the psychosis. This is an idealization, always literally false, as we know. "How false" is to be investigated, whether early or late I discuss below as one of the sequential-strategy questions. Logically it doesn't matter, but more effort and time may be wasted one way or the other. Here I merely develop the idealized model on the assumption above. Then the compensated and decompensated schizotaxics are taken to have the same frequency-distribution $f_s(x)$, and any arbitrary cut x determines the same valid positive rate $p_s(x_c)$ regardless of clinical status.

25. The arbitrary cut x_c determined at, say, Q_1 of our solid-gold schizophrenics then determines an unknown latent valid-positive rate $p_s(x_c)$ which I write p_{sx} hereafter since it is taken as fixed in what follows. That is, we write, for the schizophrenic probands,

$$p_s^+ = p_{sx} = p_s(x_c)$$

26. In earlier conversations we have discussed the feasibility of running multiple-indicator studies on the non-diagnosed relatives of schizophrenic probands. Since (a) Gottesman is our local schizophrenic-twin-study expert, (b) schizophrenic twins are hard to come by, and (c) I am doggedly persisting in my dominant-gene theory until refuted, we should, I think, work on parent-pairs and siblings. Mindful of the Lykken-Meehl view of "strong hypotheses", I had hoped to come up with some specific point-predictions of indicator-frequencies (rather than weaker range-estimates or, God help us, mere "significant differences"). Perhaps it is my ignorance of the mathematics of human genetics, but at the moment I am somewhat dubious about the possibility of making such point-predictions for parent-pairs and sibs, even on the dominant model. The most I can see now is that the dominant gene theory yields a lower bound for indicator-positives among parents, given a basis for estimating the (latent) valid and false positive rates, which (lacking a huge N for applying the taxonomic method of my PR-65-2 [*Detecting latent clinical taxa by fallible quantitative indicators lacking an accepted criterion* (Report No. PR-65-2). Minneapolis: University of Minnesota, Research Laboratories of the Department of Psychiatry, 1965.]) we don't possess. If an indicator were perfectly valid for schizotaxia, we could say that at least one of every parent-pair should be positive. So that if an indicator's false negative rate (fixing a given cut) is q_s, we can infer that the observed frequency of "both-negative" parent-pairs has an upper bound = q_s^2.

27. We cannot, however, make a point-estimate unless we know the false positive rate p_n and the coefficient of assortative mating for schizotypy. On psychological grounds we can hardly take the coefficient of assortative mating for diagnosed schizophrenia as a good enough approximation to that for schizotypy. And a little rough calculation shows that the coefficient of assortative mating is not a negligible influence (unless it is very close to zero) upon the expected sign positive rates for parents (and, therefore, for siblings).

Making a series of rough but plausible assumptions, let me spin this out a bit for you. Nothing hinges upon the accuracy of the figures I shall use — the point is merely that values of this order of magnitude would louse up any point-estimates made in ignorance

of the coefficient of assortative mating.

Kallmann's value for the overall parental incidence of diagnosed schizophrenia (retrospective study, beginning with schizophrenic probands, working back to parents, and neglecting any systematic errors in case-finding) is around 10%. On a dominant gene theory, the lower bound on schizotaxia frequency among parents of schizophrenic probands is 50% (if assortative mating and/or environmental potentiation operate, the true figure will be higher than this). Hence an upper bound on the diagnosable-decompensation rate among schizotypes [= "clinical penetrance", but I prefer to avoid this locution, for reasons given elsewhere in the memo] is around 20%. (I note that this "penetrance" value is the same as Böök's for heterozygotes, based on the Swedish data.) Taking the schizophrenia-rate (USA figures) as approximately 1%, we arrive at a lower bound on the population schizotaxia-rate of around 5%, and a gene-frequency of at least .025. Thus, among all those who carry the gene, only about 1 in 5 will diagnosably decompensate if they survive the risk period; and one person in 20 carries the gene.

We do not have any accurate estimate of the assortative-mating coefficient. If I were to rely upon my clinical impressions, I would say that there is an above-chance incidence of two-schizotype marriages, at least among pseudoneurotic cases. Among some two dozen clearly schizotypic patients studied intensively, I found about a half-dozen such (= 1 in 4), and of course I did not meet all of the spouses. Kallmann's data leave us somewhat up in the air on this. He reports spouse-concordance of 2% for schizophrenia, i.e., over twice the "chance" value. However, for "schizophrenia or schizoid personality" [$\simeq$ schizotypy] he reports 5%, which does not differ from the general population frequency estimated above. About all we can say is that a negative assortative mating (such as you once argued for on plausible psychodynamic grounds) probably does not exist. My hunch is that the coefficient of assortative mating for schizotypy is significantly positive, but how large one cannot even reasonably guess. Kallmann and my clinical experience aside [Glueck, Anderson, and George Williams have the same clinical impression], we can perhaps get some further leads about the likely range from general data on assortative mating. Anastasi summarizes (in her Differential psychology) the evidence. For physical traits the coefficient averages around .25, for intelligence it is .50 [!!], for "emotional traits" (e.g., dominance, stability) it averages .15 with some negative, for "attitudes and values" it varies a lot (.20 to .70) with an average around .60. It is easy to rationalize schizotypy anywhere in the low to moderate range, and some facets of the condition would work oppositely to others. Thus, the rejectibility-readiness of schizotypic males might lead them to avoid schizotypic women as unpredictable and aversive, whereas the social introversion pattern might lead to mating because both prefer to avoid a lot of conventional social activity, meeting new people, and the like. I really don't see how one could reason a priori about the resultant of these countervailing influences.

Suppose we were to take an assortative-mating coefficient of .40 as a pretty safe upper bound for schizotypy. Then the fourfold table for the parental generation, assumeing a population frequency of 5% and taking ϕ = .64r as the equivalent phi-coefficient (Guilford Fundamental statistics) gives us the relation

$$\frac{p_{12} - p_1 p_2}{\sqrt{p_1 q_1 p_2 q_2}} = \phi = .64r$$

$$\frac{p_{12} - (.05)^2}{\sqrt{(.95)(.05)(.95)(.05)}} = (.64)(.40)$$

$$p_{12} = .0147$$

So the table in the parental generation looks like this:

	–	+	
+	.0353	.0147	.05
–	.9147	.0353	.95
	.95	.05	1.000

Hence among parent-pairs of schizotypic probands the incidence of doublets (both schizotypic) comes to

$$\frac{.0147}{1 - .9147} = 17\%$$

which ain't hay. I conclude that we cannot safely use a fraction 1/2, or the "one and only one schizotypic parent per parent pair" even as a decent approximation to estimate latent valid and false rates for indicators.

If we could do this, we could get enough equations to provide a non-ad-hoc test of the dominant model. Thus if the value 1/2 were acceptable, there are available two equations for each indicator-variable, one for the probands and one for the parents, and we write

$$p^+ \text{ (probands)} = p_s \qquad [1]$$
$$p^+ \text{ (parents)} = 1/2\ p_s + 1/2\ p_n$$

and solve for the latent false positive rate p_n. Given the estimates p_s and p_n for indicator-validity, we plug these values into other equations, namely, incidence of double-positives, double-negatives, and single-positives among parents, and among siblings.

28. All of the above reasoning is complicated by the opposition of certain obvious (but quantitatively unestimated) factors in a retrospective study, sampling aside. For example, given a certain assortative mating coefficient, the expected parental generation distribution of male, female, and doublet schizotypy will not be identical with that among parent-pairs of schizophrenic probands, since a doublet mating will presumably have less offspring; countervailing this is the psychological argument that a two-schizotype-parent-pair, when it does produce schizotaxic offspring, is more likely to push them into decompensation, hence a bias in case-finding. (This is analogous to, but not the same as, the usual "truncate selection" problem in human genetics.)

29. Can the actually obtained frequency distribution of a continuous indicator among parents be used indirectly to guesstimate some of the parameters? I am unclear about this also, but you have the memo to Garmezy that works along such lines. It would seem that an indicator which yields bimodality (if any do) on parents of probands, and with an N large enough to locate a clear "valley"'-point, something could be done along these lines. If such a distribution were examined together with one for siblings of the same probands, I suspect we have enough equations to solve for the unknowns (three, as I see it).

So, alas, all of this is pretty much up in the air until we can get some clarification from Gottesman and the geneticists.

30. I seem to have trouble conveying to the geneticists the importance of Rado's "compensation/decompensation" continuum. I must be communicating badly, since geneticists are, of course thoroughly accustomed to sub-clinical concepts generally. Whatever genetic model turns out to be correct, one thing is crystal clear to me, namely, its corroborating evidence will have to utilize valid indicators of compensated schizotypy. To me, the notion of "compensated schizotypy" is a plain fact, not a theory. I would hold it if I were a complete environmentalist as to schizophrenia. Since some schizophrenics become diagnosable at age 45, we say that

a. They are "disposed" to schizophrenia (this is a truism, unless one believes we all are equally so), but

b. At age 25, they don't have it.

I cannot believe that a "disposed" person differs in no respect, psychologically, from a "non-disposed" person [this has nothing to do with genetics]. And the "disposed-but-not-diagnosably-schizophrenic" individual is what Rado means by the "compensated schizotype." Of course what his psychological traits are cannot be inferred from the bare fact of a disposition. Here I confess reliance upon my clinical experience, which involves patients who were decompensated enough at times to seek help, but who were at other times (during therapy) clinically "well" by ordinary medical criteria. It is of course absurd to suppose that no such individuals exist outside the ken of clinicians.

It might be argued that one should concentrate wholly upon compensated cases, identified by the indicator-family from a population of, e.g., college students. The trouble here is that the base-rate would be too low, say, 5% as estimated above. Even a short indicator-battery is impracticable if you must study 20 individuals to find one schizotype. On the other hand, a crude preliminary screen of only moderate validity could be used to define a sample having larger base-rate. Thus, some 90% of CLA students have an MMPI on file in the Counseling Bureau, so use of the Psych 1-2 subject pool (and a "tight" MMPI criterion, where $p_s \ll p_n$ to counterbalance Bayesian bias in prior probability) might be feasible. Dave Campbell could help us on this, and in Berdie's new "student research" position I'm sure he would be cooperative. But the sampling problem is bad, because we could hardly expect sibs who are not in Psych 1-2 subject pool to be uniformly cooperative, and a schizotypic selective factor might operate rather strongly here.

Of course the other objection to proceeding thus is the danger of too much "conceptual drift", i.e., even a strongly clustered indicator-family needs some linkage to schizophrenia as a clinical entity.

31. In a retrospective study of parents and sibs, I think that something could perhaps be done using even a single indicator. Suppose as a first approximation (not used in any calculation) we cut the indicator-distribution at its inspectional "valley". [If there is none, the indicator is too weak. The only indicator I know of which does this is my checklist. But I have no evidence that the MMPI scale 8 (especially rank-potentiated, as described in Section 112a below) is not bimodal in any group with base-rates near 1/2, and I have some evidence to suggest that it is.] We cut at the valley merely to obtain latent valid and false positive rates which are not too different, and in a sample with base-rate near 1/2, that will do it. Point: We don't need $p_s = q_n$, but we wish to avoid $p_s \gg q_n$ or $p_s \ll q_n$,

because that leads to excessively unstable results unless N is large. Of course if N is large enough so that N_p is decently stable, variances N_{pq} are smaller if p (or q) is small. But we don't want to be monkeying with cell-tallies of 2 or 3 when doing parameter estimates.

Suppose the incidence of one-parent-Sc pairs (in the retrospectively defined population, so we bypass the problems of assortative mating and truncate selection) is P_1. And the incidence of two-parent-Sc pairs is $P_2 = 1 - P_1$. The incidence of sign-positives among these parents is:

$$p^+(\text{both}) = P_1 p_s p_n + P_2 p_S^2$$

$$p^+(\text{one}) = P_1(p_s q_n + q_s p_n) + P_2(2\, p_s q_s)$$

$$p^+(\text{neither}) = P_1 q_s q_n + P_2 q_s^2$$

This is 3 equations in 3 unknowns but they're cubics. If we were to solve for the three unknowns P_1, p_s, p_n, then I take it that the sign-positive incidence among sibships of various sizes would be computable, and a chi-square of these expected values against the sib-rates provides a non-ad-hoc test of the dominant theory.

32. The preceding has been in terms of a single indicator-variable. Matters improve some when we rely on two, three, or four indicators, because the number of patterns to examine goes up. However the sampling instability goes up also, and we run into the same problem of excessive N as in my method PR-65-2 [Meehl (1965) *Detecting latent clinical taxa by fallible quantitative indicators lacking an accepted criterion*]. As a general policy I would try to avoid using method PR-65-2 except in the Ford Sample (and possibly U. Hospital MMPI file data) because the dominant-gene theoretical model should itself provide some of the latent values (instead of having to estimate them all from manifest indicator-statistics, as in my latent taxa procedure). Thus, the base-rate is theoretically calculable for parents, sibs, offspring of schizophrenic probands. The false negative rate q_s can be estimated for any cut by determining the sign-negative rate among schizophrenics in remission. A lower bound on the false positive rate p_n can be estimated from the gene-frequency estimated above. And so forth. Perhaps I tend to exaggerate the difficulties, I don't know. But I daresay I have at least convinced you that, even with a set of strong indicators, a clear test of the dominant theory is not easy to cook up.

33. Far more serious a departure from the idealized model than the (presumably) small influence of double-schizotypic parental matings will be the influence of nuisance variables "arising from the psychosis itself," that is, when a schizotype is clinically decompensated we expect factors of non-cooperation, overt anxiety, depression, intrusion of fantasy-distractions, etc., to influence our measures of cognitive slippage, anhedonia, and even quasi-neurological indicators. Even short of such gross clinical interferences, it must be presumed that some of the genetic potentiators listed in Section 17 will exert non-negligible effects upon the indicators, and these potentiators will have lower average values, as well as different variances, among the sibs and parents than among our schizophrenic probands; since the probands are selected for having become schizophrenic, while the relatives are not so selected. I see this as the worst measurement problem we face. An important methodological point here: In choosing indicators, it is more important that they be minimally correlated with nuisance variables of decompensation than that they be super-valid for the schizogene. Because if we had two or three indicators that were little influenced by the psychosis and largely independent of each other within taxa,

they could be used to test the theory even if they were, individually, only moderately valid for schizotaxia. Whereas a highly valid indicator of the schizogene would provide a poor test if it were influenced greatly by the psychotic nuisance variables.

34. The obvious observational check here would seem to be a direct study of an indicator-candidate's relation to clinical status. A good indicator would be one which showed only slight (although perhaps statistically significant) discriminations when comparisons like the following were run on it:

a. Paranoid vs. simple vs. hebaphrenic, etc., diagnostic sub-types of schizophrenia

b. Pseudoneurotic vs. disintegrated schizophrenics

c. Recent vs. chronic schizophrenics

35. I suggest that the most powerful group to use for this purpose would be schizophrenics "in remission." In some ways, this is an ideal group because we can find patients who are unquestionably schizotypic by virtue of having a history of one or more solid-gold schizophrenic episodes, but who are currently in a compensated state. Prior to finding better indicators of a psychometric or experimental kind, these cases provide the cleanest criterion group for construct validity purposes.

36. Short of pathognomicity, an ideal indicator would be one for which the distributions of pseudoneurotics, psychotics, chronic deteriorated, and "schizophrenics-in-remission" were indistinguishable from one another.

37. Given such an indicator, an interesting distribution-test of the dominant model would be this: We draw the observed mixed frequency distribution obtained by pooling an equal number of schizophrenics-in-remission and normal controls. Then the distribution of siblings of schizophrenic probands should look just like this one.

38. Actually, the requirement that chronic, burnt-out, "deteriorated" schizophrenics should be no different in indicator-distribution, while nice if achievable, is not necessary. The main thing would be the similar x-distributions among remitted, pseudoneurotic, and recently-disintegrated patients. Because if that were approximately satisfied, we could deliberately exclude burnt-out cases from the initial set of probands.

39. The problem throughout is to have some independent basis for arguing that the sign (+) rate among the probands provides a good enough estimate of the latent valid positive rate among all schizotypes regardless of degree of compensation (i.e., that p_{sx} for all schizotypes is close to p_x^+ as observed in schizophrenics, so that the Equations at [1] nearly hold).

40. This line of thought suggests that one could save time by studying each possible, indicator on "schizophrenics-in-remission" only, and using the p^+-rate among them as probably the most accurate estimate of the unknown schizotypic valid positive rate p_s. How does this strike you?

41. There is an asymmetry involved here between the corroborative and discorroborative function of observations, and it bothers me from my Popperian standpoint. Let me smoke the opium pipe briefly. Suppose that we had such incredible good luck as to hit upon a high-validity indicator, say, post-rotatory-ethylated-past-pointing (PREPP) — a test untried in which I persist in having an irrational intuitive confidence. Then it wouldn't take more than two decent studies, involving only a moderate N, to practically

clinch a genetic-neurological interpretation of the data, if the dominant gene model were in fact correct. One study would compare solid-gold schizophrenics-in-remission with non-psychiatric controls and with psychiatric controls very carefully screened by a set of fallible exclusion tests. (It wouldn't even be necessary to employ my latent taxa method in such an instance, would it?) Suppose we found that the PREPP test is "positive" (i.e., that the sign of the change induced by alcohol ingestion is positive) in 100% of the cases of schizophrenics-in-remission, and that the frequency distribution of their quantitative scores was very similar to the distribution of pseudo-neurotic disintegrated schizophrenics, and was not correlated with whatever handy ratings or psychometrics we had to quantify "severity of clinical involvement" or with nosological subtype. We then set an upper bound allowable on the incidence of PREPP-positives among unselected non-psychiatric controls, say, around five percent as our guess as to a safe upper bound of the gene-frequency in the general population. The very fact that there was a negligible correlation with diagnostic rubric or quantitative measures of clinical status (such as anxiety, withdrawal, or depression) would render very untenable a psychodynamic interpretation in which the neurological aberration acted as a dependent variable following upon the psychological state. Further, the arbitrariness problem of a cutting score would be greatly reduced, since it would seem to be an unlikely coincidence (in terms of what Popper calls logical probability) that the cut required to identify 100% of solid-gold schizophrenics-in-remission should be exactly at algebraic zero. This is the kind of "qualitative" distinction which struck me in reading the Worcester study of ethylated post-rotatory nystagmus. However, I couldn't blame a skeptic if he stuck to his guns when confronted with the data of such a study taken by itself. But then suppose we apply this cut in classifying individuals as PREPP-positive or PREPP-negative among an unselected sample (no refusals!) of the parents and siblings of solid-gold schizophrenics, and we find that half of the siblings are PREPP-positive and that at least one member of every parent pair is PREPP-positive. If such a finding should be replicable, the case would be darn near closed, would it not?

But If we tried out the PREPP test and only part of the first study came out positive, namely that it discriminated powerfully between the active schizophrenics and controls but not between schizophrenics-in-remission and controls, we would <u>not</u> have a definitive refutation of the theory, because we are still in the process of <u>searching</u> for indicators. That is, if we get the anticipated result, we say we have strongly corroborated the theory; whereas if we don't get the anticipated result, we don't consider it a definitive refutation. From Popper's point of view such a proceeding is sinful.

42. 1 think the first thing to see about this is that since a theory is not a single proposition but a set of propositions (or, more accurately, a system of propositions, in the sense that the propositions have shared terms, hence the logicians' reference to the theoretical system as a "network") to refute a theory is from the strictly <u>logical</u> point of view to refute one or more of the distinguishable sentences or components of which it is made up. From the standpoint of the logician, a theory can be expressed as a conjunction of propositions, and a conjunction is refuted by the falsification of any one of its conjuncts, thus:

$$T = S_1 \,.\, S_2 \,.\, S_3 \,.... \, S_K$$

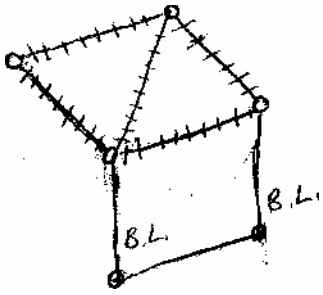
$$\therefore \sim S_1 \vee \sim S_2 \vee .... \vee \sim S_K = \sim T$$

But, as Popper points out, when a scientist refutes theory T by discorroborating even so much as one single empirical consequence that flows from its axioms as a theorem he knows that logically the theory has been falsified modus tollens; yet he does not throw up the sponge and start from scratch. After all, to have falsified a theory might mean to have falsified only one of the conjuncts, and a complicated theory composed of k propositions of which (k - 1) are true and 1 is false would be a theory that possesses a high degree of "verisimilitude." Now a problem arises in a situation such as the present one when one of the strands of the nomological net is a postulated "law" relating a theoretical construct variable to an observational variable. The current line among the boys is that, strictly speaking, what used to be called "coordinating definitions" or "bridge-laws," such as a coordinating definition relating the theoretical construct of current flow to the ammeter pointer's deflection, is actually part of the substance of the theory, witness the fact that in developed sciences the investigator or the user of an instrument is not intellectually satisfied (or even technologically relaxed) about his test situation unless the so-called coordinating definition or bridge-law can itself be demonstrated as a theorem within the network. Thus, for example, an ammeter in special physical conditions under which it is not likely to function satisfactorily (e.g., presence of an intense fluctuating electromagnetic field produced by a dynamo being operated in the basement below) can itself be included in the nomological network of theoretical physics. We don't have one kind of electromagnetic theory to deal with the phenomena under study and another special theory which deals with why our ammeters work. From that point of view, the theoretical situation in testing a genetic-neurological view of schizophrenia is simply defective in the sense that the "open concept" schizotaxia, even if filled in with my speculations about hypokrisia, does not suffice to deduce as a theorem what will be the effect of the ethyl-alcohol molecule upon such a complicated system as the kinesthetic-spatial-vestibular system (which you will recall from Rasmussen's course is one of the more complicated messes in the human CNS). Therefore, the strand in the net which links schizotaxia to the PREPP test cannot occur as a theorem but must occur in the old-fashioned (and now largely repudiated) role of a coordinating definition or a non-derivable bridge-law, and must therefore be viewed simply as one of the sentences that makes up the theoretical axiom-system. So that we are not doing anything illogical if we stick to the genetic-neurological theory, we are simply saying that we will for the time being retain all the rest of the net involved (such as the various predictions about gene frequencies in twins and siblings and so forth) but that we will consider that one strand, namely, the bridge-law to the PREPP test, to have been falsified. The trouble with this stance is not "logical" but what Popper calls "methodological." I have not had the opportunity to discuss this with him, but I am sure that from his great knowledge of the history of science he would have some helpful suggestions to make. It is obvious that it would be stupid for an investigator who is searching for indicators to drop the whole business as soon as he had tried a single indicator that didn't "work." It is equally apparent that an investigator who tried 50 or 100 indicators of a postulated "neural integrative defect," running through different kinds of perceptual and motor functions, neurological reflexes, and various kinds of cognitive tasks, and still hadn't found one, but who stuck to his guns in spite of such a long list of failures, would be proceeding irrationally.

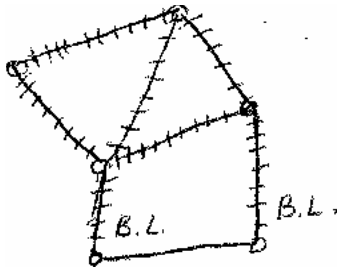
43. The situation can be expressed schematically, representing the nomological network by a network diagram in which the strands of the net represent the postulated causal relationships between theoretical entities, and the nodes of the network represent

the theoretical entities between which these relationships are postulated to hold. The fact that the nodes of the net are converged upon by several strands is the graphical equivalent of the logical fact that the theoretical terms designating theoretical entities all occur in more than one postulate when we look at it in terms of propositions. Because, of course, a set of sentences which had no such "overlap" of terms would from the standpoint of formal logic be incapable of generating any theorems, i.e., the theory would entail no empirical consequences. The following diagram shows the situation, where on the left we have the ideal set-up when a theory is sufficiently developed and explicit so that the bridge-laws to the indicators are themselves theorems; whereas on the right we have our situation, in which the bridge-law is not a theorem but may nevertheless be stipulatively included as part of the theoretical system, namely, as one of the postulates. So that in the situation on the left, the failure of an indicator to act the way it should act according the theory is a destruction of all those portions of the theory, <u>modus tollens,</u> which play a logical role in the derivation chain terminating with the bridge-law. Whereas on the right, the failure of an indicator to behave as it should behave merely refutes the conjunction of all of the sentences, and the easiest conjunct to delete from the theory is the single postulate that constitutes the (underived) bridge-law itself.

+++++++ Postulated relationship
——————— Derivable relationship (theorem)



Strong Theory: Bridge-law derivable as theorem



Weak Theory: Bridge-law not derivable as theorem, but postulated

What keeps the right-hand "weak" net from being empirically empty, a mere theorem in a formal calculus, is of course the fact that there is still an unhatched line at the bottom of the drawing, representing a theorem. Once we have postulated a bridge-law between schizotaxia as a neurological integrative defect and, say, a positive PREPP test finding on individual persons, then the genetic model implies factual consequences, namely, the occurrence of 50% positive PREPP tests among siblings of schizophrenic probands, and the occurrence of at least one PREPP positive parent in every parent-pair.

44. So the formal logic of the situation is quite clear. If the bridge-law is itself a theorem, other substantive portions of the theory have to be modified because we have a <u>modus tollens</u> falsification of the theory, and this goes beyond the merely formal truth that falsification of a conjunction means falsification of at least one of the conjuncts. Because in the case of the strong theory it means that not only is the bridge-law falsified by the observations, but at least one other statement of the theory, i.e., at least one other strand in the net, has been falsified, because the bridge-law was derivable and therefore

something besides the bridge-law has to be changed. On the right hand side, all that absolutely must be changed is the bridge-law, since if the bridge-law is false then the unhatched line (theorem) at the bottom no longer follows from the remainder of the net.

So that the problem here is not one of understanding the formal logic of the modus tollens refutation, but a "strategic" issue in methodology, namely, how far should one go in riffling through possible indicators of a neurological defect before he should face up to the fact that there probably isn't any such? And to this no rigorous answer has been given by any logician, Popper included. This is not the same kind of situation you and I complain of in much of the research of social psychology. There, they purport to be using a strong theory and then defend it by ad hoc hypotheses (that is to say, by adding strands to the net), because they want to retain the partial corroboration they get in a typical experiment and therefore they are not in a position to simply axe the bridge-law, but rather need to plug in something additional to show why some of the predictions pan out and others don't.

I talked with Feigl about this at dinner since beginning this section of the memo, and he had the following to say. He agrees with the content of the previous sections. He of course agrees that one could make two kinds of errors, both in some sense "methodologically unsound" but their conditions not rigorously specifiable. One could give up the whole network immediately upon his first failure to find a purported indicator working as predicted; or one could continue indefinitely trying one indicator after another and each time he got a modus tollens result, merely drop the postulated bridge-law to that particular indicator and consider the rest of the theory to have received no discorroboration. Either of these strategies is unsound. Feigl also had something to add to what I said in the preceding sections. He pointed out that while the bridge-law is not a deductive consequence of the rest of the theory, it isn't quite correct either to speak of it as a completely autonomous, separate postulate. Because, as he points out, there is surely some broadly "sensible" or "reasonable" basis why we try some indicators rather than others. And there is some good substantive reason, however vague and non-mathematical it may be, why we would consider it improper to retain the theory after going through a list of a hundred putative indicators without any success. After all, consider the following: An experimenter tries a dozen quasi-neurological indicators and none of them behaves in the way it should (either as a pathognomic or a fixed-probability sign). I believe we would say that he should start wondering rather seriously about whether his "neurological" interpretation of schizophrenia still remains a good horse to bet on, wouldn't we? Compare this state of affairs with that in which the investigator has the same genetic net and the same neurophysiological net, e.g., hypokrisia due to dominant gene with potentiators and so on; but he tries such indicators as art preference, political affiliation, distaste for atonal music, hair-color, and so forth. Surely no one would say that the failure of these "indicator-candidates" to pan out constitutes much of a discorroboration of the remainder of the net. From the strictly logical point of view, artistic preference or hair color are not derivable as theorems from the remainder of the net, but neither is the PREPP test. Feigl's point is that when we talk about a theory-sketch, an incomplete theory involving some open concepts, there is some kind of weak derivability, a relationship weaker than deducibility, which no one has succeeded in explicating among the logicians but which unquestionably operates in the early stages of any scientific work. It's the kind of thing that is expressed in scientific writings by such statements as, "if our theory were true, we might reasonably expect that there would be a relationship between...." And obviously

one can say something like this in connection, with schizotaxia and its possible relation to a broad class of neurological signs. It is in some (logically unsatisfactory) sense proper to say that, "If the specific etiology of schizophrenia is a gene which produces a neural integrative defect, then it is reasonable to suppose [sic!] that there should be some neurological manifestations of this, provided that the neurological test or task involved is not such as to be overly affected by other nuisance variables such as emotion, nor easily correctible by learned compensations." I don't think I can say much more about this when the logicians themselves have as yet not explicated what most of them readily admit is an important explicandum in the area of scientific methodology.

45. So, for the time being, I will speak in terms of a metatheoretical concept called by Feigl 'probability-implication'. Examples of probability-implications in social and biological science would be quasi-bridge laws like the following:

a. We might plausibly expect individuals with pituitary dysfunctions to show decreased adaptability to various kinds of biological stress, on the probability-implication that, the pituitary being the master gland, sufficiently marked pituitary abnormality should lead to a generally inadequate "endocrine integration," and therefore to reduce organismic adaptability to stress.

b. A person who has a pronounced unconscious father-surrogate complex should react more autonomically to a movie scene in which a policeman is reprimanding an automobile driver than would be true of unselected controls.

c. A pronounced preference for cats and for chow dogs over cocker spaniels should have some validity as an item reflecting n Autonomy.

d. In a mixed psychiatric population where the base-rate of schizotypy is somewhere around one-half, a striking absence of a primary or secondary SVIB pattern might plausibly be suspected to be an indicator of anhedonia.

e. If one is interested in distinguishing primary introverts from Klopfer's "burnt child" syndrome, one might plausibly expect Eckart Hess' pupillometric indicator to discriminate these when the stimulus is the recorded sound of a jolly cocktail party.

f. One might plausibly expect urethral characters to differ from controls in their autonomic response to the sound of a fire siren.

g. If white rats form Tolman-type "expectancies" with regard to relatively neutral cue-aspects of the goal box, a rat who has been thoroughly over-trained in a maze or runway situation might plausibly be expected to show less rapid and continuous ingestion of food (even under high drive) on the first occasion when, upon entering the goal box, the tactual character of the floor and the color of the walls is very different from that which has consistently prevailed during the training runs; because the disconfirmed expectancy (Festinger's "cognitive dissonance") should discombobulate the rat somewhat, and give rise to emotional respondent behavior and operant behavior of an exploratory type which will be to some degree topographically incompatible with the consumatory response to food.

It seems obvious to me that we unavoidably make use of such moderate (or even sometimes rather weak) probability-implications whenever we are dealing with open concepts embedded in a mere theory-sketch. This would be true even for an axiomatized network which is precise so far as it goes but which we nevertheless assume to be objec-

tively an incomplete postulate set because it doesn't make even a provisional claim to covering all of the likely nuisance variables that must be presumed to be operative via quantitative laws as yet unknown to us.

Adopting this position from Feigl, pending an adequate analysis of the idea of a probability-implication, about all we can expect to do in researching the open concept 'schizotaxia' is to lay down some rather general suggestions in the context of discovery for what sorts of indicators might reasonably be expected to display the postulated neural integrative defect. I am of course very much aware that here is the place that my open concept has the greatest substantive — and therefore methodological — weakness. In the following sections I allow myself a certain leeway in the use of intuition, although I am still making some intermittent attempts at tightening up the probability-implications involved.

46. The first context-of-discovery suggestion for strategy, and presumably the most scientifically defensible one at the present time, is of course to work with indicators that are similar or identical to those previously reported in the literature as discriminating schizophrenics from controls. Thus, for example, the broad domain of spatial-kinesthetic-vestibular functions is one for which we have some suggestive experimental evidence bolstered by a good deal of agreement among clinicians, that something is haywire in the schizophrenic response system where the integration of the perception of spatial depth or direction is involved, or his integration of kinesthetic feedback plays an important part, including the well-known schizoid peculiarities of body-image.

I remind myself that Joe Hunt is writing a book on the psychometrically and experimentally detectable aberrations in space experience and an associated deficit in instrumental behavior involving the perception of space; think of the studies by Cleveland and others on the distorted perception of the size of body parts by schizophrenics; of the psychoanalytic emphasis upon the disturbed ego development and Rado's emphasis upon the aberration in what he calls the perceived "action-self"; and the vestibular anomalies, whether measured by post-rotatory or post-caloric nystagmus, or by body sway, reported at Worcester; the Clark experimental psychology stuff, deriving from the developmental concepts of Heinz Werner, showing an aberration in the schizophrenic's perception of the vertical as a function of his own body tilt, where his judged vertical tends to be more like that of the child than the adult; or the claims of clinicians like Paul Schilder (in his book The Image and Representation of the Human Body), not to mention Kraepelin and Bleuler, of "soft" neurological signs such as a sub-clinical amount of past-pointing; and the strange appearance of six-neurological-content items on the empirically derived MMPI Sc key which is not attributable to a general hypochondriacal somatic-concern content since non-neurological items from Hs and Hy do not show up in anything like this proportion; putting all these things together, although some of the studies need better replication and I suppose each of them individually is capable of an alternative explanation, leads me to say that one plausible place to look for powerful indicators of the schizotaxic defect is in this general area of spatial-kinesthetic-vestibular functions.

47. A second context-of-discovery suggestion is based upon the idea that since the functioning of the CNS is certainly of a "statistical" nature, Langmuir's distinction between convergent and divergent causal series is probably of some importance here. Langmuir (the chemist) pointed out that there are some kinds of physical processes in which the influence of small aberrations or deviations tends to "wash-out" at the end of a

causal chain because the structure and constraints of the system are such that these slight deviations tend to average out rather than to accumulate and potentiate. Whereas there are other kinds of situations, such as a boulder in unstable equilibrium at the top of a mountain whose lower portions are avalanche-prone, where a slight deflection or change in the wind will be determinative of whether an avalanche occurs or not. When I think, for instance, of Hebb's view of how visual perception is acquired (even if that's been refuted, or at least in part, the example will do) I would suggest that a few added Hebbian increments in control of one neuron by another will not suffice to prevent a schizotaxic brain from ultimately forming cell assemblies and cell-assembly systems which in their molar mapping are very close to that of the normal brain. The consistent occurrence of thousands of repetitions of ocular scanning of the external world should bring it about that the visual perceptual "units" that finally get consolidated out of this mess will be the same for the normal and the schizotaxic, or so close to the same that it would be very difficult to tell them apart. For example, it might be that the average number of cells involved in a cell assembly which corresponds to a certain input element would be 10 or 15 per cent larger for the schizotaxic due to the accumulation of more Hebbian increments in fringe-elements during the pre-verbal learning process. But what difference would this be expected to make at the molar level in the thoroughly experienced organism? The "connections" are connections between distal object classes and cortical assemblies; also between an internally organized assembly and the verbal machinery. These kinds of connections will be all right, or almost all right, because the learning process will link S^D to percept, and percept to labelling, even if <u>taken internally</u> the assembly-system for percept is somewhat enlarged. (Do you follow this or am I goofy?)

On the other hand, if we have processes in which slight initial differences alter the probability of subsequent inputs or change the internal balance of feedback controls, here we might expect to have divergent causal chains, so that the end result of many trials would be quite different from that of the normal specimen.

48. Another consideration is the question whether there would be opportunities to develop compensatory mechanisms which would successfully mask the defect. I have in mind things analogous to that of the ordinary color-blind male who never finds out about his color blindness until he takes elementary psychological laboratory or tries to enlist in the Navy. If one attempted to measure hedonic potential by using a simple rating scale rather than some kind of Q-correlation or transitivity gimmick, hoping that on the average, schizotypes being anhedonic, would avoid rating described experiences as "highly pleasurable," it might not be an utter waste of time to try this simple approach, but I would bet against it on the grounds that "highly pleasurable" is a phrase that has a relative meaning, and that the schizotype simply doesn't have the same anchor points that normal people do. Similarly, while Joe Hunt alleges that you can rig up spatial perception and instrumental coordination tasks at which the schizophrenic will perform very badly in spite of being test-cooperative, we do not find that schizophrenics show a similarly <u>marked</u> degree of difficulty getting around in their ordinary physical environment. In my *Manual for the Checklist of Schizotypic Signs* [Minneapolis: University of Minnesota, Research Laboratories of the Department of Psychiatry, Report No. PR-73-5, 1964], I do point out a strange kind of disability in regard to location and object manipulation that one can, I believe, discern even in the interview, at least as an average trend, in cases known to be schizotypic on other grounds. But it is far from consistent, certainly not pathognomic as observed clinically, and I would assume that this is a molar defect where

fairly satisfactory compensatory mechanisms have been brought into play early in the developmental history so that it requires a special psychometric or experimental set-up to tap the defect sensitively enough to show more than feeble statistical trends.

49. It would seem fairly obvious that one of the ways to minimize the obfuscating effect of acquired compensatory mechanisms is to try indicators that put the patient in a really novel situation. Thus, for example, the intensity and duration of vestibular stimulation involved in studying post-rotatory nystagmus or past-pointing is extremely unusual for most people, about the only thing close to it being the rare occasions in which one rides on the devices found in an amusement park.

50. Again, I would think that the chances of gearing into a divergent rather than a convergent causal chain would be enhanced if we deal with a CNS system involving the "integrated collaboration" of several different sub-systems, having different receptor origins, requiring that exquisitely timed and finely-graded impulse patterns arrive from several different brain centers, and the like. "A complicated machine is more likely to show something wrong with it."

This might be one of the reasons why the spatial-kinesthetic-vestibular business shows up fairly well in the literature and in clinical experience. As mentioned above, the vestibular system and its relationship to the proprioceptive system was (even from the sheer neuroanatomy of the centers, nuclei, and pathways) one of the most Godawful complicated systems that we had to memorize in Rasmussen's course. Might it not be that when this great complexity, involving a system which is apparently largely wired in, rather than acquired by learning (see point above), is combined with the fact of great quantitative novelty, we get a reflection of the synaptic slippage at a molar level which it is hard to come by otherwise?

51. Another desirable, (although, as indicated above, not absolutely necessary) feature of a good indicator is that it should be relatively uninfluenced by nuisance variables that are likely to be correlates of the state of psychiatric decompensation as such. Where schizophrenia is at issue, this would mean first of all the avoidance of indicators which have the psychological character of <u>tasks</u>, because of the influence of impaired motivation to perform, as well as the probable intrusion of schizophrenic fantasy material and the like into the patient's instrumental activities in performing the tasks. This would not be a fatal flaw in an indicator if it were influenced mainly by nuisance variables relatively peculiar to schizophrenic decompensation. The trouble is that most such indicators would be affected by variables like anxiety and depression, which we already know can be considerably elevated so as to impair the test performance of non-schizoid psychiatric patients. Secondly, from the standpoint of theoretical interpretation of an indicator which seems to "work," the plausibility of the schizotaxic explanation is of course reduced if purely "psychological interferences" are available as a competing explanatory hypothesis. Of course, if an indicator <u>did</u> work well, in the sense of giving the proper number of positives among siblings and parents as predicted by the genetic model, from a Popper standpoint such a result would be more corroborative of the genetic view than of a purely social learning view, not because such a finding is incompatible with the latter, but because the latter does not make a specific point prediction of the percentages in the families and to that extent it has survived much more lenient hurdles than is true of the genetic model. So if it pans out, we would still be in good shape; the practical difficulty is mainly the likelihood that it would <u>not</u> pan out with the proper

percentages, because of a heightened incidence of false positives in non-schizoid members of the family, namely those that carried many depressive or neurotic genes in the absence of the schizogene itself.

51a. Another desirable feature, usually impossible to get in psychological test content but perhaps a bit easier to get with neurology, would be the existence of a qualitative, dichotomous, "yes-or-no" phenomenon. This is one of the things that intrigued me about the Worcester data on nystagmus, if it is true, as the authors imply in the text, but do not reveal in their graphs, that the effect of alcohol had an opposite algebraic sign in their schizophrenics from the signs it showed in the controls. The existence of such a qualitative reversal, avoiding as it does the locating of an arbitrary cut, is certainly more convincing than a procedure based upon a sliding location of a cut which is predicated upon some kind of latent model such as presented in my PR-65-2 proposal. Similarly, the kinds of perceptual "fill-in" effects we were speculating about in this connection with the stabilized retinal image would be another example of this. Or on the Rorschach it was formerly believed that the "edging" response, and the pure position-determined response, simply do no occur among either non-schizophrenic patients or normals.

52. I am conscious of the slight element of opposition, although not literal contradiction, between the aim of finding indicators that involve complex integrated cooperation of many CNS systems, such as the spatial-kinesthetic-vestibular domain; and the notion that if something like hypokrisia is involved, it would be nice to concoct some indicators that are, so to speak, relatively "pure, direct" reflections of the postulated aberration in single nerve cell functioning. For example, I kind of like the retinal image business because I think of it as getting very close to the basic neurology just because it may involve a relatively small number of neural elements or assemblies. Perhaps the idea is to look for either extreme complexity or for extreme simplicity, so long as neither is likely to be appreciably affected by learned "psychological" or "social" nuisance variables.

Thus far I have been making context-of-discovery suggestions for how to hit upon antecedently plausible indicators one by one. Although a moderate to strong validity of even a single indicator, provided it was minimally influenced by nuisance variables associated with the psychosis, would provide singly a possibility of corroborating both the genetic and the schizotaxic interpretation; it would of course be preferable to have a little family of three or four to work with. Here the main requirement is that these indicators should be relatively independent within the postulated latent taxa as argued in my PR-65-2. The context-of-discovery strategy here would seem to be one of avoiding indicators which overlap, so to speak, in their neurological or psychological content. Thus, in the broadly kinesthetic-spatial-vestibular domain, I would think the idea would be to find the best one of such a set with the intention to discard all but the most valid.

53. In general, my strategy would be to de-emphasize psychological and sociological indicators which involve as their main behavioral <u>content</u> the inter-personal traits which are developed in the course of social learnings on the basis of the schizotaxic nervous system. Because whereas clinically one is certainly struck by such important psychological characteristics as "inter-personal aversiveness," where I agree with Garmezy to such an extent that in my Sc^3 paper I included inter-personal aversiveness as one of our basic indicators of the schizotypic syndrome, I believe that the odds of being able to develop a really discriminating psychometric device to tap this facet are not good in the foreseeable future. I have, however, gone so far as to concoct a face-valid MMPI key of

inter-personal aversiveness which I hope to get some bright student to develop by a combination of criterion keying and factor analysis or homogeneity, but I don't as of now prophesy a very brilliant future for such a key. My clinical faith in interpersonal aversiveness as an indicator is based mainly upon the material one gets in intensive long-term psychotherapy of schizotypes, and (as is emphasized in the Manual on Schizotypic Signs) what is clinically impressive is the pervasiveness, the intensity, and the refractoriness of this trait. These aspects I believe can be more or less reliably judged by a perceptive clinician on the basis of extended contact with a patient, especially in regard to some of the very child-like transference phenomena. But I am far from convinced that I or anyone else can assess them in a couple of hours of mental status examination. Furthermore, when I have been able to spot it on brief contact, and corroborate the diagnosis by the subsequent appearance of more pathognomic signs such as micropsychotic episodes or anhedonia, I think I had spotted it mainly by observing the patient's schizoid-like style and manner in the interview, rather than by what he says in self-descriptive language. The self-descriptive language of inter-personal aversiveness is not in my opinion sufficiently different between schizotypes and some neurotics, not to mention garden variety non-schizoid "introverts," to make it likely that a set of MMPI-type items would do the job. So that whereas I would confidently predict that schizotypes as a group would show a more intense and a more diffuse inter-personal aversiveness than healthy people even if evaluated by means of a questionnaire or Q-sort, I would expect an excessive number of "false-positives" among neurotics.

As you know, one of the reasons why I don't find myself much interested in laboratory demonstrations of the fact that inter-personal aversiveness impairs the performance of schizophrenics is that any experiment which failed to show this would be, on the face of it, classifiable as somehow defective. That is, if I employ some kind of an experimental manipulation in the hope of arousing social fear, and then I employ some kind of performance output indicator to detect the resulting impairment in cognitive or instrumental processes, a failure to show the expected difference would surely be attributable either to the fact that my procedure was ineffective in actualizing the schizotypic disposition to social anxiety, or else that my performance indicator was insufficiently sensitive to the aversive state thereby elicited. What I am saying is that everybody who has treated schizotypic persons (or even done diagnostic examination of them) knows, beyond a shadow of a doubt, that they have an exaggerated disposition to social fear, and I can't really think that any clinical psychologist would consider this generalization to be refutable by a negative outcome of a laboratory study. From which I conclude that a Popperian view of such experiments would be somewhat negative, since if the experimenter himself "knows" in advance what the result ought to be, and could tell you in advance that he will not abandon his generalization given a negative outcome, the experiment is simply not functioning as Popperian testing. Of course this is no blanket condemnation of experimental studies on schizoid aversiveness. A study aiming to clarify the mode of its operation, or specify the cues which elicit it, or to draw the curve of its effects, is worthwhile. What I find pointless is a mere attempt to "bring schizoid aversiveness into the laboratory," in the sense of a demonstration that it exists. I know it exists, and so does everybody else. So why bother?

54. It stands to reason that the more causally remote a class of behavior dispositions is from the postulated schizotaxic effect, the greater is the prior probability that one or more nuisance variables uncorrelated with schizotaxia will generate invalid (and systematically

invalid) variance in the indicator and reduce its value for the purpose of testing a genetic model.

This policy introduces another tension in the strategy between the desirability of avoiding high level socially acquired psychological content on one side, and achieving a qualitative diversification of the indicator family on the other. Presumably from the standpoint of what would seem plausible on the schizotaxic theory, the ideal is a set of indicators each of which vary "close to the neuron" but which involve largely independent CNS systems so that only the schizogene would generate appreciable correlation among them.

55. Another strategy issue not clear to me, again I suppose because of the sketchy, open concept state of the schizotaxia theory, is the extent to which one should try to think up indicators which might be powerfully influenced by the causal convergence of two or more psychological variables, each of which originates on the basis of imposing a learning history upon the schizotaxic defect. For example, you and I have talked about studying transitivity of aversive and avoidant choice-behaviors, entertaining the notion that the "intervening" or "mediating" traits of cognitive slippage and anhedonia would yield a sizeable potentiating interaction effect. I don't see any rational way to estimate in advance whether the potentiating effect of anhedonia (working via a kind of de-differentiation or flattening of the gradients of preference) would contribute more to the validity of an indicator through potentiating the patient's basic talent for cognitive slippage, or would instead have a net effect of reducing discrimination between schizotypes and normals because of the reasonable likelihood that anhedonia itself is polygenically determined and presumably also influenced by non-schizotypic nuisance variables found in neurotics as well. Crudely speaking, if the unknown mathematical function determining preference behavior is such that average hedonic level, or the standard deviation of hedonic levels over situations, operates as a multiplier of cognitive slippage, whereas with the non-schizoid neurotic it operates linearly, one might expect the potentiating effect to pay off In improving separation of the schizoid and nonschizoid populations. But I am not sure my thinking is straight on this. We have here the familiar problem of a theory which is not rich enough quantitatively to resolve certain questions of a parametric nature.

56. In this connection, I should say a little something about my current speculations about how the intermediate psychological variables are causally related to the specific etiology on the one hand and the psychometrically or clinically observable molar output on the other. As I now think of it, the anhedonia is less fundamental than the cognitive slippage being initially a direct consequence of the synaptic slippage (hypokrisia), and the anhedonia being an acquired consequence of a "mixed" appetitive/aversive social reinforcement schedule, this imposed reinforcement schedule giving rise via hypokrisia to what I call 'aversive drift.' I am still fooling around with speculative neurophysiological accounts of this, and there are several which will do the job. I want to emphasize that in putting it this way I do not want to maintain that what is usually called 'thought disorder' is responsible for aversive drift. I don't even want to say that the molar trait 'cognitive slippage' is responsible for aversive drift. I think I can show that hypokrisia, given the mixed, reinforcement schedule which we are all put under as children and adolescents, will tend to generate aversive drift.

However, it is clinically obvious that an acculturated specimen has developed cognitive slippage at the molar level, and his inter-personal relationships will as a result of this

molar tendency show a further aversive drift which is only partly dependent on the neurological parameters, but is partly to be seen socially and psychologically. Thus, a tendency to over-perceive minimal social cues reflecting another person's latent hostility may arise from "primary aversive drift." (Roughly, this is because of an asymmetry between Olds(+) and Olds(–) feedback controls on the activation of cortical assemblies, one being "facilitative" and the other being "inhibitory." All of my speculative neurologic models make use of this idea, one way or another. Another possibility is a parametric factor in the difference between eliciting of "arousal" and "anxiety" as state-variables, but I have not as yet really worked on that one.) Once having developed such a hypersensitivity to aversive social cues in others, such that the patient begins to make erroneous inferences about the conscious malignant intentions of people in his environment, a tendency to find people mysteriously being "against him" will lead him to try to make sense of this mysterious social fact, and hence to give rise to paranoid constructions which lead him to classify a larger number of persons in social contexts as being dangerous. This in turn generates what might be called, considered by itself in relations to the patients perceptual premises, "normal anxiety." In addition to this, whether the patient handles this derivation of inter-personal anxiety by phobic avoidance, or by socially inappropriate coping mechanisms while remaining in the situation, or by counterphobic, projective, and acting-out responses, these instrumental acts provide a reinforcement schedule to other people which is shifted for <u>them</u> aversively, with the result that they begin to put the patient on a mixed regime reflecting their own resentment, puzzlement, or fear of him, so that the objective statistical properties of his social reinforcement schedule become further shifted to the aversive side. So that I distinguish between a primary aversive drift which follows neurologically from the imposition of a normal or standard social reinforcement schedule, and a secondary aversive drift which arises from a combination of his own cognitive distortions plus an objective movement of the social reinforcement schedule via social feedback effects.

56. Note again that at each step in the preceding, a complete account would have to include the influence of polygenic determiners such as the patient's own basic aggressiveness, his intelligence, his innate hedonic potential (Olds(+) center density), his anxiety threshold, and the like.

57. And, of course, the magnitude of this autocatalytic growth of aversiveness will be influenced by the extent to which significant persons react aversively to the patient's conduct. Thus, we can assume that a schizotypic mother will on the average be more angry, more frightened, and more puzzled by the social inputs she receives from a schizotaxic child than a normal (or even neurotic) mother would be. Thus, for instance, the almost universal complaint of schizophrenic patients in the anamnesis that (if there were any siblings) the mother preferred the siblings to the patient, I take for granted reflects partly (a) the patient's selective recall of the past while he reports it to the therapist, partly (b) the historical fact that as a child he over-reacted aversively to stimuli that were no different from those presented by mother to his siblings, and partly (c) that either a normal, a neurotic, or a schizotypic mother will actually respond more negatively to a schizotypic child than she will to his non-schizotypic siblings. This latter interpretation, which practically all child clinicians systematically neglect, and refuse to take seriously if you suggest it to them, is supported by a study done by Klebanoff of the child-rearing and mother-role attitudes of the mothers of schizotypic children. Using an objective structured verbal device proporting to measure the unhealthiness of mothers' attitudes to their

maternal role and toward child-rearing problems, Klebanoff found pretty sizeable differences between the mothers of schizophrenic children and the mothers of some normal controls. (In addition to its face validity, I am willing to take this finding as partial corroboration of the bridge-law asserting the "validity" of the structured instrument he was using.) These findings are of course the sort of findings that are conventionally adduced, whether based on psychometric data or clinical impressions, for the crucial causal influence of the "schizophrenogenic mother" is in the etiology of schizophrenia. However, Klebanoff had two other control groups in his study, a group of mothers whose children had suffered cerebral birth injury or other forms of early brain damage, and mothers of mental defectives. What he found was that the mothers of defective and brain-injured children showed the same "bad" maternal attitudes as did the supposedly schizophrenogenic mothers. So far as I am aware this was the first (and is still the only) study which attempts to introduce some kind of a statistical control for the backward effect upon mother of having to handle a kid who is behaviorally aberrated in some serious way.

58. I don't like to complicate things, but it seems to me rather obvious that almost all of the postulated causal arrows, once we get past the primary aversive drift and the primary cognitive slippage, will tend to run in both directions. Everybody knows that the emergency emotions of fear and rage do not conduce to thinking straight or perceiving accurately or remembering well in anybody, normal or abnormal. So that just as we have a secondary aversive drift which is potentiated by cognitive slippage, so we have a secondary cognitive slippage which is potentiated by aversive drift. It seems pretty obvious to me from my experience treating schizophrenics that on the one hand the patient begins to distort reality when he becomes angry or afraid, but on the other hand he also tends to become angry or afraid due to this distortions of reality. And in the therapeutic handling of micro psychotic episodes, I have been unable to decide at times whether my role as emotional supporter was more or less important — at least in the short run — than my role as a reality-tester.

59. With regard to anhedonia, if we adopt Rado's revised view that one does see some markedly anhedonic individuals, especially among compulsive-obsessives and among some essential sociopaths, and even — as I believe — among people who are not psychiatrically diagnosible at all, that is, they are just people who don't get a big kick out of life; I would think of this as being almost certainly a polygenically determined business arising from some quantitative deviation in the micro anatomy or neurochemistry of the Olds(+) centers. For ease of reference I have been referring to it above as if we knew it was literally a reduction in the density of Olds(+) spots. This then is a neurological dimension which would presumably show the usual wide range of individual differences in a population of organisms, quite apart from the schizotaxic gene, which I'm assuming is unrelated to it. But since anhedonia stands out so strongly in schizotypic individuals, I postulate that it acts as one of the important potentiators toward clinical decompensation among schizotypes. Or perhaps I needn't say I postulate it, all I need to postulate (which as a follower of Roger Williams I am sure you are willing to assume with me) is the existence of marked individual differences, genetically determined, in the Olds(+) centers. After all, we have very good experimental evidence right now for saying that there are such centers, and that's all I personally need to feel confident that a range of such exists, and being the kind of thing it is, genetic differences must contribute most of this variance. Taking phenomenal pleasure as the inner correlate [mind-body problem!] of the positive

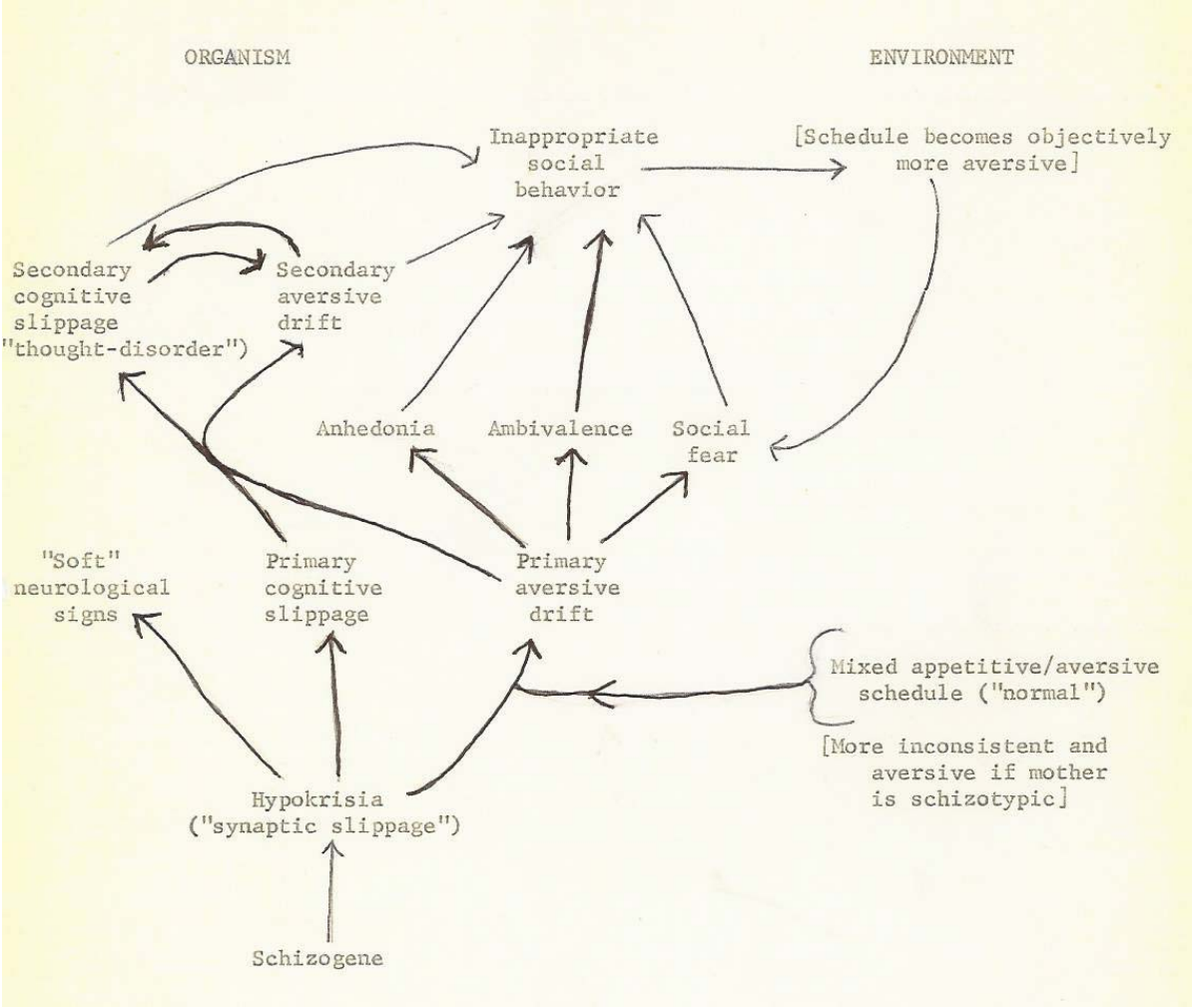
reinforcement parameter, and the obvious truth that organisms in Skinner boxes or mazes reveal wide individual differences in the reinforcement parameter, it seems pretty safe to conclude that any behavior disorder which involves marked differences in aversive and appetitive control as part of its pathology must inevitably be potentiated in a specimen whose hedonic potential falls genetically at the extreme low end of the Olds(+) center continuum. So in a way I am not adding a new postulate but taking over received knowledge as of this date and deriving the important role of primary hedonic deficit in the potentiation of schizophrenia among individuals carrying the schizogene.

However, the more I think about my clinical experience the more I lean to B. C. Glueck's view that the marked schizotypic anhedonia is a psychologically derivative phenomenon, namely, that it would not arise to the extent it does without aversive drift. In spite of my bias against etiological theories emphasizing social learning factors, I think I have to admit that my own clinical experience to date suggests that the most striking and consistent molar anhedonia among schizotypes is manifested in regard to inter-personal relations, and becomes considerably less obvious, and in some clearly schizotypic patients really not discernible at all, as we deal with life areas in which inter-personal aversiveness is at a minimum. However, I have to qualify this by saying that I am here considering self-esteem, pleasure in success, or "ego-function gratification," like Woodworth's old "mastery-motive," as being very intimately related (due to the learning history) to inter-personal factors. That is to say, if I experience gratification of my mastery motive and an enhanced self-esteem by completing a crossword puzzle which I have no intention whatever of showing to anyone else, I don't believe this type of self-reinforcement would be possible without some kind of implicit social baseline in the background. Would you agree with this? With that qualification, I think I would be willing to say, pending more quantitative research on the point, that anhedonia is a nearly pathognomonic exclusion test (<u>not</u>, as mentioned above, an inclusion test) for schizotypy, provided that the life domain considered is inter-personal <u>or historically derived from inter-personal satisfactions</u>. Whereas if the self-judging kind or issue can "be kept out of mind by the patient, I am inclined to think that many schizotypes, including some decompensated cases, can enjoy non-personal pleasures almost as well as anybody else can. (Of course we would still expect a slight <u>average</u> difference between decompensated cases and controls, because we assume that the decompensated case has probably, on the average, become so partly due to polygenic determiners placing him low on the primary hedonic continuum, aversive drift aside.) I have in mind "kicks" received from music, art, the intellectual satisfaction, of science, auto-erotic gratifications such as eating, drinking, masturbation, simple sensory pleasures of touch and smell, non-threatening pseudo-social objects such as pets and young children, and the like. I have, for example, been impressed with the fact that some of my patients who were unmistakably schizotypic, to the point that they had micro-psychotic episodes with florid delusions or even hallucinations, were able to get on rather well with other people's children, although typically not with their own, because the emotional demands of parenthood and the self-concept "I am a bad parent" seemed to get more in the way when their own children were involved. One might also mention as at least consistent with this interpretation the occurrence of "ecstatic" states in some catatonic cases, and in a few hebephrenic patients, described at length by Bleuler, especially in his <u>Textbook of Psychiatry</u> more than his book on schizophrenia. These ecstatic states are of the "gear-shifting" variety you have pointed out in connection with panic and depression episodes, and without extended fruitless speculating on the

neurology of it, my hunch would be that the synaptic slippage enables at least some schizophrenic patients to achieve a dissociation of systems (more analogous to the massive dissociation in classical hysteria or in the so-called hysterical psychosis than to the usual fragmented or scrambled splitting that Bleuler had in mind in inventing the term 'schizophrenia'). If the patient using his slippage talents, can achieve sufficient massive denial of his aversive social input, and somehow "turn off" the inner reverberatory circuits involving aversive centers, he is able to generate an autonomous auto-catalytic process of "positive" percepts which are regnant currently with a steady state of Olds(+) activation.

I also suggest that the strange kind of "wearing out" of positive cathexis which we observe so frequently in these cases may be hard to understand in terms of primary anhedonia alone, whereas it is rather easy to understand if we include the factor of aversive drift, in the sense that initially positive systems begin to be drawn into the aversive ambit so that everything becomes gradually "poisoned" by aversive associations and perceptions.

60. I would summarize the causal-developmental situation laid out in the preceding sections by the following diagram:



61. To avoid complicating the diagram, I have omitted many causal arrows which I have no doubt are operative but which are common to schizotypes, neurotics, and normals. For example, the interpersonal aversiveness and inappropriate social behavior (with resulting change in the objective social feedback regime) will, on the average, give rise to objective deprivations of motives not perhaps very different in schizoid and normal persons. We know, from clinical experience and from statistical studies, that the premorbid case shows a marked decrease in heterosexual aggressiveness, presumably because of social fear and anhedonia for the presexual social preliminaries of courtship behavior. This in turn generates frustration, and on the frustration-aggression hypothesis, a building up of hostility toward the depriving world. The hostility in turn arouses hostile-rejective social feedback, and also is internally cued to anxiety (fear of one's own destructive impulses). Reaction-formations develop similar to those typically found in non-schizoid obsessives, such that the patient is so afraid of his own latent rage that he constrictively controls social and sexual behaviors which would really fall within the acceptable range of normal, healthy self-assertion. This in turn leads to the development of a self-concept of weakness and incompetence to handle competitive situations, and so forth. So far as I know, there is hardly a single "neurotic" mechanism which cannot be employed by the schizotype in one or another life domain. There is no point in working on these as indicators because they are surely non-specific, although no doubt some of them are quantitatively exaggerated because the aversive drift implies that all cognitive or instrumental responses which are primarily anxiety-based will be exaggerated in schizotypes. And of course the cognitive slippage is constantly operative to interfere with self-corrective ego-talk of the kind which many neurotics do employ moderately successfully to keep their behavior within socially acceptable limits.

62. Pulling together the preceding sections, here is a set of rough criteria for selecting indicator-candidates, based on the double aim of (a) Likely to pan out if the theory has verisimilitude, and (b) Having a "probability-implication" as bridge-laws, such that a run of failures should (a la Feigl's line) be taken as discorroboration of the schizotaxic theory itself.

a. Sophisticated, clinical experience, especially the "old masters" (e.g., Bleuler).

b. Previous research on schizophrenia, provided trends are large rather than the usual mere "significant at .01 level" stuff, and have replicated reasonably well, as these things go.

c. Likely to behave like Langmuir's divergent chains rather than convergent, given an initial slight aberration in neuron control-parameters.

d. Opportunity to learn compensatory mechanisms limited. Hence,

e. Novelty of the indicator situation,

f. Molar disposition either very "simple," a direct, wired-in connection to small-scale CNS processes, or, at other extreme,

g. Molar disposition very "complex," involving closely timed and finely graded multiple controls from several anatomically remote centers. (An "integrative defect" is best detected by studying functions where there is a lot of "integrating" to do).

h. Relatively uninfluenced by nuisance variables reflecting the psychosis itself,

i. Qualitative, dichotomous, "Yes-or-No" effects avoiding arbitrary cutting-score problem. Or, if quantitative,

j. Reversal of algebraic sign of an effect, or opposite signs for two effects.

k. Avoid psychological-social indicators as non-specific.

l. Avoid indicators reflecting neurotic mechanisms.

m. Avoid "tasks" where non-specific nuisance variables of motivation, etc., contribute heavily.

n. Minimize links in postulated causal chain between hypokrisia and the molar disposition.

o. If not nuisance-influenced, try indicators which should show interaction between primary aversive drift, anhedonia, and cognitive slippage, such that some schizotypic traits potentiate the effect of others.

63. There are several different ways to classify the possible indicators, such as degree of molarity versus molecularity, kind of instrument employed, which genotypic trait is being tapped, and so on. I don't think it makes much difference how one slices this pie so long as he uses a classification that covers the waterfront. I am for the moment classifying indicators with reference to the alleged genotypic trait, as follows:

A. Neurological and quasi-neurological (skeletal system)

B. Cognitive slippage (including perceptual, thinking, verbal-associative, body-image, para-causality, etc.)

C. Anhedonia and ambivalence

D. Interpersonal aversiveness

E. Self-concept

F. Autonomic

G. Direct biochemical

H. Somatic (non-behavioral)

I. Miscellaneous

64. You will note that I have not included impaired performance in experimental settings or on tests except when this reflects cognitive slippage, because this is the kind of thing which is already known to be markedly influenced by nuisance variables non-specific to schizotypy, such as depression and anxiety. I don't see any point in searching for indicators in that area because although there might be a few, the odds are so much against it that you would have to run through scores or hundreds before you hit upon one that was sufficiently specific to schizotypy.

65. I also have not included history items of the kind involved in premorbidity scales, because surely they are non-specific, as are history items about underachievement, early phobic and withdrawal patterns, and characteristics not of the patient himself but of the family constellation.

66. To save space and for ease of dictation, if I cite a published study in relation to a possible indicator, I will merely cite author and year, and this means that the study is to

be found in the collection of reprints I have in the office file that bear directly or indirectly upon schizophrenia.

Some Possible Indicators

A. Neurological and Quasi-neurological

67. The vaguely delimited class of possible indicators that I have referred to previously as "spatial-kinesthetic-vestibular," because the experimental procedures are usually such that there is no way to tease apart the effect of these three components on the output variables. For examples see the references in my Sc^3 reprint, Angyal and Blackman (1940), Angyal and Blackman (1941), Angyal and Sherman (1942), Colbert and Koegler (1959), Freeman and Rodnick (1942), Leach (1960), Pollack and Krieger (1958). In his summary of the Worcester long-term project The Biology of Schizophrenia, Hoskins (1946) concludes that one of the few findings that seemed to emerge as consistent and quantitatively respectable was that there is something funny about the vestibular reaction system of schizophrenics. However, as you have pointed out, even if these results are satisfactorily replicable — and they seem to have held up on the studies done since then — we really have no good basis for identifying it specifically as "vestibular," since the influence of mental set, current inner-psychological activity, and especially the arousal variable, may be responsible for the effects. The differences in nuisance variables connected with the anatomy of the ear rather than with the CNS itself lead me to think that if nystagmus or body sway is used as an output variable, rotatory stimulation is probably a better procedure than the caloric technique. Janacek and Powers used the caloric method. But I don't think we are going to get much from that pilot study in replication, because Janacek did not succeed in getting enough control cases where we could be confident that we were not dealing with a pseudo-neurotic. (I made the Meehl error again on this deal!)

68. I am still interested in the effect of alcohol on post-rotatory past-pointing, and if Ray Johnson is getting any results with semi-compensated cases or cases in remission, I think the PREPP test would be worth a try. You recall my idea was to have a target on the wall to which the patient has to point (while blindfolded) after rotatory stimulation, the "pointing" being done by something like a small flashlight attached to his forefinger, so that a relatively slight aberration in the amount of past-pointing would be, so to speak, "amplified" without instrumentation but simply by virtue of the trigonometry, and a patient provides his own control being tested both with and without a pretest alcohol ingestion. I am betting that the "paradoxical" effects of alcohol (Worcester studies) will show up with past-pointing better than they did with nystagmus.

69. Another possibility in this general area is the Beritashvili "passive transport" situation, in which a blindfolded subject is required to retain his spatial orientation with regard to a target object after being passively transported in a complicated path around a good-sized room.

70. Still another might be the effect of rotation upon various kinds of motor performances involving speed and accuracy of eye-hand coordination, perhaps again introducing alcohol as a potentiator. I don't like this quite as well because King's book seems to suggest a considerable influence of nuisance variables associated with psycho-neurosis as

well as schizophrenia, even though the schizophrenics show more pronounced deviations than the neurotics.

71. Of course, if the data Ralph Miller is collecting on Ray Johnson's subjects look as if the spatial-kinesthetic-vestibular aberration is largely or wholly attributable to the arousal factor, there would seem to be no point in a strategy emphasizing the s.k.v. phenomena, but instead one should concentrate directly upon the arousal variable itself.

72. Another s.k.v. indicator is child-like aberrations in the perception of the vertical induced by body tilt (Wapner, et al., 1957). I believe their stuff involved a potentiation by the use of LSD-25. My recollection is that Witkin has also reported this kind of phenomenon, but I don't seem to have any reprints of Witkin's work.

73. Another possible s.k.v.-indicator is the so-called "kinesthetic figural-after-effect," which was invented by analogy with the visual figural after-effect studied by the Gestalters. Three investigators allegedly found fairly large differences, claimed good enough to be worth using in terms of group separation, in studies by Silverman (1964), Petrie (1963), Michael Wertheimer and Jackson (1957). Unfortunately, I only have a reprint of the Silverman thing since Wertheimer's supply of reprints is exhausted. The basic process here involves something that appears to be very simple from the standpoint of apparatus and would not likely be much contaminated by nuisance variables like task anxiety, namely that the patient's estimate of the size of a test block from proprioceptive + tactual input (he feels the block) is influenced by an interpolated stroking of another block. Petrie classes people into "Augmenters" and "Reducers," alleges a typology. I expected schizophrenics to be augmenters, but she says they are reducers. However, she thinks they become reducers as a defense against their own original augmenter tendency. If so, this is not a good indicator to try, since the compensated schizotypes might be augmenters and the probands reducers, a messy setup to interpret. Interestingly, she reports that alcohol makes augmenters reduce and ~~reducers augment~~, reminiscent of the Worcester stuff again.

Of course this kind of stuff could also be classified under the heading of cognitive slippage, but not knowing where to put it I am putting all of these peculiar things about information from muscles affecting judgments and the like under the neurological heading.

74. A couple of other s.k.v. possibilities each of which has been alleged to discriminate schizophrenics, but not impressively, is the face-hand test (Pollack and Goldfarb, 1957) and the finger localization test (Goodstein, 1957). Each of these again has the advantage of not involving any great task threat and of being free of the necessity for complicated apparatus.

75. I will simply list here under this one heading some borderline possibilities in the s.k.v. domain, borderline not because they have less a priori likelihood of panning out as indicators of whatever is haywire in this functional system, but merely because they have not, to my knowledge, been investigated in relationship to schizophrenia. We have the effect of vestibular stimulation on the subject's perception of the median plane (Morant, 1959), the effect of muscular involvement on visual threshold (Wapner, et al., 1958), the effect of muscular involvement on perceived size of rods (Wapner, et al., 1962), the influence of body tilt on the subject's perception of the relative locations of external objects versus his own body (Wapner, et al., 1962). A Wapner student (Comalli, 1963)

has published an interesting short paper on the effect of body postion on the subject's eye line localization. This, unlike the body tilt stuff, does not require much of any apparatus, since Comalli simply had the subject judge the position of a line as being at eye level from two positions, one standing up and one lying down. I will bet you even money that schizotaxic people perform deviantly on this simple little task.

76. Leaving the s.k.v. domain specifically, there is always the possibility that one or more traditional signs employed clinically by the neurologist, if quantified and souped up by instrumentation plus statistical treatment, would reflect the schizotaxic integrative deficit. I have not checked with Manny Meier, but I can recall (back in the days when I was working on the Neurology Service under Abe Baker) reading articles in which psychologists or physiologists had made common neurological phenomena more powerfully diagnostic, especially for sub-clinical neurological conditions such as a very early multiple sclerosis or Wilson's disease, by a combination of experimental control, recording instrumentation, and quantitative treatment of the output. As I pointed out in my APA Sc^3 speech, I am convinced that a psychotherapist who keeps his eyes and ears open and is halfway receptive to such data will, over the course of any prolonged series of interviews with a schizotypic patient, have occasion to note one or more — sometimes many! — "soft" neurological signs. So that even those kinds of neurological dispositions which are generally considered to be "clear" in schizophrenia as a functional disorder might quite possibly turn out as valid indicators if they were subjected to more precise methods of observation or various potentiating techniques of elicitation. Dr. McKinley used to think that quite a few schizophrenics, for instance, display a mild, neurologically "plus-minus" tendency to dysdiadochokinesis, and I remember his warning us in neurology rounds that one can at times falsely suspect cerebellar lesions or lesions elsewhere in the related systems on the grounds of marginal dysdiadochokinesis in a patient who is merely schizophrenic. I seem to remember that some psychologist working with a neurologist showed, for example, that if you study dysdiadochokinesis by using a simple machine with a couple of handles on it instead of merely visually inspecting the patient as he pronates and supinates the closed fist or the flattened hand, there are quantitative aberrations that are not normally detectable by clinical observation of the usual neurological type but which will show up cerebellar disease when treated quantitatively. I would suppose that the chances are better of picking up a schizotaxic indicator in those areas of neurology which emphasize "integration" and "coordination" of functions as contrasted with those which merely involve things like amplitude or latency of a tendon reflex, and the like. Who knows whether even such things as the Romberg sign might not be schizotzxic indicators provided that the behavior sample is extended sufficiently and that sensitive measures of detecting body sway are employed?

And of course any of these indicators might profitably be studied in reference to a chemical potentiator such as LSD or Stelazine or alcohol.

77. Finally, I suppose it is not utterly out of the question that a carefully constructed neurological self-reporting of symptoms inventory might do a creditable job as an indicator. I wouldn't have any faith at all in this if it were not for the appearance of those neurology items on the schizophrenia key of the MMPI which, as I may have mentioned previously in this memo, used to be a source of embarrassment for us loyal Multiphasikers but which now, of course, I receive with delight. The MMPI items which I take to be reflecting the patient's awareness that at times his neurology is not intact are the following: A-41, "I have never been paralyzed or had any unusual weakness of any of my

muscles" (F); A-42, "I have had no difficulty in keeping my balance in walking" (F); A-43, "My hands have not become clumsy or awkward" (F); A-44, "I have little or no trouble with my muscles twitching or jumping" (F); A-48, "I have numbness in one or more regions of my skin" (T); B-1, "Once a week or oftener I suddenly feel hot all over without apparent cause" (T).[2] I have come across repeated instances of such complaints, usually having a very transitory nature, in the course of extended therapy with schizotypes, and it is my impression that these are not functioning in the hysterical mode with secondary gain and the like. I say this partly because the patient has what I would consider normal anxiety over them, and does not in my opinion utilize them in the way a conversion symptom is utilized. Now of course it's only six items, so I am not going to make a Federal case about it. However, it is important to notice that there are only fifteen "neurological" items in the MMPI pool so that picking up six of them in the empirical analysis that was used in constructing Scale 8 is a pretty suggestive finding when one considers it against the related fact that non-neurological somatic complaint items (e.g., GI tract, cardiorespiratory) are present in considerably larger numbers but are almost devoid of representation in the schizophrenia scale keying. So that looks good enough for me to wonder whether it might be worthwhile at least putting a master's candidate or part of the time of a research assistant on picking up some results on a face-valid neurological complaint scale (presumably with adequate buffer items to make it subtle). Or maybe one doesn't need subtlety, perhaps what we should do is to construct a longer list of neurological items, especially of "soft" variety, and use a discrepancy score between the soft neurological items and non-neurological somatic items of the kind found on scales Hs and Hy. This would presumably contain in its psychometric structure a more or less automatic suppressor variable with regard to the nuisance variable of "general somatic concern." As you know, I myself am of the opinion that the textbook hardcore gilt-edged hypochondriac is more often than not actually a schizophrenic. I am not myself convinced that there is such a neurosis as true hypochondriasis, although I know that most clinicians would probably disagree with me on this point.

I think I could fairly easily cook up 20-25 neurological items based upon my clinical experience so that making such a key would not be inordinately difficult. And we need not worry about the fact that a neurological patient with coarse brain disease or organic spinal cord lesions or whatever would score high on it, since that type of nuisance variable is one that we can handle by excluding cases from other medical evidence.

B. Cognitive Slippage (including perceptual, thinking, verbal-associative, body-image, para-causality, etc.)

78. First of all, a general comment. As Wirt's candidate Mrs. Carole Judge (SCB) mentioned to me recently in talking about her thesis, it's scandalous that we don't already have powerful measures of subtle thought-disorder or "cognitive slippage" available for purposes of genetic studies in schizophrenia. Considering the fact that it was via the testing of "intellectual functions" that psychologists got involved in the clinical field in the first place, it is rather strange that we have to look around to find even a few live options for measuring subtle cognitive impairment.

---

[2] MMPI-2R (Minnesota Multiphasic Personality InventoryR) Test Booklet. Copyright ©1943 (renewed 1970) by the Regents of the University of Minnesota. All rights reserved. Used by permission of the ity of Minnesota Press. "MMPI" and "Minnesota Multiphasic Personality Inventory" are registered trademarks owned by the Regents of the University of Minnesota.

79. First off, I would incline not to bother with tests of intelligence or most tests of psychological deficit on the grounds that while some slight statistical discriminations seem to hold up even on such low validity things as Wechsler patterns, the overlap with normals is huge and the deficit is not schizophrenia-specific among patients. If it isn't even specific for florid clinical schizophrenia, surely we wouldn't find this a plausible locus to search for indicators of sub-clinical thought-disorder of the kind that I have in mind when I use the phrase 'cognitive slippage.' Thus, for instance, I wouldn't think of resurrecting the old Babcock (aside from the fact it takes too blasted long to give) because Babcock deficit occurs with organic brain disease, severe depression, severe anxiety states, under-education, not being test-wise, and goodness knows what all else. Of course if there was only one alternative source of Babcock deficit or Hunt-Minnesota test deficit, such as coarse brain syndrome, it wouldn't bother me because as I said before there's no reason the psychologist should be shy in using the usual medical model in which a symptom can be useful as an inclusion or exclusion test for condition X even though it is frequently produced also by condition Y, provided we have adequate means of assessing the presence or absence of condition Y from other data. But the thing is, if you get any sizeable list of alternative sources of a measured deficit, it gets to be a mess statistically and there are too many cases left ambiguous.

80. However, I would not want to abandon the whole general line involved in the traditional approach to deficit testing. Example: I don't understand why nobody has ever sat down and built a better version of the Shipley. Shipley himself kind of threw it together and as you may recall the normative data were pretty punk, but if a person were to construct a longer Shipley with more powerful items and higher reliability with a true multiple regression approach to predicting abstraction score from vocabulary score plus a couple of others (the way Howard Hunt did with his test), it might be quite useful. There are pretty good means of identifying mentally defective and organic individuals and excluding them or at least classifying such cases as doubtful, and for the remaining cases I am of the opinion after having given hundreds of Shipleys in past years that a nice healthy psychoneurosis will not in general produce marked Shipley deficit whereas even a mild schizophrenia will often do so. I was also somewhat surprised to find out the Shipley turning out as well as it did in the Turner and Phillips study, a reprint of which you have. I don't <u>think</u> that I would be inclined to include the Shipley in a battery of indicators in its present form, but I am open to conviction on the point, and I hope that Carole Judge's thesis data will provide some rational basis for deciding whether to skip the whole thing, to try to build a better type of Shipley or to use the Shipley itself. From the armchair, it seems to me that the inability of a patient to perceive the abstract relationships needed to complete the series in Shipley abstraction items up to the level of his estimated general intelligence as indicated by the vocabulary score, is the sort of thing one might very well expect to be a reasonably good indicator of cognitive slippage.

Jack Powers and I have some data which are incompletely analyzed (but in very good shape to complete the analysis) on the Shipley results of VA-MHC patients who were divided by a bootstrapsing method (initial diagnosis plus Meehl's armchair weights followed by application of the method of reciprocal averages) into schizotypic and non-schizotypic samples. Together with the Turner stuff and Carole Judge's stuff this study might lead to a definite decision to try something with the Shipley or a modification thereof.

81. If Braatz finds anything in his thesis we will perhaps have a basis for using transitivity of preference behavior as a measure of slippage. I won't exposit this since we have talked about it so much, but since I am sending carbons to some other people, the idea is briefly that one indication of cognitive slippage from the standpoint of the logician would be the appearance of intransitivities in preference behavior. That is, a person preferring A to B and B to C should presumably prefer A to C. Arnold Rose (sociology) in his study of the judged seriousness of crimes as evaluated by college students came to the conclusion that, contrary to what economists and utility theorists have said about human choice behavior, transitivity does obtain (almost one hundred percent) provided one meets two conditions, namely, that the judge is performing the task conscientiously and seriously and carefully so as to give reliable results in the sense of consistency for each choice response on two occasions; and secondly, that the items being compared are initially scaled so that they are psychometrically far enough apart so that a preference at the behavior level reflects a genuine inner preference rather than flipping a coin, so to say. Rose found that if you take crimes that are scaled so that they are significantly far apart on the continuum of "judged seriousness" in a college population, and you throw out a small proportion of cases in which internal analysis of the data — aside from transitivity itself — reveals that the student was proceeding carelessly or semi-randomly or facetiously or in some way really unreliably when he made his choices, what you find is almost 100% transitivity in this particular domain.

I am rather at a loss when it comes to selecting a plausible subject-matter or a set of stimuli for getting at the intransitivity. My initial idea was to use some kind of neutral, non-threatening stuff like kinds of animals, so that the patient is presented with cards on which there is a picture of a canary, a goldfish, a Siamese cat, a Norwegian elk-hound, and the like. On the other hand, as we have discussed, there is the possibility that one can potentiate the cognitive slippage effect on intransitivity by a cross comparison between the transitivity index of positive hedonic choices and that of aversive ones. If we can ever blast that set of items out of Stanley Schachter or make up a batch ourselves (I would tend to start by using the Murray needs to have some assurance of covering the waterfront of hedonic experiences), we can get a more complicated formula which is an intransitivity index based upon a comparison of the amount of intransitivity shown in the "plus" versus that shown in the "minus" domain of items.

It occurs to me that I am not aware that anyone has even investigated the transitivity of non-hedonic or non-evaluative judgments in schizophrenics. What, for instance, would happen if one studied the transitivity of choices on some fairly simple dimension of a stimulus modality like pitch or volume? It seems a rather obvious thing to have studied but I don't recall seeing such an investigation. Here we again run into the damn problem of the influence of psychiatric nuisance variables upon performance, and I would certainly think it undesirable to be "pushing the threshold" in this kind of situation because of the probable influence of such nuisance variables on performance and their resulting non-specificity for schizotaxia. However, we might take tones which are separated by considerably more than the difference limen but close enough so that a possibility of intransitivity arises if slippage is present. I suppose I favor content like animal preferences or judging seriousness of crimes because it seems to me that most patients, whether neurotic or schizotypic, would be able to accept the instructions that there is no "right answer" whereas the transitivity of sensory discriminations brings in a "task" aspect which it is desirable to avoid if possible.

82. Verbal community of response. There is no question that as a group schizophrenic patients show a lower community of response in verbal association than normals and neurotics. But it is my impression (without having looked at this literature lately or ever having really thoroughly reviewed it) that no very sophisticated approach was taken to the quantification of low community of response. As you know, my theory of how the clinician picks up the very subtle sub-clinical aberrations in speech that we detect in schizotypes is that the transitional probabilities for a given word following a sequence of certain words are just a little off-base in schizoid speech, so that even though nothing actually bizarre or way out in left field is being said by the patient, the listener develops a sort of cumulative impression of the talk sounding "strange" because words appear in contexts which do not violate the laws of grammar, semantics, or logic but are nevertheless of lower probability for the normal mind than for the schizoid mind. Jim Jenkins would no doubt have a lot to say about this, but I think of it as a kind of mild disconfirming of expectancies that one rapidly sets up for what word or word-family is likely to appear at a certain place in a sentence, but then the patient uses some other word.

Again, Braatz should be able to tell us whether the traditional type of association procedure, if supplemented by a slightly more clinical approach to the quantification of low community (i.e., the business of finding the median of logarithms of the ten percent low communality words on a single patient's distribution of responses), has a chance to tell us something.

Alternatively, I rather like the idea of the so-called "cloze" technique, in which a sample of somebody else's actual verbal behavior is presented to the patient with words deleted and his task is to fill in a word which makes sense. The point being that there are frequently quite a few words which make perfectly good sense and could not be called "wrong" answers and yet they are not in fact the words that were used by the original producer of the stream of discourse. So that if the sample of such discourse is sufficiently long and we have a sufficiently large number of blanks to fill in, we can score a patient's fill-ins by counting how many times he put in the word which the original producer of the discourse used at that locus. I have a reprint by Fillenbaum and Jones (1962) in which cloze technique was applied to the study of aphasic speech, the stimulus material used being transcripts of speech elicited by the TAT. I don't know whether cloze technique has been used in schizophrenia or not, but it seems to me that it is one possible psychometric approach to the kind of thing one does clinically in detecting mild schizoid speech patterns. I have a reprint by Taylor (1953) that explains the "cloze" technique, although not with reference to schizophrenia.

83. Another verbal approach to the cognitive slippage problem is in the use of proverbs, which in several investigations seem to have respectable validity (Gorham 1956, Payne 1962, Payne and Friedlander 1962, Payne and others 1964). This surprises me quite a bit, because 20 years ago I used to employ the Benjamin Proverbs in examining schizophrenic patients and formed the impression that when they "work" they work in patients for whom no proverb test is needed because there is a clinically obvious schizophasic tendency, but in patients free of such tendencies the proverbs did not seem to differentiate. So I had a rather bright and conscientious girl do an M.A. thesis on the Benjamin Proverbs, contrasting schizophrenics with other kinds of psychiatric patients and the results were thoroughly disappointing. But you will want to look at these reprints

and form your own opinions. The Gorham study is fairly impressive if you can believe the table, since with a large N and adequate matching of controls it looks as though one can achieve around a sigma separation and on a cross-validating sample the results are better than that sounds because he claims 78% hits. He has a problem apparently with discrimination not being very good at the high intelligence level. And as usual the cognitive slippage measure does not work as well in discriminating paranoid schizophrenics because of their relative cognitive intactness. The proverbs approach appeals to me partly because the paper by Phillips, Jacobson and Turner (1965) suggests that, like the Shipley and the Payne Object Classification Test, the Benjamin Proverbs show aberrations in the non-psychotic relatives of schizophrenic patients.

84. Some other possibilities in this area are what Silverman (1964) calls "cognitive filtering," based upon verbal expressions of the patient's idea of the width of a category, such as knowing the average rate a bird flies, being asked to estimate the range. Then we have a confusion between the literal and figurative functions of language, reported by Chapman (1960). Errors in syllogistic reasoning, which I would tend to be suspicious about because of the presumed high loading with intellect, have been studied by Chapman and Chapman (1959) and Gottesman and Chapman (1960). The so-called "atmosphere effect," in which the seductive power of a particular formal fallacy (such as the undistributed middle or the illicit major) is enhanced by the position of the quantity words such as "all" or "none" in the way the syllogism is presented to the subject, rather appeal to me. Since there is a good deal of normal slippage by students untrained in logic when they are presented with such formal fallacies (shown years ago by Woodworth and Sells, who invented the phrase "atmosphere effect"), one might worry that the nuisance variables play a very powerful role here. On the other hand, this is rather like the transitivity thing in the sense that a lot of the items are just difficult enough that the potentialting effect of the atmosphere might be dependent upon schizoid slippage. The whole thing is mainly worth mentioning as a possible area to work up rather than as a presently available indicator, since the Chapman and Gottesman article does not suggest that this ordinary syllogistic error business is a powerful discriminator.

85. Another possibility for tapping cognitive slippage in the "verbal-associative" domain is an idea I had in wondering about the extremely low Miller Analogy scores one sometimes sees in clinically bright graduate students who are schizotypic. It occurred to me that one might concoct a fake vocabulary test of the multiple choice variety (thereby avoiding the problem of scoring-unreliability and having a thing that is self-administered or at least not time consuming). By "fake" I mean that one would make up some nonexistent words that appeared like plausible English words, with of course a bunch of buffer items that are genuine English words, and with the incidence of fake words increasing as the patient works through the test so that it would appear plausible to him, i.e., that he was just getting gradually into more and more difficult vocabulary levels. Then we include some multiple choice alternatives that we have picked either on the basis of a prior open-ended type of study; or, since that would have to involve a huge sample, we might be willing to do it from the armchair, perhaps deliberately loading the alternatives with some kinds of thematic content. I don't really know whether this kind of thing would be best put under cognitive slippage, or somewhere else in the list but that doesn't matter. The items might look like this:

Aldize: Dislike Shine Love Give

Mangorf: Eat Help Monster Flower
Grounge: Mother Uncle Brother Sister

86. Goodenough's test based upon "homonymous homophones" is another possibility where a deviant response could presumably be due to content factors potentiating associative slippage. You take a word which in spite of being spelled and pronounced one way, such as the word 'ring', has two or more meanings and the subject picks the meaning that he associates. Thus, males and masculine women associate 'ring' with 'boxing' or with 'bell', whereas normal females associate it with 'wedding'. I don't have a copy of Goodenough's paper on the subject, but the article is "The use of free association in objective measurement of personality" Chapter 5 in McNemar and Merrill, Studies in Personality (1942). The last time I checked with Harold Stevenson he said that they were preserving intact the mess of records that the late Florence Goodenough had collected in developing norms for this test during World War II so one would not be starting from scratch. I don't have enough faith in this to push it other than to keep it on a list of things that a student interested in the area might profitably use as a Master's Plan B paper or even conceivably as a Ph.D. thesis topic.

87. I have in my reprint file only the very smallest part of the many studies that have been done on verbal behavior in schizophrenia, including a couple of statistically significant but not practically useful findings by Anker (1957), and by Burstein (1961). It's hard to tell in the case of Burstein's data whether he gets a decent separation or not, although the standard deviation of error scores on the normals is so tiny that even with a pretty large variation in the schizophrenics he may have something worth looking at. (Looking through all this damned literature has convinced me even more than I used to be that journal editors — ought to be forced by some APA policy statement by the Board of Publication to require authors who present data on clinical problems to report overlap statistics and preferably to present graphical distributions!) The Burstein task is kind of interesting in that he presents the patient with the multiple choice task of picking out the antonym of a given stimulus word and picking out the homonym of a particular stimulus word. Each item makes available among the choices a synonym, an antonym, a homonym, and an unidentified error alternative which, judging from his examples, might have some kind of screwy associative connection but certainly doesn't satisfy the task instructions. I think the idea is interesting because it relies upon the fact that Freudian "primary process" thinking often mixes up opposites and relies upon verbal similarity like klang associations.

One that has interested me for years and which I believe has been tried out with TAT material although it's not in my reprint file is the matter of Zipf's law. You remember the late George K. Zipf of Harvard, the guy who plotted the frequency of occurrence of words against the rank (or the reciprocal of the rank?) and said that it was very close to a straight line? He has some fancy theory about the principle of least effort but apart from his interpretation, my recollection is that somebody, perhaps Zipf himself, showed that in schizophrenic verbal behavior Zipf's Law breaks down or at least it falls apart sooner in passing to the extremes of frequency than is true of the normal. This I would have to look up, but while I daresay it has a tendency to work, I don't favor it much because the use of Zipf's Law requires a rather large sample of verbal output in order to generate stable frequencies. I should look up Zipf's research on schizophrenia and see whether he got impressive separations.

88. The body-image aberration is one of the forms of cognitive slippage which I would most like to pursue in a quantitative way, because from the standpoint of anecdotal clinical data it strikes me thus far as being a nearly pathognomonic sign. I am not prepared to say that it is two-way pathognomonic, but I have a very great faith in it as an inclusion test. There is of course a problem of reliability in assessing it in the interview situation, especially when one has narcissistic neurotics with inferiority feelings and has to make a distinction between the evaluative aspects of the body-image and the perceptual aspects of the body-image. My list of reprints is incomplete but I believe it is correct to say that this is one of the rather small number of schizophrenic signs which has consistently replicated. Bleuler considered it pathognomonic, as did Paul Schilder. If you want to have a look, the reprints are by Arnhoff and Damianopoulos (1961), Cleveland (1960), Cleveland, Fisher and others (1962), Wapner and others (1958) — here the relative size of one's own body and of an external inanimate object was judged discrepantly and the effect was potentiated by administration of LSD — Wapner and others (1958) where perceived head size and shape is influenced by enhancing the body's boundary through the sense of touch. There is also a study by Weckowicz and Sommer "Body image and self-concept in schizophrenia," J. Ment. Sci., 1960, 106, p. 17, which is not as impressive because it is not cross-validative and they picked out a couple of significant differences on a larger batch that were tried. I have, however, sent for that reprint as well. Clinical experience, theoretical considerations regarding the so-called "ego-boundaries" of the schizophrenic patient, and the notion of body-image as the repository of the sense of self and what Rado calls the "action-self", make this something rather plausible and I am therefore inclined to argue for trying it out with fairly high priority.

It is not inconceivable to me that the body-image aberrations which are sometimes complained of by the patient although they are not displayed in the interview might be assessed with respectable validity from a questionnaire. As in the case of soft neurological signs, I have a hunch that I could make up a set of such body-image complaints on the basis of my clinical experience that would have some validity. We included some content about body-image aberrations in the self-sort section of the Ford Project, but I don't think that the number of such items was more than two or three in all.

89. Stabilized retinal image. This one still has great appeal to me as I gather it does for you provided that it can be shown that the substitute method suggested by McKinney (1963) using luminous designs gives results which are of the same essential nature psychologically and neurologically as was true of the more complicated method of Pritchard and Heron. The business of having to spend hours getting the subject adapted to having the projector on his eyeball, and the fact that individual contact lenses have to be used to accord with the subject's normal visual correction, makes this technique unfeasible for any sizeable sample. I have a review of the stabilized image literature by Heckenmueller (1965) but he does not even cover the luminous designs method so I gather he considers it of a different nature. I don't believe that the failure of my student Brustman to come up with anything in his preliminary exploration of the McKinney stuff proves anything at all, since he was somewhat lacking in ingenuity at that stage of his development, and neither you nor I was able to dredge up the "positive" technique that we invented at a party at your house but subsequently failed to write down. I certainly think this one should be pursued further but only if you and I can come up with some good theory-based designs to use or, alternatively, get a student working on it who has considerable ingenuity and creativity. The anecdote that Hebb told me is I gather a lucky hit only, since Brustman

tells me (I don't recall whether from his own data or some correspondence he had) that it has turned out not to be terribly unusual for presumably normal subjects to fill in, in a quasi-hallucinatory way, to complete a good Gestalt and so forth. It just isn't safe to throw this one out unless one has a student who has time and drive to examine preliminary data at length and try different ways of slicing the pie. In five minutes one can come up with all sorts of different possible relationships in timing and so on which to my knowledge have not been investigated at least with respect to schizophrenia. For example, what about the oscillation of ambiguous figures in the stabilized retinal image situation? What about induced effects from the contralateral eye? In fact, the problem here is one of narrowing down what to pay attention to because of the vast number of parametric features that might plausibly do a job. For the effect of contralateral visual input on the visibility of the stabilized retinal image see Cohen (1961).

90. There are a million (mostly punk) studies on the Rorschach and as you know it has some validity but tends to pick up cognitive slippage as usually scored by clinicians other than Klopfer only when the patient is rather clinically obvious. If we use ink blots at all I would be strongly inclined to employ Holtzman's blots which were built the way they should be, and for which there already exists a multiple discriminant function for identifying schizophrenics; the cross-validation data in his book are rather impressive. I have not seen much clinical use of the test by anyone or even the amount of research you might expect, which I suppose is because clinicians are either favorably disposed to ink blots in which case they are wedded to Uncle Herman's set, or else they are not favorably disposed to the whole approach. I think I will write to Holtzman and ask him whether he has any data on pseudoneurotic schizophrenics. I assume nobody has done a job on well-compensated cases with his blots.

91. There are some assorted miscellaneous visual-perceptual tasks which have been worked on, such as perceived changes in line tilt with time (MacFarland, 1962), perception of body affected by visual-spatial context (Wapner, et al., 1963), perceived eye level (Wapner, 1963), but these have not been applied to schizophrenics. You are familiar with Silverman's (1964) stuff on size-estimation as a measure of "cognitive scanning", which did deal specifically with schizophrenia and looked fairly good. "Perceptual regulation" as measured by visual input and vigilance has been studied in schizophrenia by Dixon and Lear (1962). Significant differences but not striking were reported by Sappenfeld and Ripke (1961) on spiral after effect, retinal rivalry, and stereoscopic figure tracing.

92. The papers by Julesz, which I think you originally called to my attention because one of them appeared in Scientific American, on binocular depth perception and random dot stereopsis involving variables of contour, texture, and patterns, have not been applied to schizophrenia but my intuition is that synaptic slippage ought to do something funny in this kind of business. It's as vague as that, but my intuition is strong enough that I think one ought to put this one rather high on the list of possibilities for pilot studies by research assistants or Master's candidates doing a Plan B paper. I think you have the reprints, but they are in my file under Julesz (1963, 1964, 1965). Part of this stuff was discussed by [Herbert] Pick at one of our evening meetings last year.

93. The "novelty" factor I emphasized in my list of armchair desirable features for an indicator is certainly present in markedly distorted perceptual inputs such as the Hanover demonstrations (Adelbert Ames) and I am not aware that any work on the reaction of schizophrenics to these contradictory visual cues has been published. This is another one

which I would put fairly high up for pilot investigation, and at least some of the Ames demonstrations can be done with small and cheap apparatus. Aneisokonia is something I have never studied up on but aneisokonic lenses exist to produce such distorted visual input in people with normal vision. Here also the aspect of novelty may present the schizotaxic brain with a resolving problem which it would handle differently from the normal. I don't assign any priority to this because of my almost complete ignorance of the psycho-physiology of aneisokonia. I think I will ask Herb Pick whether he knows of any research that anyone has ever done on the reaction of mental patients to the aneisokonic distortions produced by these lenses.

94. In auditory perception, I'm inclined to assume that the schizophrenic hallucination results from a combination of primary slippage with katathymic potentiators. There is a study by Adams and Berg (1961) on deviant perception of ambiguous auditory inputs among schizophrenics, but it in terms of statistical significance of items rather than in a form which enables you to infer how good a scale separation might exist. I don't think much of Berg's "deviation" hypothesis in general, but if it is any good at all, it should be good for schizophrenia.

Signal-to-noise ratio looked gorgeous in one study but failed to hold up on attempted replication. I only have two reprints here, Stilson and Koppell (1964) and Ludwig, et al. (1962).

Impairment of verbal function associated with delayed auditory feedback (Fillenbaum, 1963) is another one which I would intuitively expect to be a good bet, and reading those British reprints you gave me increased this hope. I do not know whether anyone has specifically investigated schizophrenia in this regard.

95. Discrimination of weights, like every other discrimination task, tends to differentiate schizophrenics, but I know of no data that involves pseudoneurotic or compensated cases. I would be suspicious that this is just another example of poor performance due to inattention and preoccupation, but the proprioceptive aberrations in schizophrenia which pop up in so many different contexts leads me to think that Rado may be right in talking about a "proprioceptive diathesis" as fundamental to the disease. I have made no attempt to derive such a thing from the hypokrisia concept. It might be, of course, that hypokrisia will give you aberrations in any domain if you study it sensitively enough, but that the special role of kinesthetic feedback in the integration of behavior, and especially in the development of the relation between cognitive and executive ego functions, makes the proprioceptive aberration more striking clinically than those of other modalities like vision and hearing. The only reprint I have in this area is by Wurster (1965) which as usual does not present data in such form as to give you the faintest idea of how powerfully he could discriminate his schizophrenics from his controls. The patients were cases of chronic schizophrenia. It was a study of the effects of anchoring on judgments of weight.

It has occurred to me that one simple approach to the use of weight discriminations that might cancel out the effect of non-specific nuisance variables and be getting at something more fundamental than the degree of task involvement and so forth, would be to compare the amount of impairment in weight discrimination, measured by inflated values of the Weber fraction, when weights are judged in the usual manner of psychophysics by hefting, versus "passive" judgments in which proprioception plays little or no role and the input is entirely tactile. In both cases nuisance variables involving motivation and affect would tend to impair discrimination, but a specific impairment in the kinesthetic modality

such as Rado and others have alleged to exist would presumably show up if the proper difference score or other comparative index of the amounts of inflation of the Weber fraction were devised. I kind of like this idea and would include it among easy-to-do pilot studies.

96. Disturbances of time and space orientation discriminating much better than I would have expected were reported by Delagarza and Worchel (1956) and they used paranoid schizophrenics who would be expected to be the least "confused" of any group of disintegrated schizotypes and also equated them with the control group for test cooperativeness on the Shakow-Moran rating scale. Since getting balled up in space occurs in my schizotypic checklist (I hope without too much diagnostic weight since I am chronically disoriented in buildings or in cities!) I think this one is also worth trying out some day. It isn't just a crude matter of orientation as in the mental status examination, but involves more subtle cognitive tasks such as the patient being asked to state in words what direction you have to go to get from one specified city to various other cities. From the armchair it doesn't strike me as very plausible but their table, unless there is something I am not spotting in it, is really quite impressive.

97. Object-sorting tests are of course favorites with investigators of schizophrenia ever since the original development of the Vigotsky and the emphasis upon object sorting by Rapaport and his collaborators at Menninger. These are highly discriminating of disintegrated schizotypes even in their original qualitative form as in the Hanfman-Kasanin, and when you get a schizophrenic who shows nice slippage it is fascinating to watch him deal with a test like the Hanfman-Kasanin. When I was working on Station 60 in the old days I used to give these just to watch the patient do it even though the diagnosis was perfectly clear to everybody. If possible I think it better to use a format in which objects are represented on cards and the response is objectively scorable, provided you don't sacrifice any of the qualitative advantages of an actual open-ended task involving physical objects. My reprint collection is certainly only a small part of all of the studies along these lines, but I have the paper by Feldman and Drasgow (1951), as well as a copy of their test which they sent me. This is the one that yielded perfect discrimination between schizophrenics and controls matched on several factors, the controls being picked up hanging around the Greyhound bus depot! It can't be that good, of course. But it looks good enough that certainly it should be tried out. I am thinking of trying to get Lloyd Sines to mandate that some of the clinical psychology trainees administer this routinely for a while to patients on the station to see whether it holds up anywhere near as well as their original sample. Other reprints employing object sorting are by Lidz, et al. (1962), Lovibond (1954), McConaghy (1959), McGaughran and Moran (1956), McReynolds (1964), and of course your friend Payne does another object sorting study every whipstich, e.g., Payne (1962), Payne, et al. (1963), Payne and Friedlander (1962). None of these studies or any I have seen have employed pseudoneurotic or semi-compensated patients. But the overall finding is so consistent that presumably a psychometrically optimized object sorting task should be included in any pilot battery among non-disintegrated cases.

98. A disturbance in sorting or classifying behavior involving concepts rather than objects or pictures of objects, with the emphasis upon distractor cues and intrusion of associations is suggested by the work of Chapman (1956, 1958) and Chapman and Taylor (1957). This Chapman stuff, especially the distractor score business is along the same lines as the British Chapman whose reprint you gave me and the graph presented in his

paper "Distractability in the conceptual performance of schizophrenics" is pretty impressive as it stands although as usual you can't figure out the overlap. If you haven't read the American Chapman's papers, of which I have four or five in the file, you'd probably find it worthwhile.

99. As in the case of "soft" neurological signs discussed above, might it not be possible to develop a self-report inventory of cognitive slippage complaints? It seems that in spite of the lack of insight in the traditional mental status sense displayed by disintegrated schizotypes, the MMPI Scale 8 does reflect the patient's awareness that his ego function is impaired. For example, while the schizophrenics studied as criterion cases when they built the MMPI were all psychotic and therefore according to the textbook tradition would be lacking in "insight," we find that Scale 8 includes such items as "I cannot keep my mind on one thing," "I often feel as if things were not real," "There is something wrong with my mind," "I have had periods in which I carried on activities without knowing later what I had been doing," "I cannot understand what I read as well as I used to," "I am afraid of losing my mind," "I have had very peculiar and strange experiences," "I have strange and peculiar thoughts," "I have more trouble concentrating than others seem to have," "I have had blank spells in which my activities were interrupted and I did not know what was going on around me."[3] I think that it might be possible to build a questionnaire for subjectively experienced cognitive slippage that would be pretty powerful. Of course the self-sort pool in the Ford Project also includes such item content and we will be in a position to "validate" the self-sort cognitive slippage factor (assuming such emerges from the factor analysis) against the one or more cognitive slippage factors we get from the clinician judges on these same patients. So there's nothing you and I should be doing about this pending analysis of the Ford data. And of course we will have MMPI item responses on a thousand or more cases who have been Q-sorted in the Cookbook portion of the project. The cognitive slippage factor is one of the most powerful factors in the pool and showed up as such even in the preliminary factor analyses of patients all of whom were presumably "only neurotic" as judged by the therapists.

C. Anhedonia

100. I am going to be unhappy unless some sort of anhedonic indicator can be included in family study data, but I must admit this is mainly on grounds of wanting to corroborate theory and validate clinical impressions, rather than a strong conviction that such an indicator will function powerfully. As is pointed out in my Manual for the Schizotypic Checklist, the main problem here is in assessing the trait reliably. Even if we assume that anhedonia is non-specific, which I am willing to assume (since Rado changed his mind about it and when someone who has gone on record in print changes his mind as a result of further clinical observation I figure he must have been sort of "forced" to do so), in terms of the genetic theory presented earlier in this memo, we still expect to find a primary low hedonic potential in schizophrenics because being at the low end in this respect is one of the most important potentiators of clinical schizophrenia. I think most clinicians would agree that the pure obsessional anhedonic uncomplicated by

---

[3] MMPI-2R (Minnesota Multiphasic Personality InventoryR) Test Booklet. Copyright ©1943 (renewed 1970) by the Regents of the University of Minnesota. All rights reserved. Used by permission of the University of Minnesota Press. "MMPI" and "Minnesota Multiphasic Personality Inventory" are registered trademarks owned by the Regents of the University of Minnesota.

schizoid features or by anxiety and depression as impairment factors is a pretty rare bird. From which I conclude that such cases would not constitute a heavy source of classification error. But the problem is measuring the anhedonia as distinguished from depression, anxiety, and the vicious-circle feedback from social reality that makes so many neurotic persons unhappy. I think I can discriminate it on the basis of extended interview conversations, but that doesn't do us any good for our genetic research purposes. I am not going to take much time on this area because of my relatively weak conviction that it will be possible to develop a powerful indicator.

101. It might be possible to develop an anhedonia key for the MMPI. I have a memo in my green notebook on schizophrenia dated November 14, 1963, "Schizotypal Items on MMPI (Armchair)," which I think you should probably have a look at. Here I classified items a priori as probably reflecting one of the four basic schizotypic variables or neurological content. In the case of anhedonia I divided the face-valid items into those which I called "anhedonia, general," meaning that the patient simply asserts a generalization of the anhedonic type. I believe that these items will be excessively saturated with depression. Then I had a group of "specific" items which tell us whether the patient claims he likes or enjoys certain things or not. My thought was that these items if properly counterbalanced for the main nuisance variables of occupational level, social class, intelligence, and masculinity-femininity, might be used as a cumulative hedonic measure. I'm not very happy about this because of the Strong data which indicate, if I remember correctly, that the tendency to like or not like things is in itself differentially related to certain patterns of vocational interest. On the other hand, Berdie presented evidence during World War II that a general lack of interest as seen by the counseling psychologist was indicative of psychopathology. I am afraid that the psychometric problems in counter-balancing items and all would be a big pain in the neck, so I don't rate this terribly highly. I have considered, now that the MMPI item responses have been put on computer tape (I believe something like 6,000 are now available locally), doing a purely internal consistency analysis such as a factor analysis of this kind of material and then seeing whether a general anhedonic factor emerges that is correlated with formal diagnosis, especially in discriminating schizophrenia from other painful affective states, like anxiety and depression.

Of course, it might be possible to do this by a differential score method, which is certainly what one does clinically in assessing anhedonia from a series of interviews, that is, we argue, roughly, that any patient who doesn't like activities x, y, z and so forth, is either anhedonic or is having a fairly intense kind of interfering affect such as depression or anxiety. So we reason that an individual whose anxiety and depression are not too great but who is high on self reported anhedonia has the disease. The trouble is that this one, while it would work as an inclusion test, would almost certainly not work as an exclusion test, because of the sizeable number of anhedonic schizotypes (especially the semi-compensated or pseudoneurotic ones) who also suffer considerable anxiety and depression.

Here again the results of the Ford Project should permit a decision as to whether anhedonia as such can or cannot be adequately assessed by MMPI items.

102. Several years ago, Jack Powers and I did some preliminary work on developing a semi-structured interview which among other things would get at the anhedonic tendency. I do have a set of leading questions which have been roughly scaled for their

hedonic pulling power on the basis of how some judges rated them (I believe you were one of the judges here). If a semi-standardized interview is to be included at all as part of a battery of indicators (as I rather think it should be, although I hate the thought), then I think this line should be followed up further. The next step would be to take those leading questions which the judges agreed had face-valid pulling power for eliciting a pleasure-toned verbal report from a normal person and run a series of normals, tape recording the interview and finding out empirically what the pulling power is. You might save time therefore by sort of a Guttman scaled sequence such that if a patient gives a pretty scoreable hedonic plus response to the first three or four items you don't pursue it further, whereas if he fails to give one, you pursue it to the bitter end, running through the whole list of 15 or 20 such. Quantification of things like number of words spoken, and in my clinical experience even the latency, might do a job here. If a person is not so grossly depressed that he manifests a general retardation in responding to mental status examining questions, there is something mighty suspicious about him if he has to pause and think quite a while when you ask him, "What do you do for fun?" The main disadvantage of this line of approach is that one cannot really do an adequate pilot study relying upon research assistants or the average Ph.D. candidate, but we would have to do the interviewing ourselves. And I don't have enough confidence in the likelihood of its working out to want to spend very many hours at it.

An old Columbia Psychoanalytic Institute sibling of Glueck's named Buchenholz has done some preliminary stuff on the subjective descriptions of pleasure experiences and I have his reprints (1956, 1957, 1958). The reports are phenomenological studies and somewhat interesting reading but I don't get very far when I ask how this sort of thing can be used to construct a practical instrument for detecting anhedonia.

103. Here again the Ford study might provide a self-sort instrument assuming that we can find items and identify a factor in the self-sort pool which correlate with the anhedonia factor that emerges in the clinician, judge pool. I would not think it therefore desirable for us to spend any time on this, pending analysis of the Ford data.

Jackson and Payne (1963) have developed a "shallow affect scale," a structured test of forced-choice format, built by a combination of face validity and a remarkably high internal consistency (corrected odd-even reliability of .96). It has not to my knowledge been applied to schizophrenia, and none of their external validity data are very impressive to me. I am pretty sure it would discriminate chronic schizophrenia, but more dubious about pseudoneurotic and compensated cases. Since this is a structured test requiring no time except the patients' and a clerk, I might try to get Lloyd Sines to run a series of these on our hospital patients, which could be scored by the Station 60 psychometrist, again relying upon formal diagnosis (plus perhaps MMPI) as a sufficiently valid basis for crudely classifying into schizophrenic and non-schizophrenic patients, to decide whether the shallow affect measure is worth studying further.

As mentioned earlier in the memo, I don't think it's clinically correct anyway to say that schizophrenics have "shallow affect," except for the very chronic, burnt out cases. Even disintegrated schizotypes are quite capable of strong affects of the "emergency" emotion variety, and Jackson and Payne unfortunately included both aversive shallowness and appetitive shallowness in the item pool. But of course with such high internal consistency, it might be possible to split their items as we are in the Braatz study and get

separate scores. It is of course very handy to have a scale on which the authors have already done the necessary calibrations to achieve a good forced choice composition.

I suppose there is a rather crude and obvious approach to this which to my knowledge has not been tried, namely a graphic rating scale with suitably chosen verbal anchorings in which we try to anchor an "hedonic zero" and employ a non-forced instructions on a mixed pool of appetitive and aversive items. This is so crude and obvious that one assumes that it wouldn't work, but what the anhedonic patient will sometimes tell you explicitly in the interview he may also be willing and able to tell you on this basis. So that without investigating questions of transitivity, we might be able to show that schizotypes generate a mean and variance for aversive items that is rather like that of normals and neurotics; but that the variance of appetitive items shrinks considerably and that the mean placement of appetitive items is shifted downward toward the zero point compared with normals and neurotics. What do you think about trying this on a few cases? Individual differences in the error of central tendency would at least partially be taken care of by an appropriate index in which the means and variances of the appetitive and aversive subsets were somehow compared.

D. Interpersonal Aversiveness

104. I am very skeptical about this one for convenient research purposes. Even though I included it in my set of four defining properties of schizotypy, this is on the basis of intensive clinical experience with borderline cases, plus the "big fact" that the psychotic and dementia simplex schizophrenias are obviously interpersonally aversive to a degree not found in other types of mental patients. But we do not expect the pseudoneurotic patient or the compensated schizotype to sit around not looking at others like a state hospital schizophrenic does. In the pseudoneurotic or early disintegrated case, the interpersonal aversiveness is mainly discernible on the basis of transference reactions in the interview which are either of a subtle stylistic type (such as the patient's tendency to turn his face and body slightly away from the interviewer), or on the basis of the therapist's opportunity to observe many episodes of transference testing operations, exaggerated response to minimal cues of love or rejection by the therapist, and the patient's narration of many aversive episodes in his relationships to others in his environment. I don't think most of this sort of stuff can be assessed from short behavior samples as by history taking or mental status examination, even by a clinician who is unusually perceptive. This means that we are going to have to get at interpersonal aversiveness by other than mental status or life history techniques for research purposes. But when I try to think of psychometric ways to do it, I find it hard to imagine item content which will discriminate the interpersonal aversiveness of schizotypes from that of many neurotics. Garden variety "social introversion" is just too broad and has too much nonschizoid variance to be at all useful for this purpose. Furthermore, I believe that compensated schizotypes and schizotypes with a great deal of extra-punitiveness and other externalizing or aggressive defense mechanisms going for them, do not appear particularly shy or socially timid and do not always have a reportable self-concept to that effect. On the MMPI scales 8 and 7, for instance, there is as much content reflective of interpersonal aversiveness on Scale 7 as there is on Scale 8, if my memory serves me. I have, however, identified a list of face-valid interpersonal aversiveness items from the MMPI pool in the same memo I mentioned above, and here again. I think the thing to do is do some runs on the Ford data to see

whether an interpersonal aversiveness key on the Mult can be constructed which is sufficiently specific to the schizoid type of aversiveness to be worth using.

105. Encouraging possibility here is the Ford self-sort pool, that part of the project being Schofield's bailiwick; and I know that he plans to investigate the interpersonal aversiveness pattern in the self-description with an eye to identifying schizotypes. So here also we should await the outcome of the Ford stuff.

E. Damaged Self-Concept

106. This indicator domain is rather like the previous one, in that I believe very few neurotics have as severely damaged a self-concept as the schizotype does, but this is at a kind of theoretical level and hard to instrument for practical research purposes. I think that the "severity" of the damage is impressive to the psychotherapist partly on the basis of its refractoriness to either objective experiences of success in life or affection and approval from significant others including the therapist himself; and partly a matter of the diffuseness of the damage in the sense that no life area appears to be even moderately free of this taint of "I am no damn good." Assessment of the former is a pretty subtle qualitative matter involving a great deal of clinical perception, and assessment of the latter involves covering a wide content domain. Furthermore, this wide domain isn't just a scattering of shots as regards content because for most people, most specific items are not particularly relevant one way or the other. In other words, if a person says he doesn't have a high concept of himself with regard to playing tennis, this doesn't tell you much unless you already know that he has played a good deal of tennis and has been apparently motivated to do well at it and seems objectively to be adequate or superior at it. We have a problem here rather like that of the phobias or obsessions in a Multiphasic scale, like Scale 7, where you recall there are very few of the classical textbook signs that show up statistically. The reason for this of course is that any one such symptom occurs in only a tiny fraction of obsessive or phobic patients. If we wanted to find out about a person's damaged self-concept with regard, say, to the pursuit of avocational activities, we would have to cover the waterfront, but even that wouldn't do the job statistically, because what we really need to do is identify the manifestly important areas for each subject and then find out what discrepancies exist between his realistic performance and his subjective self-estimate. I suppose all of this can be done by a complicated enough type of instrument in scoring, but it would be a big mess.

A set of self-concept items with factor loadings (based upon Q-technique) exists, and I have a reprint of the paper which is by Fagan and my former student George Guthrie (1959). This article is interesting and their item pool might be a good place to start, but no differentiation of schizophrenics from controls was involved in their investigation.

107. Here again a damaged self-concept inferred from self-sorts will probably be identifiable in the Ford project and if it looks like a powerful discriminator there we might consider using it.

And again, item analysis of the MMPI data in the Cookbook portion of the Ford project may enable us to construct a damaged self-concept scale.

108. You will remember that I have been gathering data on single self-description adjectives with the idea of building an adjectival self-sort Q-pool entirely devoted to schizotypy. Brustman intends to pursue this further by gathering additional data and Jack Powers is still accumulating cases from the Nicollet Clinic for me. This is one of the

things I had been interested in having run on the station when I had my discussions with Koutsky, and it is again one that could be very easily done via the psychometrist and the clinical psychology trainees, provided we could convince ourselves that the available criteria are good enough to be worth bothering with. I now think it would be better not to do a hard sell job on Koutsky and the residents with regard to Meehl's Checklist until you and I can decide upon a standard set of things we want to run on everybody coming to the service for a year or two, and that of course is part of the purpose of this memorandum.

F. Autonomic

109. I haven't really tried to keep up on this literature and you no doubt know it better than I do, even on the basis of incidental learning. I don't know, for instance, what the status of the Funkenstein test is these days. We have Funkenstein data on some Mental Hygiene Clinic cases which are in the appendix of Shirley Corrigan's thesis, but are there any studies of the Funkenstein on borderline cases? Have you seen a study by Szent-Gyorgy (1961) on demographic latency time? Janecek got interested in this and I believe Hastings purchased (on the Wells fund) the necessary apparatus, but Janecek never did anything with it. My recollection is that he had seen somewhere an article (not the one by Szent-Gyorgy who does not report findings on schizophrenia) that schizophrenics have a tendency to be aberrated in this respect. You will find dermographia itself as a symptom mentioned in Bleuler. I don't know anything about the physiology of the phenomenon but it occurs to me that there might be some kind of close connection between the capillary bed business and Bleuler's clinical impression about dermographia. I am of course very suspicious about autonomic indicators of this type is schizophrenia because I figure that most of them are not <u>direct</u> reflections of the aberrated neurology but rather of the patient's "chronic vegetative condition," and hence would not be expected to appear in compensated or pseudoneurotic cases. But your instincts in this are perhaps different from mine.

G. Direct Biochemical

110. Here also my interest is moderate to low and my competence zero. I have a fascinating reprint by Bishop (1960) which studies the effect of schizophrenic plasma injections upon original learning in the rat, and which shows very large differences and no obvious defect in design. I think you ought to have a look at this one just for kicks. I also know of an investigation where schizophrenic cerebrospinal fluid was placed directly upon the brain of some test animal such as the rat or mouse and allegedly had very different effects neurophysiologically from that of normals. I am trying to find where I put down the reference to that one. There is a Ph.D. thesis just completed at McGill University of the bio-assay type for which I agreed to function as one of the off-campus readers, and it looks pretty good. The old spider-web phenomenon — you know the one where the schizophrenic blood serum screws up the spider's ability to spin his web — has been satisfactorily replicated, according to what Werner Simon tells me (see Don Jackson book on etiology); but the trouble is that it is confined almost entirely to catatonic patients. If this is true, it is surely of some theoretical significance in relationship to the patient's "somatic compliance" for being able to develop the specific catatonic accessory symptoms of the disease. Since you and I don't have the technical skills and bio-chemical know-how for this kind of thing, it shouldn't be high on the list of priorities, except that some year it might be fun to fool around with this if we had a good resident or senior medical student as a collaborator.

The McGill thesis showed schizophrenic blood plasma, no fancy extraction process involved, louses up Skinner-box, behavior (VI schedule, food-reinforcement) in rats, and effect is counteracted by chlorpromazine. Fun idea: Could we get this result on college students (from Psych Dept. subject pool) selected for schizotypy by initial MMPI screening plus interview on cognitive slippage measures? A pretty easy study to do.

The Smith and Sines (1960) study on the odor of schizophrenic sweat is another one which everybody laughs at but which I have been unable to find anything wrong with on reading it a couple of times. I believe that Sines is not pursuing it chiefly because it's pretty hard to know what to do next, although I think in the article they did suggest that they were going to try to track down the mysterious X substance. I am certainly not suggesting we should go into this racket, but I am willing to admit that this experiment contributes to my "organicist" convictions.

H. Somatic (Non-Behavioral)

111. Here again there are buckets of studies, most of them defective for fairly obvious reasons such as failure to control chronicity, chronological age, etc. I believe with Kretschmer (and against Paterson) that there is a body-type linkage to schizophrenia, but it is surely nonspecific. I don't think anyone, even Sheldon, would argue that it is linked closely enough to be worth using as an indicator for research purposes on the compensated schizotype.

There are, however, some non-behavioral somatic things that look somewhat better, one of which is being pursued in connection with the VA Hospital study, namely, the capillary bed business. From a genetic point of view of course it is always worth hoping to find some associated objective pleiotropic trait which is of very high penetrance and not influenced by the potentiators that determine decompensation. For example, somewhere I read about a study in which detailed mapping of the pigment spot distribution on the iris turned out to be a fairly powerful differentiator between schizophrenics and controls. (I thought I came across this finding in Bellak, but I can't seem to locate it via the index of his book.) It might strike you as very improbable, until you consider that the eye is embryologically a growth outward from the brain, and keep in mind that in other branches of genetics pleiotropic effects of superficially unrelated phenotypic traits are typically mediated by biochemical aberrations that influence the development of several organ systems.

I. Miscellaneous

112. The Gottesman twin data thus far collected suggests that even the existing MMPI scales when dealt with in a halfway sophisticated manner may be more powerful than you and I might have supposed. Here again I plan to do some work on this in connection with the Ford Cookbook data. I am particularly interested in the following possible approaches:

a. Some years ago I invented a method of combining pattern and elevation which I have not published but which definitely improved matters in my small sample of private patients. I am also going to apply the same technique to the sixty patients on whom we have Q-correlations with the ideal pseudoneurotic schizophrenic from the old Mellaril study in which you participated. The method consists of assigning rank numbers from 1 to 10 to the 10 MMPI scales including Si, or as I would now do it, to the 11 MMPI scales including both Si and Es. We then

subtract each patient's intra-profile rank number on a Mult scale from 12, so that high "rank-numbers" for a given Mult scale will represent a high position within that patient's profile configuration. So that if we consider the clinical scales Hs, D, Hy ... .Ma, Si, and Es, then for a patient whose Sc T-score is the second highest point in his profile, the intra-profile rank weight to Scale Sc would be 10 = 12 – 2. So that each scale receives an intra-profile rank weight number ranging from 1 (lowest score in his particular profile) to a highest possible value of 11 (if that scale is the highest score in his particular profile). We then multiply this intra-profile rank weight by the absolute T-score of the scale in question. Thus if a patient has a T-score = 83 on Scale Sc and this is the second highest T-score on his profile, he gets what I call a rank-potentiated score of 830, i.e., his rank-potentiated score is 83 X 10. These rank-potentiated "product scores" will of course have different means and variances for the several scales, and so we have to calculate another standard score which could be based upon either the general population norms or a psychiatric sample set of norms. In the Mellaril data, I had Powers do an analysis of variance on Scale 8 in which the input dichotomies were simply (a) whether the patient's T-score was above or below the median of the patients and (b) whether the intra-profile rank weight was above or below the median of the patients. The output measure was the Q-correlation with the pseudoneurotic schizophrenia stereotype, and a significant interaction effect was observed, so a product-function should improve discrimination.

Of course a better way to do this would be to form a second degree equation in which we have the T-score itself, the intra-profile rank weight itself, and finally the product function, as three variables in a regression system, but I have not done that yet. Here again I have not done much work on this except to satisfy myself from these two small groups, where I have a decent criterion, that there is some information to be gleaned by such a configural method. To do it right requires a large N with a decent criterion, namely the Ford data.

b. Another configural approach which should be tried and which I have fooled around with in connection with my private patient sample involves a combination of conjunctive, summative, and disjunctive rules. Thus, for instance, we might view Scales 8 and Es as sort of "core schizoid" scales, but go on to recognize that a schizotype who is decompensating can do so either in the intropunitive, pseudoneurotic way or the extrapunitive, acting-out, paranoid-aggressive projective way. The one avenue is represented by Scales 4 and 6; the other is represented by Scales 7 and Si. So we want a formula which (a) adds the two core scales and then (b) adds whichever two of the other four are higher, so that the two subgroups do not cancel each other out in the discrimination. Of course any such mixed formula might be best applied to the kind of rank potentiated T-scores discussed in the previous section. So that if we take each score to be already potentiated by its intra-profile rank weight, we might define an index like this:

$$y = Sc + [\text{Median of 2 highest among Pd, Pa, Pt, Si}] - Es$$

c. Going back to the actual item responses, in Torgerson's book there are some interesting things in connection with what he calls the "relevancy" of items, pages 91-93. He defines "concordance" for any item pair by a formula in which the number of patients hitting both items is divided by the product of the numbers of

patients hitting the two items separately. If items are scaled on some dimension, those pairs of items which are "near" to one another on this scale should be, he argues, responded to concordantly more often than pairs which are far apart on the scale. One might do some preliminary scaling (following a factor or cluster analysis of items face-valid for something such as anhedonia) and then choose item pairs which are close together. Due either to cognitive slippage, or to the fact that somehow the schizophrenic psyche is simply not "organized" the same way other people's are, we might predict that schizoid patients would show deviations from normal relevancy measured by a function thus:

y = (Scale diff, discordant pairs) – (Scale diff, concordant pairs)

where concordant = TT or FF, discordant = TF or FT

We would expect this second order difference to be smaller for schizophrenics.

d. Another thing is related to this but different, and studies Guttmann reproducibility. So far as I know nobody has looked into this with Multiphasic scales and it certainly should be looked into. We take items which are first identified by the fact that they have a heavy loading on some factor (such as depression, anhedonia, social fear, or cognitive slippage); we further reduce the subset by applying a scaleability criterion as found among normals; then we predict that <u>total departures from scaleability will be higher in schizoid individuals</u>. (This may be one way to distinguish schizoid anhedonia from similar things like depression and anxiety, because presumably an altered factor composition in the schizoid group should result in some reordering of the Guttmann scaling, which would in turn bring them out of line with the ordering we get by the studying of normals.) And I suppose some of the same kind of confused thinking that we are hoping to pick up in the Braatz study of transitivity might be expected to operate with regard to item scaleability in Guttmann's sense. I haven't thought this one through (as must be obvious!) but it has the advantage that we can look into it without much trouble, both in the Ford data and in the University Hospital stuff now being put on computer tape.

113. One aspect of Guttmann scaleability which has interested me for years is shifting the emphasis away from reproducibility as a scale property and toward <u>non</u>-reproducibility in a preliminary scale as a means of identifying sub-groups of patients. In his original paper on the method Guttmann briefly mentions this possible clinical use but to my knowledge never followed it up. Someone else may have done so but I am not aware of it. I assume that part of the problem of scaleability, over and above sheer item unreliability, is the fact that people are differently organized (Allport) so that the so-called "common factor" idea is only a very crude approximation to the reality of the situation. It occurs to me that it might be interesting to "purify" not <u>scales</u> but <u>samples of patients</u>, by eliminating sub-groups progressively who are the sub-groups responsible for failures to Guttmannscale. Suppose we started with a set of items identified by an initial factor analysis (plus perhaps some empirical keying considerations, plus item content), these three hurdles jointly indicating that the items were as a group roughly measuring anhedonia. We then do a scaling operation and eliminate a few of the least scaleable items until we get a set of items that are reproducible to some moderate value, not as high as Guttmann originally required, but still respectable. You may recall he started out saying that there should be 95% reproducibility, and then found you couldn't accomplish this

without using very trivial examples in the attitude scaling field (such as I would rather have $10,000 than $5,000, and rather have $5,000 than be out of a job, and so forth). He watered down the requirement and of course most people using reproducibility in actual research, as in our PAR project, have watered it down a great deal. Suppose we settled for a set of items with a reproducibility of around 75%.

This provisional scale is used to identify a sub-set of patients who, when scored on it, show the largest amount of non-scaleability in their response patterns. Now of course if we throw out a batch of the least scaleable patients, we are in danger of capitalizing sampling errors, since obviously starting with a big N of patients and any moderate-length key, it would be algebraically possible to keep culling non-scaleable individuals out until you have a tiny set of patients who are exactly scaleable. But this would be reflecting mostly the capitalization upon sampling errors and specific mental organization of particular individuals so that even these scaleable people would not necessarily have achieved their perfect scaleability in the same way psychologically.

To avoid this, we start out with an initial scale where the criteria for inclusion are set tolerant enough so that the scale has quite a few items in it and permits being split randomly (or in some more systematic way) into to two sub-sets of items having comparable properties. We might, for example, match items approximately for their loading on the first factor in the set as well as for their approximate popularity ("difficulty level," "pulling power"). So we divide our preliminary semi-scaleable set of items into what I shall call the odd set and the even set. We then calculate each individual patient's reproducibility on the odd numbered set. This is our criterion operation. We then arrange the patients in order of their reproducibility on this odd item set. We choose some arbitrarily small-to-medium fraction of the whole group of patients, chopping them off the bottom of this distribution of reproducibility, say, we take out 20% of the patients from the sample, namely those who are least reproducible on the odd set. We now compute the reproducibility of the <u>even</u> set based upon the whole sample and also upon the reduced sample. If we are doing anything psychological and not merely monkeying with sampling errors in an ad hoc way, the reproducibility of the truncated sample on the even set of items should rise, because what we have done, we hope, is to take out the persons who are organized in a very different way from the way most persons are organized. If we find that this increase of scale reproducibility has taken place as hoped, we go back to the odd-numbered set and chop off another fraction of our sample of patients on the basis of their position on the patient reproducibility distribution for the odd set. We then recalculate the even set reproducibility coefficient on the further reduced sample. We continue in this manner, throwing out cases on the basis of their individual reproducibilities on the odd-numbered items, moving progressively upward in the distribution of those odd-scale reproducibilities, until we get to a place where further purification of the sample does not cross-validate in the sense of showing an improvement in the reproducibility on the even set. At that point we conclude that we are beginning to capitalize on sampling errors and specifics.

The first thing that intrigues me about this procedure is the possibility that even in the personality field it might be possible to achieve extremely high reproducibilities of the sort that Guttmann originally envisaged when he invented his method. On the other hand, since the initial distribution of item difficulties and even the initial factor loadings reflect a kind of averaging out of the various sub-types of personality organization, they are in themselves partly erroneous. So that in using the method, it might be that one should purify

both the item set and the patient group concurrently. I am not clear on how to do this in a way which provides the necessary cross-validation at each step, but I suppose one might do it in the following way. In each cycle, we use the presently available odd and even sets that have survived from the previous cycle. When a rise in reproducibility on the even set is shown to occur as a result of excluding the least reproducible patients from the odd set, we conclude that we have somewhat purified our sample of patients, who are now more homogeneous in their mental organization than the sample we were dealing with at the end of the previous cycle. Having inferred this by the successful cross-validation, we go on to assume that this pure sample will give us a better idea of the item properties for the pure factor we are aiming at, and, therefore, we rescale the items in both the odd and even set on the basis of the item statistics for the reduced sample of patients. We throw out the items having low reproducibilities in this reduced sample; or, alternatively, we might compare the reproducibility of each item at the end of the previous cycle with its reproducibility after exclusion of some of the patients and see whether its reproducibility rises or falls. If its reproducibility falls, this presumably means that the item in question was behaving as it was on the basis of the contribution of factors which are operative in the cases now excluded rather than the idealized single factors surviving, so we throw those items out. We then go through the next cycle of identifying patients who contribute to lower reproducibility but now employing the reduced item set. In other words we alternate between purifying the patient sample and purifying the item set. Obviously, such a procedure as this requires a very large number of patients to work with, and we also have to begin with a sizeable number of items. On the other hand, it has always seemed possible to me that we have too many items on the typical MMPI scale since theoretically there comes a point (in any unweighted system of item scoring) in which the addition of further items which are relatively lower in criterion discrimination power contributes less to the separation of the group means than it does to intragroup variance. I have often thought that a 15 or 20 item scale would do better than a 40-50 item scale. I have some old unpublished research on this, done with Dahlstrom, in which we were using a criterion of neurosis versus psychosis and we found that while in the criterion sample every additional discriminating item improved separation, when we plotted an inverse overlap measure on a cross-validation sample, this hit-function passed through a maximum at scale length somewhere around 15 to 20 items, when the items are arranged in order of their absolute percentage discrimination in the criterion sample. In other words, you arrange the items (all "significant") simply in terms of their percent separation between neurotics and psychotics in the criterion sample, and you form scales by taking blocks of the best ten, the best 20, the best 30, the best 40, and so on up to the best 60 items, all 60 discriminating at the one percent level of confidence in the criterion analysis. You then plot the curve of group separation for these scales, each of which contain the previous scales of stronger items as a proper subset of itself, and that is where we found that the best scale on cross-validation is made up of the best 15 to 20 items on the criterion sample. So far as I am aware, nobody has researched this with more suitable criteria, which is one more example of a very important issue that has not been studied empirically.

I admit that I don't know where to go from here on the proposed purification procedure since I have been thinking in terms of the first factor, meaning of course the first factor which represents the kind of mental organization, i.e., the definition of a certain variable that is not a shared dimension in the sense that the other patients not

excluded do not locate themselves on it in terms of their item patterns. It's not clear to me what I expect to happen on this analysis if one now returns to the original set of items and deals only with the people who have by now been excluded. I suppose one can ask whether the anhedonia variable even emerges in a new factor analysis after one has taken out the pure cases who scale on an anhedonic set?

In the Ford Project this procedure might be combined with factor analysis of the Q-sort data to give kind of a mutual support for validity of the process. Presumably, if the bootstrapsing of scaleability by the above method were doing what I would hope it would do, one result should be that the factor pattern on the non-multiphasic data should begin to emerge more clearly in the sense of the varimax solution having a greater power as measured by some appropriate index such as proportion of variance accounted for by the first factor and the like. I haven't thought this through either, but you get the general idea.

114. I am not going to discuss configural scoring in general but will merely say that I remain convinced there is [merit] in it if it's done right. I am aware that several efforts (as by Ken Clark and Hal Williams) were not very encouraging, but we don't have to go too far to explain why that might be. And some other stuff, such as the work of Benno Fricke at Michigan, indicated that there is definitely something to be said for configural scoring. It may be that item doublets is not sufficient to get worthwhile configural effects, but with a huge N such as we have now in the University Hospital tapes and also in the Ford Project, and the possibility of rapid computer searching for pairs, triads, or even tetrads, I just can't believe it wouldn't work. I do think that one needs to optimize his odds by a little less blind empiricism, such as by selecting triads on some basis of content or on the basis of a preliminary factor analysis or whatever. One way to do it that I don't think has been tried is to pick item pairs such that the items in a pair are very different in either their difficulty level or in their apparent factor composition when these properties are investigated in two different groups of patients. Thus if two different items have about an equal difficulty level, say 60% among normals but among diagnosed schizophrenics one of them has a difficulty of 70% and the other of 30%, I should think such an item pair would be a good one to study configurally. (You remember the method proposed in my original paper on configural scoring was a direct study of the four-fold tables, in which we select item pairs on the basis that the phi-coefficient between the two items in one criterion group differs markedly from its value in the other criterion group.)

I also think that if the configural scoring method has any psychometric advantages in the personality field the diagnostic problem where we might expect it to be most useful would be in connection with schizophrenia since the impairment of "personality integration" is most marked in the schizophrenic and, speaking intuitively, I would suppose one reflection of personality disintegration is the failure of traits to "go together" the way they do in most people.

115. There are some miscellaneous MMPI ideas to be found in my memorandum to Jack Powers dated July 17, 1961, pages 9-ff. I think there is nothing much there that presently charges my batteries that I have not included in the present memo, but you might want to have to have a look at this old one.

116. We talked about the fantastic separation reported by Rodnick and Shakow using a somewhat ad hoc composite index of reaction time, and I wrote to Rodnick about it (April 7, 1966) and got his reply (April 20, 1966). Rodnick says that he did relate the reaction time index to an estimate of the psychiatric adjustment of the patient based upon

the psychiatrist's rank ordering of the patient's ward adjustment unconaminated by reaction-time index. Rodnick says he was very surprised to find a rank order correlation of about .90. He says that Shakow and Zahn at NIMH have also found a marked correlation between an index of "psychiatric status" and the reaction time index. He seems pleased by this, but for our family-study purposes this is presumably not desirable. I am not worried mainly about the fact that the psychosis must be greatly boosting it, but mere that this would suggest that nuisance variables (rather than any more fundamental neurological impairment) are mainly responsible for the effect. Hence we would expect too many false-negatives among well-compensated schizotypes, and too many false-positives among non-schizotypic neurotics, especially those in whom anxiety or depression played an important role. The more I think of it, the more it seems to me armchair obvious that no matter how much you soup it up mathematically, a study of reaction time is almost certain to be influenced by non-specific nuisance variables of this kind.

116a. I think we did talk briefly once about Wolf and the facial asymmetry business, which as he described it (when I took his graphology course) was very powerful, but you can't trust Werner Wolf very far in his validity claims. I guess I sort of like this because it gears in with my intuitive, fuzzy notions of "poor integration," and I think in terms of Bleuler's dramatic examples of patients who smile with one half of the face and weep on the other side and the like. Wolf's procedure was to split a person's photograph down the middle and then fuse the two right halves to make one composite photo and the two left halves to make another composite photo. How to deal with this psychometrically, since presumably almost anybody's face, however asymmetrical, would at least be recognizably very similar in the two fake photos? I suppose the easiest way to do it would be to present the pairs to a sizeable group of subjects, like a class of students, with fake instructions to the effect that they were going to see pictures of twins and their task was to say whether the twins were fraternal or identical. I would think Wolf's sign has the disadvantage that in younger people there hasn't been sufficient time yet for the facial lineaments (which are after all mostly products of mimetic behavior) to develop a marked asymmetry; whereas in older people we begin to get the influence of coarse brain lesions of a sub-clinical type. But I would like to put a student on this because I have always thought that someone should try to replicate Wolf's findings. He was a very bright man.

117. Related to the Wolf business on facial asymmetry is the "corrugator phenomenon," which Viktor Frankl says he originally described in 1935 (courtesy Schofield). The phenomenon consists of fibrillary twitches of the corrugator muscles, and Frankl considers it a "typical"sign of incipient schizophrenia. Frankl here gives himself credit for picking up one muscle group, but Bleuler mentions a fibrillar phenomenon in the facies, and I was fascinated to notice on recently re-reading Ole Kraepelin (Lectures on Psychiatry, 1906!) that in his clinical demonstrations of "dementia praecox" he pointed out the occurrence of small twitchings of the facial musculature as a diagnostic sign in two patients.

I may have mentioned to you my observation, years ago now, that state hospital "crock" cases often show some sort of a funny business about their lines in the forehead which I don't know how to describe accurately except to say that it's sort of a spidery, asymmetrical, loused-up business (partly angle-lines instead of vertical or horizontal) which again we cannot assume would appear except after many years of aberrated

mimetic activity. And most cases do not show it, even late. However, it occurs to me that one might get at this sort of integrative defect or motoric dyscontrol of the facial muscles by some movie camera procedure supplemented by marking certain reference spots on the patient's skin, and then running through the standard neurological items such as "wrinkle your forehead," "shut the eyes tightly," "smile," "frown," "clench the teeth," and so forth. I admit this is kind of crazy far-out idea but the tie-in with neurology makes it theoretically interesting. Slow-motion or mathematical analysis of the reference-spot motions might reveal the defective mimetic integration.

118. Looking over responses to the sentence completion test out at the VA Mental Hygiene Clinic several years ago, I developed the notion that the schizophrenic's evaluation of other persons probably involves an altered distribution of positive and negative cathexis in the sense that he considers some human characteristics much more important than others and that this preference is not the same as is found in normals. I have in mind, for instance, the fact that for another person to be "insincere" or "hypocritical" seems to be particularly objectionably to schizotypes and I suppose this is because of the massive distrust phenomenon. A preferential Q-sort of adjectives or brief phrases characterizing other people's attitudes and conduct might be worth looking into. Here again, my main worry would be that while it would discriminate schizophrenics from controls, it would not be sufficiently specific, with the result that many garden variety non-schizoid "introverts" and several types of "neurotic" patients would show up as false-positives.

119. I can draw this list of indicators to a suitably psychotic close by mentioning a wacky idea I have about delirium. This really doesn't bear directly upon family studies, but it does bear upon the problem of a disposition to cognitive slippage. While influenza is not an important etiological factor in schizophrenia, the great flu epidemic of the 1920s did apparently precipitate schizophrenic disintegration in a rather large number of individuals who, up to that time, had remained compensated in terms of gross clinical criteria. There are patients in the state hospitals of Minnesota even today who were apparently mentally healthy until they fell ill of influenza sometime in the early 1920s as young persons, and the delirium associated with the infectious disease did not resolve itself. (As Menninger puts [it] in discussing this epidemic, "Schizophrenia is a long delirium.") Now everyone knows there are marked individual differences in the readiness with [which] people develop delirium — technically, as you will recall from the standard nomenclature, delirium is a psychosis even if it only lasts for ten minutes — when they fall ill of a toxic-infectious illness. It occurs to me that it would be fascinating to study the relation between MMPI profiles (when clinically well) of patients who developed fever delirium in connection with an infectious disease in comparison with patients of comparable age who suffered from the same infectious disease and whose recorded chart temperature at the time of the appearance of the delirium was equal to theirs. It is even conceivable to me that a certain critical temperature would be involved in the development of delirium among schizotypes which would be different from the critical temperature for other patients (cf. Hoaglund on pacemaker parameters in fever-treated paretics). I think that Health Service data would be the best here because we have MMPI records on almost all students which were obtained when they were freshmen. This is a nice "file study" which it would be worthwhile putting an eager-beaver undergraduate medical student on working up.

How Schizotaxia Leads to Schizotypy (Speculative Psychophysiology)

120. In my memo to Feigl anticipating Carnap conference [Meehl to Feigl, March 1, 1966, "Some stomach-aches of psychologies concerning correspondence rules, . . . open concepts," especially Sections 18-20] I used the open concept schizotaxia to illustrate one of three types of conceptual openness, that of progressively "filling in Orphan Annie's eyes," i.e., providing additional explanatory text and/or formal postulates which enrich the partial interpretation of theoretical terms that are implicitly defined [better: whose specification of meaning is contextually provided] by an avowedly incomplete nomological network. Orphan Annie's eyes are the open circles located at the nodes of the postulated network, the law-like functional relations between the theoretical entities ['entity' covers structures, events, states, and dispositions] being the strands of the network and the entities themselves being the nodes. In a pure upward seepage epistemology, the entire empirical meaning of the theoretical constructs is given by their formal role (i.e., locus in the network) together with the fact that some of the terms (or, on another interpretation, some of the theorems) are linked to observational terms (or to observational statements) via so-called "meaning-postulates," "correspondence-rules," "operational definitions," or "bridge-laws." Without such linkages to the data-base, the network would not even be an empirical theory, let alone a useful one. But I do not subscribe to the upward-seepage view, which is called so because all of the empirical meaning is considered, on this view, to "seep upward" via the network's formalism (plus the correspondence-rules) from the observational statements. Like you, I hold that only a part of the interpretation of a theoretical term comes from this combination of formal role + operational definition, and that, in all sciences, additional meaning is usually provided by interpretative text, often of a "model" or "analogical" kind, as Mary Hesse, Feyerabend, and other philosophers contend. In what follows I presuppose this interpretation of scientific theories. [Note added 5/29/66: C. G. Hempel, long a defender of the upward seepage doctrine, seems now to have abandoned it, if I correctly understand the paper he read at our recent Philosophy of Science Center conference on interpretation and confirmation of theories.]

In the Feigl memo [Section. 20] I sketched out such a sequence of progressive additions to the model, each one presupposing the cumulative interpretation which precedes it, but not logically entailed by the preceding. In such a process Orphan Annie's eyes are being gradually "filled in" by interpretative text which restricts the model progressively, thereby becoming more "risky" in Popper's sense, because the enlarged theory asserts more (has more content) at each stage of interpretative increment.

It is necessary to emphasize that a rather minimal ("weak," "very partially interpreted") theory will suffice for purely genetic research. That is, one need riot proceed very far in the sequence of additional textual interpretations to generate family-concordance predictions which can function as corroborators or falsifiers of the genetic theory. In such a case we are (like Mendel speaking non-committally of "factors") getting almost all of the empirical meaning of 'schizotaxia' via upward seepage. But not quite all, because we already take terms like 'gene' from genetics itself, hence the received network of interpretations involving genes occupying positions on chromosomes, population gene-frequency, mutation-rate, selection pressure, and so on come into the interpretative text automatically. Furthermore, my denial that schizotaxia is a coarse brain disease, and my assertion that it is a "neural integrative deficit," vague as these

statements are, do provide something by way of interpretation beyond what pure upward seepage from phenotypic statistics alone would give.

But this is not enough, for two reasons. First, in the context of justification, a theory of schizophrenia can hardly be satisfactory as an explanation of the disease unless the theory gives some account of why the major symptoms are found together in this group of patients. So that, while we do not demand a complete mathematical postulate set setting forth in all details exactly how 'hypokrisia' works (e.g., the numerical coefficients in the differential equations of Olds(+) and Olds(–) feedback), we surely want to provide some plausible explanation of why hypokrisia gives rise to such striking phenomena as anhedonia and diffuse, refractory interpersonal aversiveness. It just won't do to merely postulate intrinsically unrelated causal relations like this:

> Hypokrisia leads to cognitive slippage.
>
> Hypokrisia leads to anhedonia.
>
> Hypokrisia leads to ambivalence.
>
> Hypokrisia leads to interpersonal aversiveness.

If 'schizotaxia' were left as a very open concept, saying no more about it than that it is a neural integrative defect (a) predisposing to schizophrenia [output anchoring] and (b) caused by a dominant gene [input anchoring] such a minimally-interpreted construct could be corroborated by family studies, if they came out as beautifully as I imagined in the Feigl memo [Section 18]. And it would even be fairly safe, given such gorgeous data, to view the inherited defect as "neurological," arguing from the fact that its neurological indicators "follow the gene" rather than following clinical status or social and psychodynamic variables, or thematic content. However, such a situation would gratify the geneticist more than the psychologist! [Analogy: A showing that diabetes fits a genetic model, with no knowledge about Islands of Langerhans or glucose-tolerance curves, would not be a very satisfying theory to a physiologist, a pathologist, or a biochemist.] Furthermore, while 'schizotaxia' has been introduced mostly by upward seepage (plus a leetle vague reference to its being neurological-but-not-coarse-brain-disease), the 'hypokrisia' construct is a different kettle of fish. 'Hypokrisia' spells out — however speculatively — what the neural integrative deficit consists of, in terms of synaptic control functions. And while the terms employed in that definition are terms possessing antecedent meaning from another science (and, hence, the construct would be "empirically meaningful" to a logician even if no molar derivation to schizophrenia had been made), yet in a genetic + psychological theory of the sort you and I care about, hypokrisia would be a kind of fake construct (i.e., it would have no intelligible connection with the molar phenomena we originally set out to explain). Roughly, one could put it thus: For the behavior-geneticist, 'schizotaxia' is sufficiently interpreted as dispositional + neurological; 'hypokrisia' doesn't add anything in his bailiwick. But for the psychologist-with-genetic-interests, 'hypokrisia' doesn't add anything to theoretical understanding, unless some explanation of its relation to the molar phenomena of schizophrenia is included in the picture. Without this, 'hypokrisia' (while meaningful) is a scientific supernumerary.

[NB: I presuppose throughout the elementary distinction between the empirical meaning of a statement (its semantics) and the available evidence (its current epistemic status). Some psychologists are confused about this, having been brainwashed into a simple-minded positivism which even 1928-Vienna-Circle didn't hold. Obviously a well-

formed-formula stated in neurological language is "empirical," even though there may be no evidence presently available to confirm or refute it.]

The other reason why we want more than pure dispositional, upward-seepage context for 'schizotaxia' is in the context of discovery. A blind empirical trial-and-error search for schizotaxic indicators is surely less likely to succeed (or, if it does, the search-process is going to be less economical) than a search guided by tentative interpretations, however vague, open, and speculative these may be.

121. Since this is a context-of-discovery memo, I shall not apologize for the presence of numerous far-out guesses, over-simplifications, analogies, or appeals to clinical experience. If I happen to recall a gem from the research literature to illustrate a point, I don't claim to know how representative it is in all cases. As you know, my semi-Popperian philosophy of science differs from that prevailing in psychology. The so-called "positive, inductive support" for a theory may be very slight, since, as Popper says, the "justification" for propounding a theory need consist of nothing but the existence of a problem. The theorist has the responsibility to show that his theory is testable, and that's all. So if someone asks, "But, my dear fellow, where do you get such ideas?", my Popparian reply would be, "Get them? I didn't 'get' them — I just made them up out of my head!" So that when I cite "evidence" in what follows, you should view it mostly as (a) Defining the problem-situation, and (b) Suggestive in the context of discovery. So much by way of apologetic meta-talk. On to the speculations!

Much of the following is repetition, with modification and elaboration, of my memo to Janecek [Meehl: "Schizophrenia and vestibular function," July 7, 1961] and of my APA-prexy speech. You may wish to refer to the latter, but it was so densely written that parts of it I can hardly follow myself. More important is the fact that I now reject neurological theories number one and three, and am betting on number two, 'hypokrisia,' to do the core job. But of course part of the first theory, primary anhedonia due to an Olds(+) deficiency, I now include among the polygenic potentiators of schizophrenia, but not schizospecific, as discussed in Section 17 above.

122. What are the big facts that define our problem-situation? You and I hold that, except in rare (and lucky?) instances, it is poor strategy to theorize — or to test theories — by paying attention to small trends or barely detectable effects. Focusing attention on such small trends becomes important in the advanced state of well-corroborated and tightly-knit theories, when the scientific task is to refute the received doctrine by pushing it to the limit, knowing that all theories are lies, but some are blacker lies than others. Newtonian physics was a very white lie, and it would have been bad strategy for Newton, Laplace, et al., to begin by investigating the minor perturbations of planetary motion. Or suppose Robert Boyle had done a significance test on the relation PV = K with high-pressure values included? 'Nuff said. So we don't start by trying to explain all of the umpty-ump trends (mostly showing 95% group overlap, I daresay) reported in Bellak's book. To encompass all this stuff even in one's memory is impossible, let alone to explain it theoretically.

123. So — the "big facts." For theorizing about hypokrisia, I am going to take as my first big "fact" what is not yet highly corroborated, namely, that the schizotaxic disposition is genetically determined.

124. I also take it as granted that we are not dealing with coarse brain disease, nor with a defect in storage, nor with a defect in CNS "complexity." The fact that Kurt Gödel (and, as I see him, Isaac Newton) were schizotypic — the former is, at times, clinically schizophrenic — pretty much rules out defects of this sort.

125. Another big fact is that schizotypy and schizophrenia consist, behaviorally and phenomenally, of socially learned contents (i.e., you cannot inherit a delusion about Jesuits or a hypochonriacal pre-occupation with food fads).

126. Another big fact is the etiological significance of intra-family interaction patterns, especially the potentiating influence of a schizophrenogenic mother. (Although here see G. H. Frank, "The role of the family in the development of psychopathology," Psychol. Bull., 1965, 64, 191-205 and reply by M. Zuckerman, "Save the pieces! A note on 'The role of the family in the development of psychopathology'," Psychol. Bull., 1966, 66, 78-80.) I believe that almost all statistical studies of family factors (and ditto for most experimental studies) are, and must be, intrinsically ambiguous as to influence of genes versus social learning. The designs are incapable, methodologically, of unscrambling the causal chains. Two generations ago family background data were uncritically taken (by scientists with a heredity bias) to establish genetic influences. Today, analogous studies are uncritically taken, by behavior scientists with an environmental bias, to prove social learning influences. You and I are agreed, I think, that etiological disentanglement cannot, in general, be achieved by methodological emphasis upon thematic content, nor by comparisons of parental behavior toward a schizophrenic proband with that toward a healthy sib. A little sophisticated reflection shows all such designs to be systematically ambiguous. Looking at it in this light, I am not myself convinced that there is any really compelling evidence for the schizophrenogenic-mother concept. But I am willing to include it as a "big fact" on theoretical grounds! It is hardly conceivable to me that a behavior disorder in which interpersonal aversiveness and massive distrust are so central could be uninfluenced by the earliest social interaction with the primary nurturing figure, mamma.

127. Another big fact is (I shall rashly assume) the occurrence of "soft" neurological aberrations not wholly dependent upon clinical status.

128. Then of course we have as very big facts the core phenomena of schizotypy itself, which I take as consisting of the tetrad: cognitive slippage, anhedonia, interpersonal aversiveness, and ambivalence.

129. Then we have the big fact of prognostic malignancy. It is as true today as in the days of Kraepelin and Bleuler that if you are picking a mental disease to come down with, you would be damn well advised to pick something other than schizophrenia. With the possible exception of chemotherapy (which pretty clearly affects at least short-term status — as, I am convinced, psychotherapy does) I do not believe we can assert that there is any known method, organic or psychological, to influence materially the long-term course of a schizophrenic illness.

130. These big facts define our problem situation. The first strategic issue arising is where to plug in the schizogene's influence? Since we know that many — perhaps most, or all — genes are pleiotropic, one could simply postulate that the gene independently affects several neurological functions, also the affective parameters, also the cognitive parameters. But this 'would be pure postulation ranging over the explananda, and not very intellectually satisfying. It has, to quote Bertrand Russell, the advantages and

disadvantages of thievery over honest toil. Alternatively, one could take one of the molar "big facts," say, the interpersonal aversiveness, and attempt a purely socio-psychological derivation of the others. (This is the line taken by most non-biological theorizers, as is understandable.) I view this approach as somewhat more promising than postulating pleiotropism, but I remain unconvinced that the amount and more importantly, the unique quality of schizoid cognitive slippage will ultimately be explainable solely on such a social basis. And (anticipating the data!) I am assuming that compensated schizotypes will manifest both "soft" neurological signs and subtle cognitive slippage, even if they are less anxious, less depressed, less withdrawn, less preoccupied, etc., than some non-schizoid neurotics. Hence in the diagram in Section 60 I postulate a direct relation running from hypokrisia to both cognitive slippage and aversive drift.

[NB: Some clinicians have misunderstood me on this point, perhaps by too-easily assimilating my views to those of writers like Babcock. I do not make cognitive slippage the cause of aversive drift, any more than I make aversive drift the cause of cognitive slippage. Each of these, as a molar defect, I view as flowing, quite independently, from hypokrisia, the neural integrative defect. I believe schizotaxics would learn to be afraid, mistrustful, and anhedonic even if they could think and perceive straight; and, similarly, I believe that they would think and perceive crookedly, and have screwy associations, even if they were unafraid and capable of normal pleasure. The latter point is already clear to me from clinical experience, because a therapist who takes the facts as they come cannot fail to note that some neurotics in some states are more anxious, angry, etc., than some schizotypes — yet the neurotic does not, and I believe cannot, "slip" cognitively to the same extent or in the same way.]

131. However, clinical experience and experimental research persuade me that these two molar aberrations, while primarily flowing from hypokrisia independently, do subsequently potentiate each other. Hence in the diagram I have a place for "secondary cognitive slippage" and "secondary aversive drift," and causal arrows run in both directions between them. Even if we had no psychometric or experimental studies, I would rely confidently upon my clinical experience to put these molar-interactions in the theory. It is perfectly obvious to me that, at times,

a. Schizotypes perceive wrongly and think poorly because something has made them feel frightened, angry, deprived, rejected, guilty, or ashamed; and that

b. Schizotypes feel frightened, angry, deprived, rejected, guilty, or ashamed because they perceive wrongly or think poorly.

I have no theoretical stake in the question as to which of these causal directions predominates, although I believe that most of the time they run both directions simultaneously (as they do in normal persons, for that matter!).

So, granted that an over-learned set of "bad habits" of aversive feeling and screwy thinking tend to exacerbate one another in the acculturated schizotaxic [= schizotype], I take it that there is a primary tendency to cognitive slippage and a primary tendency to aversiveness, flowing directly from the neurology of hypokrisia.

132. As I see it, putting the "emotional" and "intellectual" facts of schizophrenia together in terms of a single genetic aberration constitutes the theoretical puzzle we face. We are so accustomed to the schizoid syndrome that we perhaps do not react to this association as being a big puzzle. But it is, isn't it? Phenotypically, these facets of the

syndrome have nothing in common, no obvious "content-overlap." If we assume — as we surely must — that the schizogene acts directly upon a parameter of CNS function, not upon "content," the behavioral/ phenomenological combination: [cognitive slippage + anhedonia + interpersonal aversiveness + ambivalence] is really a puzzler. I ask myself, what the heck could be wrong with the control parameters of neurons that would generate this particular molar configuration? Why not hyper-hedonia, for instance? Why not symmetrical ambivalence, without aversive bias? Why not somatic aversiveness (n Harm-avoidance) as much as interpersonal? Why is the schizotype so much more mixed up about people than about, say, geography? (No doubt these are among the considerations that lend so much plausibility to pure social-learning theories of the disorder. And they are powerful ones, admittedly.)

133. If I have even a semi-insight into this mystery, it could be summarized by the following: The hypokrisia gives rise directly to primary cognitive slippage; primary aversive drift arises when a mixed schedule of positive and negative reinforcements is imposed on a hypokrisic nervous system, because of the algebraic asymmetry in the quantitative effects of hypokrisia upon positive and negative Olds-center feedbacks; and this aversive drift is especially marked in the interpersonal domain because this domain typically provides the most mixed, unpredictable reinforcement-schedules. This is Meehl's theory of schizophrenia in a nutshell. And I must confess that, mathematical and neurophysiological details aside, I experience a persistent, strange [and quite unjustified!] conviction that something like this is rather close to the truth. Sinning against Skinner, I hypothesize that

a. Cognitive slippage is primarily due to "synaptic slippage." In a way, I solve the first half of the molar-correlation puzzle by taking the [hypokrisia ⊗ cognitive slippage] sequence to be obvious, which it is, isn't it? Even without details, we could very plausibly argue that if the individual tubes or transistors of a computer are aberrated in their mutual influence properties, the computer will tend at times to mal-compute. This being granted, we have remaining as our main puzzle the second half of the molar-correlation — why the aversive drift? Of course the details as to primary cognitive slippage have to be worked out too. I refer here only to the broad outline of a solution, which looks easier for cognitive slippage than for the affective-motivational phenomena.

b. The effect of a sequence of scrambled appetitive and aversive inputs on a brain with "synaptic slippage" is biased aversively, because of the difference in algebraic sign between corticopetal Olds(+) and Olds(–) feedbacks.

Too simple? Mebbe so — but I guess the simplicity is partly what appeals to my intuition! The mere possibility of pulling together the superficially dissimilar facets of the schizotypic tetrad by invoking nothing but "slippage at the synapse," given the known fact that social reinforcement regimes are invariably probabilistic (and often even concurrently mixed) has a strong cognitive appeal.

134. Expanding this somewhat, there is a chain of reasoning as follows: Synaptic slippage [= hypokrisia] explains cognitive slippage, or at least an unusual readiness to acquire it, and to exhibit it under suitable motivational–affective states. Synaptic slippage also explains ambivalence, anhedonia, and pain-dependency, all three of which are roughly formulable in terms of a "mixing," "scrambling," "fusion," "mutual-watering-down" or "cancelling-out" of plus and minus hedonic tones. The puzzle lies in the bias

toward negativity. Prima facie, hypokrisia is "symmetrical"; it would lead to spillage of plus into minus and minus into plus. Whence comes the schizoid tendency to aversive drift? The theoretical consequence that there will be a tendency to "mixing" or "hedonic confusion," with bidirectional effects, is not itself adverse to the theory, since such hedonic confusion has long been recognized as one of the strange facets of the disease. But that this confusion is, in addition, typically shifted toward the aversive pole, is equally well known; and it is this bias which appears, at first glance, difficult to derive from a "neutral" aberration like hypokrisia. One thinks, "Hypokrisia will attenuate the adequacy of inter-assembly controls, so that the controllee assembly-system over-reacts, or is less stringently modulated by its driving input. Thus, we expect pulses from exteroceptive projection areas to Olds centers to elicit hyperactivity in the latter, whether the latter are Olds(+) or Olds(–) centers. Both positive and negative Olds-center activity will be exaggerated by hypokrisia. So whence comes aversive drift?"

My solution is to reason that the exaggerated, inadequately modulated Olds-center activity will start out as symmetrical, but will not remain so. That is, hypokrisia will yield faster linkage growth and, for a <u>given</u> linkage, heightened Olds-center activity for a <u>given</u> perceptual-system activity. And this exaggeration will, initially, be equal for Olds(+) and Olds(–) center control. The asymmetry arises <u>from that point on</u>, because Olds(+) activity provides facilitative feedback to the driving (and other coactive?) systems; whereas Olds(–) activity provides inhibitory feedback to the driving (and other coactive?) systems. Hence a heightening of Olds-center activity, attributable to the "neutral" fact of hypokrisia, becomes a non-neutral fact at the next step of the chain; because an exaggerated Olds(+) feedback has the opposite effect from an exaggerated Olds(–) feedback. Next inferential step: Under predominantly positive controls, an exaggerated inhibitory feedback tends to suppress controller-system (and other coactive?) function, and hence to retard linkage growth. Similarly, under predominantly negative controls, an exaggerated facilitative feedback tends to excite controller-system (and other coactive?) function, and hence to accelerate linkage growth. Aversive drift arises because exaggerated activity in Olds(+) versus Olds(–) centers is opposite in feedback sign. If all were excitatory in function, I could not derive aversive drift from the "neutral" construct of hypokrisia.

Since the derivation of an aversively biased hedonic confusion involves the reinforcement regime's being "mixed," we further infer that the more mixed it is, the more aversiveness and confusion there will be. And I take it as a fact that, in general, interpersonal regimes are the most mixed — both in the sense of singly ambiguous cues, and in the sense of frequent co-presence of positive and negative cues.

We do not infer that aversive drift will be absent in other, non-social domains, nor that cognitive confusion will be lacking in them. We even allow that, in some patients, accidents of life-history (or extreme non-specific constitutional parameters) will render other life-domains aversive to as great a degree as the social (e.g., somatic concern and <u>n Harm avoidance</u> in those misdiagnosed schizophrenics commonly labelled "hypochondriasis"). But this requires a rather special concatenation of circumstances, whereas <u>all</u>, social-reinforcement schedules are badly scrambled for <u>all</u> of us. Hence the statement in my APA-prexy speech that all schizotaxics become [= learn to be] schizotypic <u>on all actually existing social regimes</u>. The relation between schizotaxia and schizotypy is one of bi-conditionality, i.e., $(p \supset q)$ and $(q \supset p)$; it is <u>not</u> a relation of nomological necessity $(p \overset{c}{\longleftrightarrow} q)$.

135. It is hopeless to try sketching out a "complete neurophysiological theory" in the present state of knowledge (human knowledge, let alone mine!). I am inclined to speculate on the main outlines, relying on a combination of

a. Engineer's block-diagram notions as to "how a mammal's brain has to work if it's to do its job, on the average."

b. Gross molar behavior generalities about learning.

c. The main results of more direct study of brain function, especially the Olds-type stuff.

d. Clinical impressions about schizophrenia.

e. A modified Hebbian view of CNS processes in the small.

136. I assume that something like Hebbian cell-assemblies exist, and that they are formed by a combination of genetic wiring (especially mere anatomic closeness, perhaps with quasi-random connections initially) and repeated concurrent firings. Like Hebb, I doubt that "reinforcement" is necessary for the consolidation of perceptual assemblies, sufficiently massive and consistent repetition of inputs being enough. I think of a specific synaptic locus (say, under a single terminal knob in the synaptic scale) as developing a "Hebbian increment" of control whenever a pre-synaptic spike reaches that locus in a proper time-relation to the passage of a spike-wave over that region of the post-synaptic cell. The result of building up such Hebbian increments at a synaptic locus (whatever may be the structural change involved, but there must be a structural change, since deep anesthesia or EST do not destroy the old learning residues!) is that on subsequent occasions, a pre-synaptic spike arrival at that locus produces either a larger or a longer-lasting local disruption of the controllee cell membrane. Since we know that the firing of a cell normally involves a suitable spatio-temporal "patterning" of such local disturbances, which fades out without reaching spike threshold unless they are combined with other local disturbances suitably timed-and-placed; it follows that the accumulation of numerous Hebbian increments at various loci brings the post-synaptic ("controllee") neuron under increased control of future same, or similar, patterns of confluent pre-synaptic impulses.

137. Consider a cell-assembly which has undergone enough "consolidation" (internal reverberatory cell-to-cell control such as Hebb theorizes) so that it functions as a unit. Or, at a level more suitable for our purposes, a system of cell-assemblies, linked either serially or with mutual feedback connections. Thus, in the Hebb-Pritchard retinal stuff, a "meaningful molar unit," such as perceiving part of a human profile, would consist of an inter-linked system of cell-assemblies. I postulate that, even though a well-consolidated assembly or assembly-system [henceforth I shall refer merely to a 'system,' which term will cover the single cell-assembly as a limiting case] is internally knit and tends to function, as a unit, it is nevertheless capable of different levels of activation. This is, I think, almost unavoidable from its structure. The assembly's elements differ in their "centrality," number and diversity of linkages, and two activation-intervals will not be identical in composition when some of the elements are in absolute or relative refractory phase, and so forth. It is generally thought that individual neurons probably differ in spike threshold and similar parameters. Some measure of average rate of unit activity (no profit in guessing at this function) characterizes the activation level of such a system during a short time-interval. I take it that if two anatomically remote systems become linked (e.g., part-percept of mother's frown with an Olds(–) center) by repeated co-activations, then at

any given stage of such linkage-growth, the activation-level of the controllee system is an increasing function of the activation-level of the controller system. That is, I think of the controller system as literally driving the controllee system. Such differential (graduated) control is mediated by the usual combination of (a) Number of fibers activated, and (b) their average frequency of discharge [and its standard deviation? Probably relevant too].

138. What must be the quantitative properties of the linkage-function between two systems? Again there is little point in speculating about details, but I think at least a bit can be said. For one thing, the controllee activation cannot increase without limit; so the effect of input increments must decrease at some point. Besides, in statistical processes like this, whether of biological or social systems, we usually find the economist's "law of diminishing returns, that is, after a sufficiently large number of elements have got into the act, we reach a point where the incremental payoff from additional "cooperators" is lessened. Neurophysiologically, this seems very plausible, as Hebb and others interested in nerve-net speculations have pointed out. The wiring plus the learning are both statistic-cal, with "safety factors" built in. When, a consolidated controllee system is being driven at high activity, its own internal linkages are doing a large part of the work of driving. Similar but distinguishable input patterns from the controller system may each have reached maximal linkage. "Over-driving" may disrupt the normal replacement of units in refractory phase. Perhaps most important is the fact that neurons fire "to capacity," so that if a spatio-temporal input pattern to a single unit suffices to get it over spike threshold, the fact that there are additional inputs makes no difference to the cells which are one step removed in the chain of control. So that at very high controller activation levels, much of what happens at synaptic loci in the controllee system amount to mere "works of supererogation." If I recall correctly, this point goes back to Sherrington, if not before, in connection with his concepts of summation, convergence, recruitment, etc. Many such factors could be mentioned which, taken together with the obvious truth that controllee activation has a physiological limit, strongly suggest that after reaching some value of controllee activation = y, the linkage function $y = f(x)$ has a negative second derivative. And this fits with the generalization that practically all molar-data curves, whether of drive, perception, habit-strength, or whatever, become and remain decelerated at some value of the input variable (including input variables like "time" or "repetitions" or "occasions").

139. Is the linkage-function decelerated throughout its own empirical range? This is stickier to decide from the armchair, and fortunately we don't need to answer it. My hunch is that we start with a rising limb of positive acceleration, so that there is a flex-point where dy/dx changes sign from (+) to (–). I think this mostly on two theoretical grounds, either of which alone would suffice for the result, and which are not incompatible but tend to potentiate each other. The first is a sheer matter of probability. Oversimplifying, assume that each neuron in a controllee assembly requires the confluence of k spike-arrivals at the knobs of its synaptic scale during time-interval Δt in order to fire (we neglect position effects). Assume a quasi-random distribution of inputs over the controllee cells. Given a fixed mean frequency of controllee cell discharges, there is a probability p of a spike arrival at each locus during Δt. If there are n loci on the controllee cell, the probabilities of various input-concurrence numbers [0, 1, 2, . . . k, . . . n] are given by the expansion of $(p + q)^n$, which is quasi-normal for the usual n's involved in CNS synapses (e.g., in some ventral horn motorneurons it runs as high as 1200, which is input cells, not loci). If $np < k$, the average controllee cell does not fire. The proportion of controllee cells fired is

approximated by the normal curve integral up to k, and as this normal curve slides along the abscissa (with increasing values of the controller frequency) the integral is ogival. [I note in passing that this derivation also yields the deceleration of the linkage function y = f(x) after a certain point.] The fact that the input probabilities are not strictly random, and that the value k presumably varies over controllee cells, affects the parameters of this ogive but does not destroy the general conclusion.

The second reason for expecting an initial region of positive acceleration is, of course, the antocatalytic aspect. Even if the controllee assembly is incompletely consolidated (i.e., its internal accumulation of Hebbian increments is far from asymptotic), it is easy to see that recruitment of controllee cells (starting from zero input driving) will tend to yield a positive acceleration. At very low input levels, most of the controllee cells fail to spike. As the input activity rises, high-threshold controllee cells (or those receiving fewer controller-assembly inputs) are recruited because they begin to receive inputs originated intra-assembly (i.e., the assembly begins to "cook internally," rather like the critical mass situation in nuclear reactions). I have seen derivations of this effect from several kinds of postulated cell-assembly structures, and I get the impression that it is fairly general, in the sense that highly specific assumptions about the assembly-organization are not required ad hoc, but merely that the assembly does have multiple internal linkages (by definition!), and the accepted fact that most neurons require a properly timed confluence of pre-synaptic spike arrivals to get over threshold.

140. With regard to the accumulation of Hebbian control increments by coactivations, I believe I have a proof (not included here) that the increased probability of an increment developing due to increased firing-frequency is symmetrical as between controller and controllee, i.e., it doesn't matter which of the two cells is firing faster. But it seems that the variance (over cells) in net increments is not symmetrical as between controller and controllee. (I forget which way it goes, but won't look it up, because the consequences of this theorem are far from obvious.)

141. I postulate that the coactivation of systems which are linkable [= anatomically connected] builds up linkage between them. This is not really a "postulate," since it follows from the basic Hebbian postulate concerning development of local Hebbian control increments on single cells. If the systems are anatomically remote, the resulting linkage grows very slowly, and perhaps also to a very low asymptote. ["In the brain, everything connects with everything, but not much, on the average".] Linkage growth between anatomically remote systems is quantitatively important only if such growth is facilitated by strong potentiating inputs, notably Olds-center feedback and (I suspect) blips of "general arousal." Hence the quantitative impressiveness of "reinforcement," "emphasis," "excitement," and such-like factors when we study the acquisition of instrumental acts or (alleged) sensory-sensory conditioning, "incidental learning," and so on — in contrast to the development of fundamental perceptual units, intra-modality expectancies, or, as Jim Jenkins would argue, many meaningful verbal connections that the child acquires by hearing, without either emitting them (or, even if he does so, without being reinforced therefor).

142. In spite of the present experimental complexity (and even some confusion) about Olds-center function, which seems to get worse rather than better as the facts [?] multiply, I rely upon the block-diagram plus crude molar-behavior generalizations here, and rush in where angels fear to tread. What must a mammal do to stay alive and

reproduce, "on the average"? Suppose no one had ever studied the brain, or even studied molar behavior experimentally. Given the three biological facts that (a) Mammals don't come "all-set-to-function" as much as invertebrates, (b) The environment offers probabilities of need-satisfaction under fluctuating conditions, and (c) The environment presents threats of danger by other members of the species, by predators, by inorganic catastrophes; can't we say some pretty plausible things about our block-diagram from the armchair? The two kinds of machinery clearly required for things to "pan out" on the average are: A feedback mechanism which strengthens cognitive and instrumental processes if they (tend to) result in need-relevant consequences; and a feedback mechanism which weakens (in the long run) and stops (in the short run) processes that result in danger. Teleologically, the latter has a higher priority, since (except for oxygen) an organism can postpone need-gratifications, but most danger-situations have a kind of "emergency" feature to them. I assume that this difference explains why God or evolution has wired the mammal in a somewhat different way with respect to parameters of the appetitive and aversive control-systems. Without prejudging the technical controversies in learning theory, it seems generally agreed that there is a difference here. "Positive reinforcement" generates habits, expectancies, and cathexes; these are then activated by fluctuating motivational states. "Extinction," as Skinner emphasized in Behavior of Organisms, is a different sort of thing from "negative reinforcement," the latter having as a short-term effect to produce stoppage of whatever is getting the animal into trouble, and the long-term effect of rendering a danger-associated cue a stimulus whose removal acts as a positive reinforcer.

143. Both positive and negative reinforcers seem to have an "arousal" function, in addition to their more specific effects as strengtheners or inhibitors of specific instrumental acts. As I see it, we normally deal with a delicate balance between the "stoppage" effect of a danger-signal and its "arousing" effect. Teleologically, what is the biological requirement here? When an organism does something that puts it in immediate danger, the obvious requirements would seem to be these:

a. Whatever it just did should be, at least for now, suppressed (e.g., quit pressing the lever if doing so currently results in being shocked)

b. Riffling through other available acts which fail to turn off the danger signal should result in fairly quick suppression of each of them

c. But as long as the danger signal persists, the organism must "keep trying" [I set aside that important class of defense-behaviors that, for some species, involve immobility.] Hence arousal is necessary.

d. A certain "alertness" and "heightened discrimination" is also desirable — hence the reduction of two-flash threshold as a good arousal measure

e. but protective inhibition — general closing down or turning off — may be useful last ditch measure [pencil addition, not PEM handwriting]

144. How the CNS parameters are (as in all biological systems) wired-in so as to yield "healthy" average values for these effects, we know that overshootings, oscillations, etc., such as analyzed mathematically in servomechanism theory, often occur. As Ashby says, it is not a defect in his (or any other) theory that it won't explain perfect and universal adaptation, since we know that maladaptations do occur. One trouble with Rado's type of conceptualization is that the adjustive or adaptive concepts are built into the theory —

whereas such concepts should, at most, be used in context of discovery, when building models that "work" on the average. (Cf. my remarks on "biopragmatic" concepts in Meehl, P. E. Psychopathology and purpose, The Future of Psychiatry, pp. 62-63)

145. One of the most striking malfunctions, relevant for present purposes, is the "spread of massive inhibition" under extreme threat. Freud (Inhibitions, symptoms, and anxiety) thought of the impaired ego-function of neurotics as partly attributable to this, in the sense that their id-impulses produce such a chronic heightening of the defense [= inhibitory] mechanisms that even rather content-neutral acts or cognitions are impeded. Social psychologists' interviews with normal persons who have witnessed a crime or been involved in a catastrophe (fire, earthquake) suggest that when the aversive Olds(–) feedback is sufficiently strong, the organism will undergo both an "instrumental paralysis" ["I knew what to do, but I couldn't seem to move"] and a cognitive-perceptual stoppage ["I got test panic, and my mind went blank," or "When it happened, it seemed like I couldn't hear anything, and my visual field shrank to where I literally couldn't see anything but that revolver"]. And of course Pavlov made a great deal of the "spread of inhibition" in his work on neurotic dogs.

I stress this partly because I see it as a "limiting case" of my theory of schizotaxic feedback, but partly because I have been (before I dreamed up the theory!) very much impressed with a watered-down form of the phenomenon, a form that we often observe in psychotherapy. It is true, of course, that one can view simple repression as a variant of it (as I gather Freud did, after 1926); and simple repression is not peculiar to schizotypes. A good healthy normal can fail to hear things, and, a fortiori, a well-defended hysteric can. But I have in mind the Bleuler phenomenon of "thought-deprivation," and what Bus Glueck calls "interview driftouts," which I believe are so characteristic of schizotypes as to be one-way pathognomonic (inclusion test). I even suspect that given a long enough therapeutic series, and a good-introspector patient, it can be used as an exclusion test, i.e., every schizoid patient will show it some time in the course of, say, 50 hours. But I'm less confident of its exclusion-test pathognomicity.

It differs from neurotic perceptual defense or repression in its massivity. The patient gets talking about some highly charged material, or you offer a clumsy interpretation, and he just "drifts out," almost like a petit mal episode. The most careful probing by the therapist, or the most honest and practised introspection by a bright, verbal, self-aware patient (who has learned that "thinking of nothing" cannot be used for "thinking of something irrelevant" or "looking at your ugly wall-color") convinces one that the patient's self-report was accurate, namely, his mind went completely blank. Bleuler, who considered it to be pathognomonic, makes the interesting observation that schizophrenic patients ignorant of psychiatric lingo will often reply immediately to the unexplained query "Do you experience thought-deprivation?" [must be higher Flesch-count in the German!] by saying "Oh, yes — is that what it's called? I call it [so-and-so]." And, as you have no doubt observed clinically, schizoid patients will sometimes narrate extra-session episodes of thought-deprivation. (I had one schizoid patient for whom this was among the main presenting complaints. She complained of "not being organized*[1] in the morning, thereby doing a poor job as wife-and-mother. But it turned out to be more than the usual "morning inefficiency" that beta-types like you and my wife consider par for the course. Morning was a role-stress time for her, she felt guilty over being resentful about cooking breakfast, etc., and as a result she would suddenly find herself standing by

the stove, holding a pan or utensil, realizing that she had just "come to" from a drift-out interval. She found these episodes frightening, as indicative that "there must be something terribly wrong with my mind" [alas, how true!]. For a further discussion see Bleuler and my Checklist Manual (pp 33-34, 59-60.)

I realize the tempting danger of focussing on phenomena that fit the theory easily. However, if Bleuler is right in viewing thought-deprivation as pathognomonic (and I believe he is), this counts as another one of the "big facts" and is deserving of special theoretical attention. Given a broadly psychoanalytic model, which in this domain agrees with animal learning concepts, I would view schizoid thought-deprivation as a kind of "overshooting" of Freudian defense [aversive stoppage], where the "turn-off-danger-signal" mechanism goes too far and turns off everything.

146. I realize that Skinnerians like Travis Thompson object strenuously to the intermixing of molar "inhibition" concepts with the strictly neurological process of inhibition. I certainly don't make a flat one-to-one identification of them, which would obviously be absurd. Thus, in order to perform an integrated instrumental act like lever-pressing, both excitatory and inhibitory processes must take place in the CNS. Nothing aversive need be involved in acquiring, shaping, or maintaining such an operant. If I recall correctly, in Milner's revision of the cell-assembly theory, he gives reasons for thinking that certain cortical cells [was it Golgi Type II? I don't have the paper here] have to function as inhibitors within an assembly in order to make it work properly. Such considerations do not bother me, because I do not postulate that all 'CNS inhibitory processes' (neurological meaning) involve 'Olds(-) inhibitory feedback' (molar meaning). I merely postulate that Olds(–) feedback has an inhibitory effect upon ongoing "higher processes." That is, Olds(–) feedback is neurologically inhibitory; but many other CNS functions are also neurologically inhibitory. It's a simple matter of class-inclusion. I do not dispute that Olds(–) activity may exert a facilitative effect on other CNS processes (say, as a very likely candidate, on the arousal system.) From the block-diagram viewpoint, Olds(–) activity should work both ways, namely, stoppage of ineffectual sequences but activation generally to keep the organism "alert" and "trying," until something works.

147. I suspect that, on the instrumental side, the wiring provides something which is analogous to "short-term memory" on the input-perceptual-cognitive side. I am pretty hazy about this, but what I have in mind is that a kind of trigger-assembly controls a related family of motor-assemblies (such as the topographically different ways to press the lever). The trigger-assembly (or, more likely, assembly-system chained into one of Hebb's "phase sequences") can continue to "cook" in a more or less steady state, given a constant exteroceptive input and reinforcement schedule. The problems of sequencing and phasing are complicated, and you have thought about this more than I have. All I mean to suggest along these lines is that, while inhibitory processes must be involved within the motor-system, and between it and the trigger-system (otherwise the animal would louse up its own operants, e.g., by emitting the "lifting-feet" phase before the lever gets down); nevertheless we can distinguish these inhibitory processes from the Olds(–) originated inhibition, which, ceteris paribus, tends to stop the ongoing "cooking" of the trigger-system itself. Crudely put, I am saying that while inhibitory neural discharges are part of what goes on in successfully (motorically) performing an act, this doesn't prevent us from invoking another inhibitory process, with a different CNS origin and a different

block-diagram role, whose teleological function is to inhibit performing the molar "act," at least for the short term.

148. In this connection you recall that Olds has reported that the operant rate under "normal reinforcement" (usual Skinner-box pellet delivery) is boosted by concurrent pulses of intracranial Olds(+) stimulation, even when the latter are not response-contingent; and, by contrast, such an appetitively-maintained rate is reduced by concurrent (but non-contingent) Olds(–) intracranial stimulation. Naturally I like this finding, which I interpret as reflecting the raising and lowering, respectively, of the steady-state "cooking" of the lever-pressing trigger-system. [Reminds me of a maze study MacCorquodale and I published years ago, where we got a nice Blodgett-like pseudo-latent-learning drop by pre-feeding the rats just before the critical run. He called it 'drive-conditioning,' but I would now view it as a mere "souping-up" effect, based upon Qlds(+) activation.] In Honig's new book, Morse (p. 56) says that it is usually difficult to shape "holding" or "inactivity" types of behavior by positive reinforcement because the latter operation tends to "enhance behavior" — a phrase he uses several times in the chapter to mean what I referred to above as a "souping-up" effect, as distinguished from the long-term alteration in strength [= habit growth].

149. If the linkage between two systems S_1 and S_2 is submaximal, or if they are maximally linked but the input system's activation-level is sub-maximal, the effect of hypokrisia should be to drive S_2 more than in a normal brain with identical coactivation history. Some elements of S_2 are overdetermined to fire by confluent inputs, and the hypokrisia makes no difference to them; other elements of S_2 remain subthreshold in spite of the hypokrisia. But those elements which are marginal (near- threshold for a given input pattern) will tend to fire in a hypokrisic brain on occasions when an orthokrisic cell would not. That is, in the terminology of Section 2, the optional transmission probabilities for inputs in the synaptic signal space which would normally be somewhat below $p = 1/2$. To the extent that the controllee system S_2 is well-consolidated, this input effect will be autocatalytically boosted by the hypokrisic intra-S_2 controls, further enhancing the effect. The effect will, of course, be greatest for intermediate values of either linkage or S_1-activation level, i.e., at weak linkages or weak drivings, or asymptotic linkages and strong drivings, the hypokrisia makes less difference. At the low end of the linkage or driving function, most elements are too far below threshold, and the autocatalysis hasn't got a leg to stand on; at the high end, the law of diminishing returns takes over, all the controllee cells being driven at or near their physiological limits.

150. I want to consider first the non-instrumental case, say, a perceptual-cognitive process involving three perceptual CNS systems S^+, S^o, and S^-. (Example: S* is a perceptual system driven exteroceptively by taste of Gerber's Baby Food; S° is a perceptual system driven exteroceptively by sight of mother's pseudo-smile; and S^- is a perceptual system driven exteroceptively by rough, bruising-style grasping of little Schizotaxic Johnny's wrist). We assume that all three systems are already in an advanced state of internal consolidation.

151. [I can make theoretical hay with the fact that, among these three, the "ambiguous social" system S^o is, at any given stage, likely to be less consolidated, but for simplicity I neglect that here. But it's rather important, because the child's perceptual resolution of a "bad-smile/good-smile" ambiguity will involve the extent to which fringe elements in S°,

or, better, elements shared between S^{o}_{bad} and S^{o}_{good}, become interknit with one of these overlapping systems more than with the other. That is, primary aversive drift should operate even in the early stages of assembly-formation, so that what percept-systems become consolidated — apart from their linkage to other systems — is already biased negatively. In other words, I believe that the acculturated schizotaxic literally sees more aversive cues, not merely that he "over-reacts" or "pays more attention" to them.]

152. We assume further that, whether by innate wiring or early and intense conditioning, the perceptual systems S^{+} and S^{-} have developed a strong linkage to an Olds(+) and an Olds(–) center, respectively. By "strong" linkage, I mean near-asymptotic, or at least well past the flex point of the linkage-growth function.

153. Ideally, the analysis of what happens next, given a sequence of quasi-random inputs $S^{+}S°$, $S^{-}S°$, and S° should be done in the most general way possible, which [pace - LaBerge!] would involve studying the differential equations for the feedback systems involved in the manner of general servo-mechanism theory. So I, poor deteriorated non-mathematician, am slowly and painfully learning some of this stuff. [A few years back, I did some preliminary work along these lines, which looked promising but was interrupted by my Mysoline-depression. For example, I find a pencilled auto-memo dated June 1961 in which I proved theorems like the following: "In mediated control, the growth-derivative of the mediated control function is equal to the product of the first link's growth-derivative and its control-derivative, plus the growth-derivative of the second link." How about that? Bet you never knew that before! So I hope to return, to this kind of foolery later, when I know more. Meanwhile, I believe that the step-wise discontinuous treatment that follows is easier to explain, and that it will generalize okay to the continuously changing case. Main point: What is described in the following in terms of discrete, finite phases really takes place continuously and concurrently, but with certain rates and values moving ahead faster than others, etc.]

154. An overall picture of what follows may be conveyed, by a somewhat complex remark, thus: "When the predominant hedonic balance is positive, a slippage-heightened negative feedback suppresses the activation in sub-maximally-driven systems, thereby retarding their further linkage growth; when the predominant hedonic balance is negative, a slippage-heightened positive feedback facilitates the activation in submaxinially driven systems, thereby accelerating their further linkage growth; hence a "neutral," "ambiguous," "inconsistent," "overlapping" system will tend to develop a bias toward the aversive side.

155. Assume that $S^{+}S°$ and $S^{-}S°$ coactivations occur over a quasi-random sequence of occasions. Consider first the positive case (exteroceptive inputs of Gerber's Baby Food taste concurrent with battle-axe mama's pseudo-smile). We represent the brain linkages (as distinguished from the events) with brackets and dots, thus;

$[S^{+} . O^{+}]$ Strength of linkage from perception of Gerber's taste to Olds(+) center activation

$[S^{-} . O^{-}]$ Strength of linkage from perception of rough handling to Olds(–) center activation.

156. After a few S^{+} S° and S^{-} S° coactivations, the linkages and feedbacks look as in the diagram (see next page). We note that $[S^{+} . O^{+}]$ and $[S^{-} . O^{-}]$ are very strong, as assumed in text above. Both $[S° . O^{+}]$ and $[S° . O^{-}]$ are of only intermediate strength,

since the perceptual system S° does not represent a "primary" reinforcer, nor has it been coactivated so often, or so consistently, with intense Olds-center activity.

157. Next phase: What will happen when S^+ and S° are activated concurrently (both being driven by exteroceptive inputs)? By stages, we reason:

a. In a hypokrisic brain, the Olds(–) activity driven by S° will be exaggerated.

b. The Olds(+) activity will not be exaggerated, since it is already being driven near-maximally via the strong [$S^+.O^+$] linkage; or, if exaggerated, its exaggeration will be less than that of O^-, because we are in the region of marked deceleration in the driving-function. [Roughly, if you're experiencing an orgasm or receiving the Pulitzer Prize, the sound of a pleasant tone won't do much for your momentary hedonic level!]

c. From (a), and the coactivation-linkage postulate, it follows that the linkage growth of [$S^+.O^-$] and [$S^o.O^-$] will be faster on these trials than in a normal brain.

d. Hence, even on predominantly positive trials, perceptual systems S^+ and S^o are acquiring more Olds(–) linkage (to drive O^- on subsequent occasions) than they would normally.

e. The heightened Olds(–) inhibitory feedback does not appreciably depress S^+ or S^o activation, nor tend to deconsolidate them, because they are both thoroughly consolidated and are both exteroceptively driven on these trials. I.e., I assume that only the most intense O^- feedback, as in "panic," can inhibit effects of exteroceptive driving of a well-consolidated perceptual system. The organism is wired so as to have only a limited ability to use "denial-type" mechanisms. I am reminded of the fact that successful suggestion of negative hallucinations is scaled at the very deep end of hypnosis scales, most subjects not being capable of it.

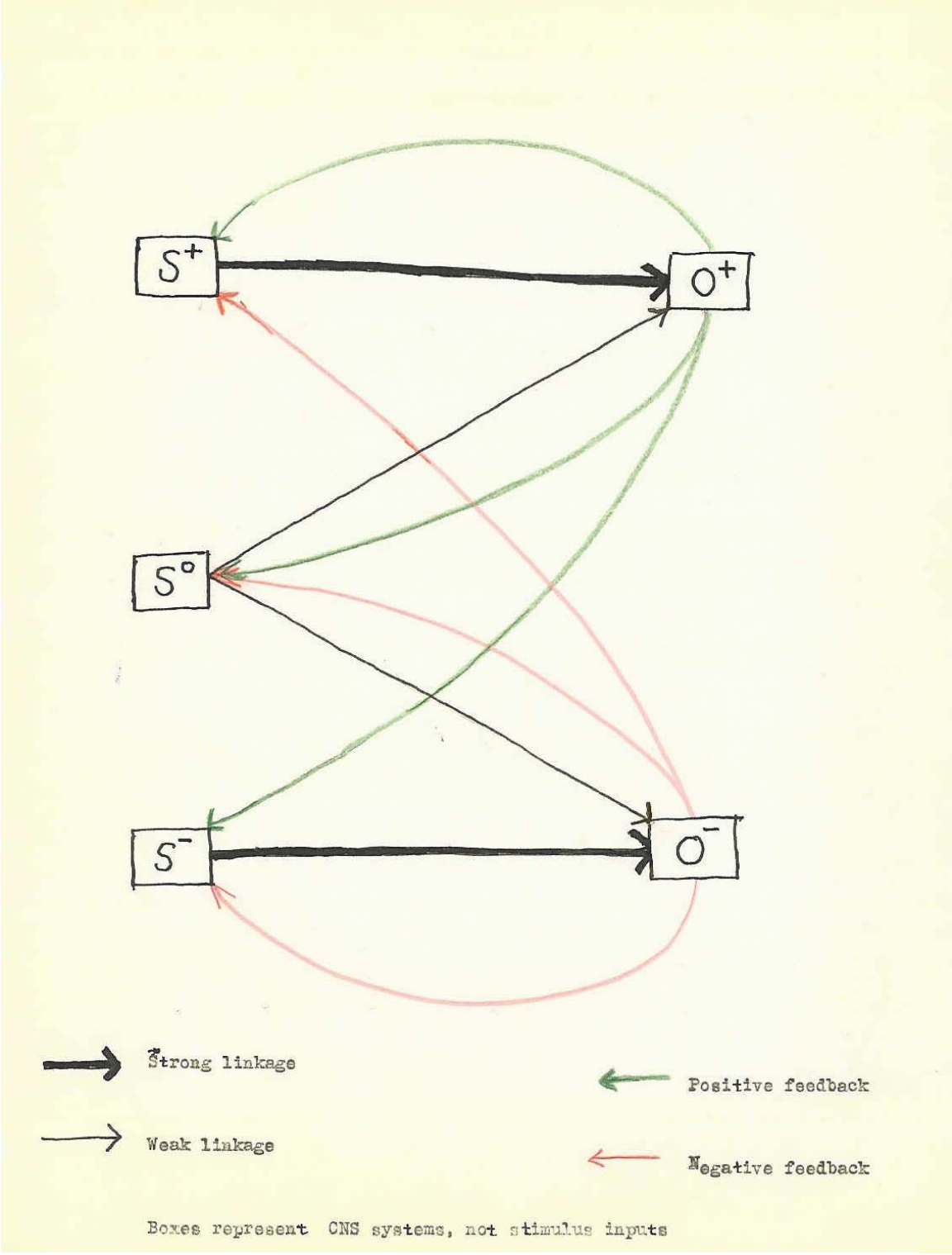
So, after a time, both S^+ and S^o will have accumulated a little more O^- linkage than they should. [You are thinking, "But it works the other direction too!" I know, I'll get to that when we take up the predominantly aversive case. Can't do everything at once, until we do it properly with a system of partial differential equations!]

158. Now let's interlard a few trials of S^+ alone. Then the activation of S^o is via perceptual linkage only, no exteroceptive driving. [My example is not very suitable here, but I tend to agree with Cantril and Ames that actually a very large part of "perception" is perceptual filling — as you were discussing recently in connection with Elizabethan English. After 10-15 minutes of "souping up" the ole rusty Shakespearian cell-assemblies, one literally hears better during the rest of the play. A better example here would be two parts of mother's face, one clearly plus, the other ambiguous.]

On these S^+ trials, we expect:

a. No heightened O^+ feedback, for the same reason as above.

b. Some heightened O^- activity, due to the exaggerated [$S^+ . O^-$] linkage accumulated in the preceding phase.



c. To the extent that "perceptual interknitting" has occurred during earlier phases, so that the linkage [S^+ . S^o] has some strength, we will get some "induced" S^o activation on this basis, in the absence of exteroceptive driving of S^o.

d. This induced S^o-activation will further contribute to the heightening of O^- activity (see preceding phase).

e. The resulting exaggerated O^- activity cannot exert much inhibitory effect on S^+, which is exteroceptively driven.

f. But, since S^+ is normal and O^- exaggerated, the linkage growth of $[S^+ . O^-]$ is exaggerated.

g. More importantly, since S^o is being only moderately or weakly driven by the $[S^+ S^o]$ linkage without exteroceptive support, this S^o activity (unlike the S^+) is quite susceptible to inhibition by the exaggerated O^- feedback. Hence the "perceptual fill-in" or "cognitive association" activation of S^o will be attenuated on these pure S^+ trials.

h. But, again appealing to the coactivation postulate, this reduced S^o activity on the pure S^+ trials means that the further growth of $[S^+.S^o]$ perceptual knitting due to these trials will be retarded.

i. On these trials, the depressed S^o level results in its developing less linkage to O^+ than would occur in a normal brain.

j. The absolute effect upon growth of $[S^o.O^-]$ linkage on these trials is not predictable, since two opposing influences are operative:

(1) The depressed S^o activity, tending to retard linkage growth.

(2) The heightened O^- activity, tending to accelerate linkage growth.

What can be said is that, whether the net effect of (1)-(2) is to slow or speed linkage growth, at least this net effect is favorable to the growth of $[S^o.O^-]$ linkage when compared to that of $[S^o.O^+]$ linkage. Because the O^+ activity is normal, hence any depression in concurrent S^o activity will retard linkage growth with no "compensating factor" to help boost it back up again. Whereas in the $[S^o . O^-]$ linkage, the retarding effect of reduced S^o activation is partially compensated for by the heightened O^- coactivity. As S^o drives O^-, the latter tends to "shut it down," but whatever S^o activity remains is coactivating with a heightened O^- level. So perhaps points (i)-(j) should be combined in one comparative statement,

k. On S^+ trials, the $[S^o . O^-]$ linkage growth is either accelerated or, if retarded, is less retarded than the $[S^o . O^+]$ linkage growth. That is, there is a tendency to aversive drift, in terms of the "neutral" system's linkages to Olds(+) and Olds(–) centers.

159. Meanwhile, what has been happening in the interlarded series of "negative" occasions? Take first the S^-S^o pairings, analogously to Section 157 (you may wish to follow the comparable points, to keep an eye on my bias!) By stages, we reason:

a. In a hypokrisic brain, the Olds(+) activity driven by S^o will

b. be exaggerated.

b. The Olds(–) activity will not be exaggerated, since it is already being driven near-maximally by the strong $[S^-.O^-]$ linkage; or, if exaggerated, its exaggeration will be less than that of O^+, because we are in the region of marked deceleration in the driving-function. [Roughly, if you're having a wisdom tooth extracted or awaiting execution, you are not appreciably distressed by the humidity.]

c. From (a), and the coactivation postulate, it follows that the linkage growth of [S^- .O^+] and [S^o.O^+] will be faster on these trials than in a normal brain.

d. Hence, even on predominantly negative trials, perceptual systems S^- and S^o are acquiring more linkage (to drive O^+ on subsequent occasions) than they would normally.

e. The heightened Olds(+) facilitative feedback does not appreciably enhance S^- or S^o activation, nor tend to hyperconsolidate them, because they are both thoroughly consolidated and are both exteroceptively driven on these trials.

So, after a time, both S^- and S^o will have accumulated a little more O^+ linkage than they should.

160. Now let's interlard a few trials of S^- alone. Then the activation of S^o is via perceptual linkage only, no exteroceptive driving. On these S^- trials, we expect:

a. No heightened O^- feedback, for same reason as above.

b. Some heightened O^+ activity, due to the exaggerated linkage accumulated in the preceding phase.

c. To the extent that "perceptual interknitting" has occurred during earlier phases, so that the linkage [S^- . S^o] has some strength, we will get <u>some</u> "induced" S^o activation on this basis, in the absence of exteroceptive driving of S^o.

d. This induced S^o activation will further contribute to the heightening of O^+ activity (see preceding phase).

e. The resulting exaggerated O^+ activity cannot exert much facilitative effect on S^-, which is exteroceptively driven.

f. But, since S^- is normal and O^+ exaggerated, linkage growth of [S^- . O^+] is exaggerated.

g. More importantly, since S^o is being only moderately or weakly driven by the [S^- . S^o] linkage without exteroceptive support, this S^o activity (unlike the S^-) is quite susceptible to facilitation by the exaggerated O^+ feedback. Hence the "perceptual fill-in" or "cognitive association" activation of S^o will be heightened on these pure S^- trials,

h. But, again appealing to the coactivation postulate, this enhanced S^o activity on the pure S^- trials means that the further growth of [S^-.S^o] perceptual knitting due to these trials will be accelerated.

i. On these trials, the enhanced S^o level results in its developing more linkage to O^- than would occur in a normal brain,

j. The growth of [S^o . O^+] linkage will be exaggerated, because both S^o activity and O^+ activity are exaggerated,

b. However, we note that since O^- is highly active and O^+ only weakly active, the heightened S^o activity will boost linkage-growth of [S^o . O^-] more than it will linkage-growth of [S^o . O^+] on these trials. [Note an asymmetry here with (k) under Section 158].

161. Putting these results together, we find certain pairs of influences working in the same direction, others tending to counterbalance each other. But note the following consistencies:

a. The normally "opposed" linkages grow abnormally, thus, both $[S^+ . O^-]$ and $[S^- . O^+]$ linkages are exaggerated. I see this as one basis of the schizotypic ambivalence, anhedonia, and Rado's "pain-dependency." If we conceive of a statistical distribution of "occasions" in a human life, the abscissa being "net hedonic value," I think that the (non-deteriorated) schizotype's distribution is

   (1) Shifted to the left

   (2) Smaller in standard deviation

   (3) Leptokurtic, with an excess of "agonies" and "ecstasies"

   Properties (1) and (2) flow from my theory; property (3) does not, as far as I can see now.

b. The growth of $[S^o . O^-]$ gets an edge over the growth of $[S^o.O^+]$ whether we deal with the S^+ or S^- trials, although the causes of this are not the same in the two cases.

c. The perceptual knitting of $[S^+.S^o]$ is retarded while that of $[S^-.S^o]$ is exaggerated.

   Now, <u>both (b) and (c) are forms of aversive drift</u>, the former explaining why near-neutral perceptions and cognitions get <u>negatively toned</u>, the latter explaining why the content of perceptions and cognitions is itself biased aversively.

162. We get opposed effects on the growth-rate of $[S . O^+]$ for S^+ trials (where it is retarded) and for S^- trials (where it is exaggerated.) But the more important point is that, in <u>either</u> the S^+ series or the S^- series, this S^o linkage to Olds(+) centers is retarded in growth <u>relative</u> to the "opposite" linkage of S^o to Olds(–) centers.

163. Finally, consider the pure S^o case, after the preceding phases have all run a considerable course. Here we have no exteroceptive driving of a powerful positive or negative perceptual system, so the activations of S^+, S^-, O^+, and O^- are all due to the linkages by which S^o activity can drive each of them. On these "pure S^o trials" (<u>by far the commonest type of occasion in the acculturated organism</u>), we get the following trends:

a. Since $[S^o . S^+]$ is less interknit than $[S^o . S^-]$, S^o activity induces weaker activity in the S^+ system than in the S^- system. [Example: Garmezy's story about the tendency of schizophrenics to perceive ambiguous little line-drawing faces as hostile]

b. On such trials, by the coactivation postulate, the difference in (a) will lead to faster growth of $[S^o . S^-]$ than of $[S^o . S^+]$.

c. The fact that S^+ is less active than S^- means that O^+ is less active than O^-, on these trials.

d. This bias favoring Olds(–) center activation is enhanced further by the fact that the $[S^o.O^-]$ linkage has been growing faster than the $[S^o.O^+]$ linkage has.

e. Since (c)-(d) yield jointly an Olds(–) bias, on these S^o trials the further linkage-growth of $[S^o . O^-]$ is accelerated over that of $[S^o . O^+]$.

164. As you can see, the whole thing has an autocatalytic aspect, whereby matters tend, on the average, to go from bad to worse. Perceptions, thoughts, and feelings are involved in mutual feedback relations, which is, in my opinion, just what we typically see at the molar level in working with schizotypes.

165. I have avoided using a very easy derivation of aversive drift, namely, that based on the idea of a built-in "prepotency" of Olds(–) feedback, i.e., the wiring diagram assures that danger-signals will have priority over appetitive stimuli. There may be something to this, and of course it is not incompatible with the preceding. All we need in that approach is to say that if hypokrisia (a "neutral" defect) boosts Olds(+) and Olds(–) activity equally, the wiring diagram makes a given Olds(–) increment exert more feedback effect than an "equal" Olds(+) increment. [Of course the whole business of "equating increments" is too crude, which is another reason for attempting a more general development.]

166. One global formulation of all the preceding is this: On primarily positive occasions, a slippage-heightened Olds(–) activation tends, ceteris paribus, to suppress "whatever else is going on" hence retarding the linkage growth of the "whatever else" into the positive network; but, in contrast, on predominantly negative trials, a slippage-heightened Olds(+) activation tends to soup up "whatever else is going on," hence accelerating its interknitting into the aversive network.

167. It may be too obvious to mention, but I found myself forgetting it momentarily: The heightened O^+ activity on S^- or S^-S^o trials will not make the trial "pleasant," but merely somewhat "pleasure-tinged," i.e., the net hedonic tone on those occasions remains predominantly negative. Similarly, the S^+ and S^+S^o trials are positive in net hedonic tone, in spite of the heightened O^- activity. "Aversive drift" means "drift in the aversive direction," not "hedonic tone below zero." But on the pure S^o trials, the aversive drift will actually tend to make the net hedonic level negative.

168. I am inclined to lump together anhedonia, ambivalence, and pain-dependent-pleasure as all being one primary concept, namely "hedonic confusion." That is, I think of the synaptic slippage as generating a "mixed," "scrambled," or "conflated" hedonic control system, in which most percepts and cognitions are relatively more "doubly-tinged" than in normals or neurotics. [Perhaps Bleuler's 'ambivalence' is therefore the best word to cover the three concepts?] Clinically, pleasure is often more pain-tinged, pain is often more pleasure-tinged, and — a third possibility — quite a few experiences seem to get sort of "washed out" by the opposition. If Brady's report that Olds(+) and Olds(–) centers can exert a mutually inhibitory effect is true, this figures. And, of course, molar data in animal work have given at least some indication of that for years, not to mention Jones and Watson's famous kid with the bunny-fear, etc.

Now, if we happen to be focussing our clinical attention on the watering-down of what would normally be "strong, unmixed pleasure," we label the effect 'anhedonia'. At the opposite end of the hedonic continuum, where we are usually impressed with the strange pleasure-tinge given to normally unpleasant experiences, we call it 'moral masochism' (Freud), 'pain-dependency' (Rado), and the like. And if we are looking at something in the middle region, in which we expect most normals would perhaps experience some element of mixture or conflict, but not as marked as in the schizotype, then we label it 'ambivalence.' What I am suggesting is that all three are essentially of the same nature, they are all "hedonic scrambling" and/or "hedonic attenuation" due to

the imposition of mixed plus/minus reinforcement regimes on a CNS that has slippage in its hedonic control processes.

169. The preceding "derivation" [ahem!] of aversive drift is with reference to two forms of learning, which I label 'perceptual knitting' [=the growth of linkage strengths between systems S^+, S^-, and S^o] and 'acquired cathexis' [= the growth of linkage between these perceptual systems and the Olds(+) or Olds(–) centers.] I think no important differences for present purposes arise when we consider the development of "expectancies," where the systems being linked are not all perceptual but, more broadly, "perceptual-cognitive." Thus, instead of considering a system S^o as another perceptual unit, we can carry out a similar derivation where the activity of S^o is a Tolman-type expectate [see MacCorquodale-Meehl chapter in Estes et al., Modern Learning Theory], a kind of "inner representation" — not necessarily reportable as a perception or a verbal-tokening event — of an exteroceptive expectandum. Or, in the reverse direction, we have the case where activations of the S^+ or S^- systems are expectates (e.g., CNS representatives of "anticipated food" or "anticipated shock") and they are being activated by the system S^o, a perceptual system driven by an exteroceptive cue.

You will have noted that the whole business would therefore be formulable in traditional reinforcement language, by speaking of S^+ and S^- as "primary positive and negative reinforcers" and S^o as acquiring "secondary [=conditioned] reinforcing properties." I prefer the broader language because it does not commit me to acceptance of the S-R-reinforcement framework; but the processes I postulate do, in many respects, correspond to those molar-level laws. For many purposes, acquisition of the [positive or negative] "reinforcing property" maps into a perceptual-expectancy-cathexis model. But since the complete reduction of the latter to the former is still sub judice (and, as you and I agree, is probably an over-simplification) it seems wiser not to presuppose such a complete S-R reduction in one's theorizing.

170. One reason for focusing attention upon the perceptual-cognitive-cathexis acquisition is that the main trouble with schizotypes seems (to me at least) to arise "prior" or "more deeply" than at the level of instrumental acts. I have no stake in denying the existence of a parametric aberration in instrumental-reinforcement functions, but it strikes me as unparsimonious when we can very easily explain the instrumental aversive drift in terms of an aversive bias in (secondary) stimulus control. Consider: When Ayllon can control the reinforcement schedule using brass tokens that do have strong secondary reinforcing properties, the instrumental-act results are dramatic. And this surely fits one's clinical experience in traditional psychotherapy with schizotypes, because what we observe (or infer) in that context is the relative feebleness of the usual secondary (mainly "interpersonal") rewards as instrumental act controllers, combined with their heightened aversive power. Thus, even when we manage to get the patient to "perform", his social operants, however objectively successful, do not show the resulting increase in strength we find in normal or neurotic persons; and this weakened reinforcing power is correlated with phenomenological reports indicating pleasure-deficit. (See, for example, my Checklist Manual pp. 21-23) Long before the Skinnerians arrived on the scene, the more perceptive and resourceful staff in state hospitals were aware that many chronic, burnt-out schizophrenics could be "shaped up" considerably by reliance upon such incentives as candy, tabacco, money, open-ward privileges, and the like. Thus Bleuler, in his Textbook of Psychiatry, writing of a chronic schizophrene with hypochondriacal

delusions, says that no efforts to treat via reasoning or insight-giving availed, but he goes on to report that "treatment by ignoring or diverting her brought it about.... that she again worked daily and did not ask for treatment" (pp. 430-431). But most pre-Skinnerians lacked (a) zeal and (b) detailed quantitative understanding of reinforcement scheduling principles, both of which the operant-modifiers possess. Then came the psychoanalytic emphasis, with its derogation of behavior as "mere symptoms," and a resulting disuse of even those common-sense techniques of behavior control known to Bleuler (and, for that matter, to my late grandmother, with her 3rd-grade education! It takes a thoroughly brainwashed M.D. or Ph.D. to deny the usefulness of rewarding healthy behavior in psychiatric patients.)

I do not, of course, suggest that an adequate theory of schizophrenia will omit analysis of the complex psychodynamics of defense, diffuse tendency to avoidant behavior, vicious-circle social feedback systems, and so forth. (I remind you that these all have a place in the master-diagram in Section 60.) My position is that we can easily understand, in their essentials, the "big facts" of schizotypic social learning in terms of [learning theory + neo-Freudian psychodynamics + social psychology], provided we can (a) get the process of "unhealthy habit-formation" started on a large enough scale, and can (b) explain why it fails to undergo correction, but rather shows such a pervasive, persistent, recalcitrant bias in the unhealthy direction. I do not myself talk or write much about the received doctrine of schizoid structure and dynamics, but this is because I take so much of it for granted, so why repeat it? It bores me to chew over stuff that has been clear to everyone for over a half-century now; and I am puzzled by the evident relish with which some psychologists stridulate and lucubrate on the obvious. Thus, there is no great mystery (on anybody's learning theory) in how it happens that socially inappropriate behavior by the schizoid patient leads to an objectively aversive shift in the social-reinforcement regime provided by other persons in his environment. In psychotherapy we can often discern the details of such mutual social feedback processes with a minimum of inference. So that once we get our theoretical foot in the door — once we answer the question "How can a mutated gene produce a permanent and pervasive tendency to both cognitive slippage and aversive drift?" — the rest is fairly easy.

Therefore I am concentrating on the explanation of what the diagram calls primary cognitive slippage and primary aversive drift, this being where (given a genetic-neurological etiology) the "psychological mystery" lies. So I conceive of the primary aversive drift as largely (I don't say wholly) a matter of non-instrumental learnings, that is,

a. The overgrowth of aversive-content perceptions, expectancies, and "cognitions" generically, at the expense of their appetitive-content competitors; and

b. The overgrowth of aversive-toned cathexes at the expense of their pleasure-toned competitors, i.e., the schizotype's list of secondary reinforcers is predominantly negative. Thus a neutral-expressioned, unfamiliar human face is, I believe, at least a faint positive reinforcer for normal people; whereas for a schizotype, the human-face percept is, per se, an aversive stimulus. Think of how vast a chain of consequences follow from this one simple difference in stimulus valence!

The development of schizotypal avoidant patterns (withdrawal, safety-measures, testing-operations, life-domain restriction, dereism, intellectualization, renunciation of competition, substitute-gratification, the whole kettle of Meyerian maladaptive habits) seems to me such a straightforward consequence of aversive drift plus cognitive slippage

that I do not spend much time on it, especially since some able minds and great clinicians have worked it out so well already that only the parametric details need to be filled in. However, it is perhaps worth while to raise the question whether, in addition to the influence of primary aversive drift upon perceptions, expectancies, and their cathexes, which then in turn engender a shift of the instrumental act system toward the avoidant side, the hypokrisia might also operate directly to bias instrumental learning aversively? The theory doesn't need this, as I have indicated; but it would be nice if such a consequence were independently derivable. I doubt that it is, without making some additional assumptions. But there is no harm in playing around a little with such ideas.

The main reason why I have little confidence in such speculations is that they involve even more debatable "basic principles" of learning than the preceding. For example, do all expectancies involve stimulus-response bonds, or not? Or is it maybe the other way around? Or neither? (Someone should formulate Skinner-box generalizations in Tolmanese, by the way. I hazard that most of the "rock-bottom generalizations" in Skinnerian thought could be derived from an expectancy model.)

171. Anyway, I assume that when [S^D.R] is increased by the subsequent presentation of a reinforcer S*, this process involves a facilitative Olds(+) feedback to cortical systems $\underline{s}^D$ and $\underline{r}$ which are still active. Armchair argument: The anatomical loci of cortical events $\underline{s}^D$ and $\underline{r}$ being remote, and countless other events s^D_2, s^D_3, . . . s^D_R being all potentially linkable to the event $\underline{r}$ (and to other events r_2, r_3 . . . r_m), we might argue plausibly that unless some coactivation (s^D, r) persists into the time-interval which includes the reinforcing events [S* → s* → O^+ → O^+-feedback-arrival], the post-response reward could not influence the linkage growth. This is the Bill Heron puzzle about "a later event acting backward," which of course as he put it was incorrectly formulated, since the effect of reward is not "backward," but upon subsequent occasions. Yet Heron was pointing to something important here. If neural linkages grow by coactivations, there <u>is</u> a puzzle about how a post-coactivation event can influence the growth. The more you contemplate this, the spookier it seems. But if we postulate that some coactivation persists after an instrumental response is made, so that concurrent events of the type (s^D, r, s*, O^+) can take place, the puzzle vanishes, although many difficult parametric problems remain.

I suggest that this armchair argument is bolstered by a "big fact," not about schizophrenia, namely the fact of retrograde amnesia following suspension of normal CNS function by concussion, EST, grand mal seizures, etc. I realize that the two <u>Bulletin</u> review articles are inconclusive as regards the notion of post-response reverberatory consolidation, but that doesn't bother me at all. Because

a. Most of the data on rats are at least <u>consistent</u> with the consolidation theory;

b. As critical tests of the theory, the designs are mostly faulty or inconclusive, as both reviewers point out.

The experimental material is actually more complicated to interpret than the "big fact" itself, in my opinion. So far as I know, the phenomenon of retrograde amnesia following massive, sudden, immediate suspension of "normal" CNS activity is as solid as the law that mammals die without air. Any neurologist who could find a single case, well-documented, of an exception to this rule would become famous (if the profession believed him, which they wouldn't). I find it strange — but, like the "significance test"

business, perhaps another example of psychologists being pseudo-sophisticated and hence actually naive — that a bunch of crummy experiments, mostly capable of two or three different interpretations, almost all involving unsolved parametric problems, and very likely not all even dealing with the phenomenon of interest, are taken to be more valuable than the clinical "big fact." The reviewers (especially the recent one) seem to have the notion that if you examine several studies which are ambiguous (e.g., results could be due to factor A, or B, or C), this somehow weakens the clinical generalization you started out with! It's as if we cooked up some feeble experiments to find out whether schizophrenics are shy; the experiments collectively turn out to be inconclusive, on several counts; whereupon we conclude "Considering all the evidence, we must say that schizophrenic shyness is a doubtful concept." Or a social psychologist does some monkey-business with kindergarten children and play money, concluding that "Controlled research fails to substantiate the common claim that human beings work for money incentives." Baloney, I say.

Of course we cannot dogmatize about <u>how</u> it works. All I insist upon is that the "big fact" is as solid a generalization as many accepted "laws of scientific [= experimental] psychology." Since this undisputed "big fact" of clinical neurology fits so well with the armchair requirement of coactivation, I reason that Olds(+) feedback due to S* presentation exerts a facilitative effect upon post-response reverberatory activity in cortical systems $\underline{s}^D$, and <u>r</u>, whereby their linkage is increased. Look at it this way: Two anatomically remote systems S^D_1 and S^D_2 are both wired to system R (which they <u>must</u> be, given that S^D_1 and S^D_2 are each potentially linkable by conditioning to R). If the molar sequence $S^D_1 \rightarrow R \rightarrow S^*$ increases the strength $[S^D.R]$ at the expense of $[S^D_2.R]$ by virtue of the fact that $S^D_2 \rightarrow R \rightarrow S^*$, how can this take place if, when the Olds(+) feedback due to S* arrives at system S^D_1, there is no persisting process in R which is different from what would have been present if R had been coactive with system S_2 instead? I don't insist that S_1 inputs to R must still be arriving (although that seems plausible to me); but, if not, it seems that the neural elements in R must be currently activated in a pattern different from what would be present had S^D_2 been the stimulus, otherwise a differential effect on linkage growth is hard to explain.

So I assume that a major contributor (perhaps the necessary and sufficient one, but that is more than I need for present purposes) to linkage growth is the facilitation of post-response reverberatory activity in R, or in both S^D_1 and R, by the Olds(+) feedback which subsequent presentation of S* elicits. The <u>differential, growth advantage</u> of $[S^D_1.R]$ over $[S^D_1.\ \bar{R}\ ]$, or of $[S^D_1.R]$ over $[S^D_2.R]$, I take to be a consequence of this enhanced reverberatory activity, which leads to accumulation of more Hebbian increments in synaptic control "on the average." I do not maintain that Olds(+) feedback is the only type of facilitation. Other types occur also, notably the "non-drive-related" pure stimulus reinforcement (see Kish chapter in Honig). I view most of this group as due to stimulus-induced blips of heightened RAS activity, the corticopetal RAS barrage being facilitative and hence prolonging or heightening reverberatory coactivations. I think the Kish chapter supports this interpretation, although he himself doesn't make it.

172. What will be the effect of hypokrisia on the parameters of the positive-reinforcement situation? With anatomically remote systems, we are operating in the low region of the linkage function, where it is either linear or positively accelerated. (We can safely assume this because we know from molar data that, in general, pure "law of

Exercise" learning is very weak, for anatomically remote systems.) Might it not be the case that, due to hypokrisia, the reverberatory activity is already heightened somewhat, so that the facilitative effect of Olds(+) feedback is attenuated? That is, the Olds(+) feedback "normally" puts some near-threshold elements over threshold in the post-response phase, enhancing somewhat the "activation level" of the system (s,r); but if the hypokrisia has already enhanced it, the increment yielded by Olds(+) facilitation will be less.

[I don't want to get into the mind-body problem, but we might speculate that the "hedonic tone" of an activity or thought is a relative matter, involving a difference between the cortical state with and without Olds(+) facilitation. So that perhaps the "pleasurableness" of one act or event over another is, so to speak, somewhat "washed out" in a hypokrisic brain by a quantitative reduction in the difference between cortical activations with and without Olds(+) facilitation.]

Of course this does not mean an absolute reduction in reinforcing effect. However, as Estes and others have argued quite convincingly, when the experimenter focuses on a particular response of interest to him, he is nevertheless dealing actually with a batch of competing responses, most of which he need not even identify except by negation — these other responses are $\overline{R}$, and, if made, they lead to $\overline{S^*}$.

[Might it be that the stereotypy seen in chronic, deteriorated schizophrenics is partly attributable to such a heightened linkage-growth lacking specific reinforcements? Sometimes we can cook up a plausible psychodynamics for these, but it's not always easy to do without stretching things. Bleuler and others have described a kind of slow "drifting" of such stereotypy in dilapidated cases, where it almost seems that pure happenstance can lead to a meaningless, mechanical ritual becoming habitual for long periods. But I don't want to press the point, because I admit the many possibilities for subtle reinforcements being operative.]

173. Now consider the aversive case. Here I assume that termination of an aversive stimulus S^- in the sequence $S^1S^- \rightarrow R_2 \rightarrow \overline{S^-}$ functions by virtue of a sudden "lifting" of the Olds(–) inhibitory feedback, permitting a heightened reverberatory activity of $\underline{r}$ (or of s_1 and r). When an "ineffectual" response is made, the Olds(–) feedback persists, and tends to "hold down" reverberation and hence retard linkage growth. We may assume also that some kind of counterbalancing occurs between the arousal component of aversive stimulation (mediated by the ascending RAS) and the inhibitory component (mediated by Olds(–) feedback). If we accept the idea advanced by Bullock and others that a good deal of synaptic input, especially to dendrites as contrasted with specific input to the axon hillock, is insufficient to spike but merely "soups up" the neuron so that it spikes more readily if critical impulses arrive at certain privileged loci (such as axon hillock), it is plausible to suppose that many neural elements in assemblies undergoing Olds(–) inhibition are, so to speak, "latently activated" — hyper-irritable due to RAS input but prevented from spiking by the Olds(–) inhibition. When a response "succeeds" in terminating the aversive state, we might expect that the hypokrisia would now lead to an exaggerated post-response reverberation, and hence a linkage-strengthening effect more pronounced than in the normal brain. So here again the hypokrisia, in itself a "neutral" fact about synaptic control parameters, could result in an asymmetry between the appetitive and aversive linkage growths. And again, as in the non-instrumental learning of perceptions and cathexes, what generates this asymmetry, given the "neutral"

fact of synaptic slippage, is the business of algebraic sign — the fact that positive reinforcement strengthens operants by onset of facilitation, whereas negative reinforcers strengthen operants by offset of inhibition.

174. I realize that neither of these biases yields an aversive drift, taken singly. I have only argued that hypokrisia will retard the differential growth of positively reinforced operants over their competitors, with both somewhat "over-growing"; whereas hypokrisia will not retard, and may even facilitate, the differential growth of operants reinforced by aversive offset over their competitors, both overgrowing. Broadly speaking, "Schizotaxics should be less positively conditionable and more aversively conditionable." But this prediction holds for operants only, and assumes that we are dealing with experimental situations where we know just what is appetitive and what is aversive. For the developed case, this is a difficult thing to know, as Garmezy and others have emphasized. However, I believe it fair to say that, on the whole, the trend of the evidence does support this molar prediction. Even if true, it is too easily derivable from alternative theories to provide any support for mine.

We know that most interpersonal, vocational, educational, etc., tasks of life involve the simultaneous presence of neutral, positive, and aversive cues and outcomes. E.g., if I study instead of loafing, I may pass or fail; if I try to converse with a stranger, he may smile or look bored. In a kind of "average" sense, although many situations will provide a clear preponderance of cues favoring an appetitive operant R_1 or an avoidant operant R_2, many "borderline" stimulus-situations do occur, of the type $S_1.S_2^-.S^D$, and in such cases the fact that $[S_1.S^D.R_1]$ growth (by reward) has been retarded in relation to $[S_1.S^D.R_2]$ whereas the topographically overlapping $[S_1.S^-.R_2]$ growth (by aversive offset) has been enhanced in relation to $[S_1.S^-.R_1]$, gives the avoidant response R_2 a "statistical edge" over the class of such mixed-input occasions. And of course this process tends to become autocatalytic, since when R_2 draws ahead, it means that R_1 tends not to occur and therefore it in turn gets behind in the total number of "objective" positive reinforcements given to it.

175. Needless to say, the aversive drift of operant control then results in an altered environmental-probability, because other people learn to respond to the patient on the basis of his aggressive/avoidant dispositions rather than his (rarer) appetitive efforts. In psychotherapy these patients often complain that their tentative adient social moves seem not to "work," and I don't believe this is always a distorted perception, since the patient's providing of a slightly altered reinforcement schedule to his wife or employer cannot be expected to have an immediate effect on these other persons; and his new "positive" behaviors are still too weak to be maintained for any extended period lacking obvious immediate social payoff.

176. As mentioned above, I categorically repudiate the Utopian aim of explaining all clinical and research findings on schizophrenia, a demand I wouldn't impose on my theory or on anyone else's. It seems fair to say that most "findings" in, say, Bellak's book can safely be left on the shelf for the foreseeable future, either because

a. They arise in defective research contexts (e.g., studies using college students as controls when the indicator is known, or plausibly supposed, to reflect nuisance variables like intelligence, social class, test-wiseness, docility, age, cooperation, etc.)

b. Studies unsuccessfully replicated, even when we cannot pin-point the source of error in the original study.

c. Studies showing mere "trends" which, while statistically significant, exhibit such huge overlap that the finding can hardly be of theoretical significance from the standpoint of etiological inference (or of practical use in clinical diagnosis, or as an indicator sufficiently valid for genetic research).

It seems plausible to postulate that some of these "faint-trends-with-overlap" are attributable to generic nuisance variables common to many psychiatric patients, including neurotics and fairly "normal" people reacting to chronic stress, unhealthy living habits, hospitalization effects, and so forth. Just how these nuisance variables operate, and why they show an average trend to differentiate schizophrenics, is a legitimate research interest; but surely it is not optimal strategy to fix our attention upon them in the present state of knowledge. I would agree that some of these may actually reflect the primary etiology; but the number and phenotypic diversity is so vast that one would have to possess unusual luck as a researcher in order to select the right ones out of the heterogeneous mass. Example: The Worcester group (as summarized by Hoskins in Biology of Schizophrenia) found something fishy about thyroid function. I have subsequently seen some later replications of this. What can one do with such a "trend" fact as this? Most solid-gold-diagnosed schizophrenics are normal in thyroid function, even granting the finding to be adequately corroborated. So what use is the trend? It is easy to invent an ad hoc theory, but even if true, it leads nowhere. I would think of three plausible "explanations." First, the endocrine system is a delicately balanced homeostatic affair involving complex mutual feedbacks of both biochemical and CNS control linkages. Almost any "neural integrative deficit," whether Meehl's hypokrisia or some other, could lead to a malfunction in the feedback controls. As Paul Hoch says, if you scrutinize almost any complex biological control system in schizophrenics carefully, they seem a bit haywire. Which system will be phenotypically deviant in a particular case presumably depends upon other genes and long-continued life habits, varying widely from case to case. Secondly, even if no direct influence of a neural integrative defect were involved, there are many psychosomatically-mediated possibilities for such an effect (chronic anxiety, long-continued de-arousal, Selye's "stress" factor, dietary peculiarities of the kind Kety has teased out, "sexual stasis" a la Reich, activity-inactivity cycles, and so forth). Thirdly, we already know that there are individual differences among non-schizotypes in thyroid function, whatever their origin. Given the assumption that non-specific factors (such as energy-level and irritability) function psychologically as statistical potentiators of schizotypic decompensation, such a non-specific thyroid aberration could serve as one factor determining whether a particular subgroup of schizotypes decompensates, and when these cases appear in a general sample of schizophrenics, they will produce such a statistical trend. The thyroid deficiency in such cases is not genetically linked, or a pleiotropic effect of the schizogene, but plays the same role as would inherited ugliness or low IQ.

176a. I do accept the theoretical burden of explaining most (not all, even here!) of the "big facts" of clinical symptomatology. But, as stated above, here I take over much of what is currently accepted in the psychodynamics of schizophrenia by non-genetic clinicians. Take the accessory-symptom pattern we call 'catatonia.' The biggest fact about catatonic symptoms is that their incidence is tremendously influenced by the

clinical handling and the hospital milieu. It seems pretty well established that catatonic attacks in a hospital population reflect the adequacy of psychiatric care, and perhaps (as in hypnosis) the social expectations set up by staff (and by observing other patients?). I view the catatonic episode as a particular effort of defense, the detailed psychology of which was brilliantly analyzed by Bleuler in his little-read monograph The Theory of Schizophrenic Negativism. Since reading that book, I have held a largely "psychological" view of catatonia, and the superficial analogies to neurological phenomena or quasi-catatonic animal behavior induced by bulbocapnine or "animal hypnosis" as misleading. Thus my theory of catatonia is compatible with Bleuler's, because given a prior derivation of ambivalence and interpersonal aversiveness, the necessary psychological states which Bleuler presupposes in his derivation are available to me also. Of course I am open to the possibility that a non-schizotype might be unable to cook up a catatonic phenomenon like waxy flexibility on the same psychodynamic basis. It could well be that, whereas the motivation to defend against interpersonal intrusion by catatonic molar behavior will determine the behavior content (e.g., mutism versus echolalia, or negativism versus echopraxia), it still requires something special in CNS linkage parameters to produce a dramatic phenomenon like waxy-flexibility. Without fruitless speculating, one can at least hazard the guess that just as striped-muscle hysteria requires a "somatic compliance" in addition to the learned defense-system (Freud), so the schizotaxic brain is capable of kinds of dissociation of sub-systems that the normal brain can rarely or never achieve. If this were so, we might ultimately learn that hypokrisia plays a critical role in making catatonic defenses available, but that which schizophrenics become catatonic is determined partly by "accidents" of social learning and partly by non-specific polygenic parameters of CNS function. I think on balance that the learned interpersonal factors are likely to be more important here. You may recall that in their ecological study of schizophrenia, sociologists Faris and Dunham showed that catatonic schizophrenia is concentrated in the "immigrant" precincts of the city, whereas the paranoid form is in the "one-room loner" section (I cite from memory). The latter could be selective migration, but the former seems more plausibly explained by their social-interaction theory.

177. I have not said much about those florid kinds of cognitive slippage Bleuler calls "accessory symptoms" (delusions and hallucinations). Here again, it seems parsimonious to postulate that primary cognitive slippage (flowing directly from hypokrisia) provides the neurological substrate which renders such molar distortions relatively more available to schizotypes. Thus, by analogy with the stabilized retinal image prediction, I assume that the convergence of "multiple linkages to the assemblies involved in "hearing a voice" can produce an intensity of perception more than normally possible. (Phenomenological studies, I think by Carney Landis, suggest that many schizophrenics do "hear" the hallucinated voice as sounding different from a real voice. Early cases may even say it is "an inner voice, like my conscience," and such like.) I suspect that the reason why auditory hallucinations occur so much more often than visual is that the "inner voice" is closer, quantitatively, to processes found in normal "thinking" and "inner speech." So it requires only a little hypokrisic exaggeration to hallucinate a voice, but a lot more to hallucinate a vision. Clinical support for this view is the fact that vivid, sustained, patterned visual "scenes" are found mostly in hebephrenic patients — the subgroup who show the most massive thought-disorder generally, and whose psychometric level on standard intelligence tests is often in the feeble-minded range.

They have a very malignant prognosis, and are typically very "scattered," "dull," "confused" even when affective factors are (apparently) minimally operative. My guess is that in hebephrenia we have the hypokrisia potentiated by non-specific polygenic variables of CNS function, variables which we also have evidence for saying exist as individual differences in the non-schizoid population, of the kind that lead to "normal" differences in cognitive style and intellectual ability (e.g., "sharpeners" versus "levelers" in Bruner; "attenders" versus "high-distractibles"; the obsessional brain versus the hysterical; and whatever else is involved in producing the huge individual differences we find in "clarity" versus "fuzziness" of thought, or even perception).

But I do not mean to over-neurologize matters. In my master-diagram the primary cognitive slippage interacts with social learning to yield a secondary cognitive slippage, and here I willingly rely on the received psychodynamics of schizophrenia. I do not deny — rather I insist — that the weakened social cathexes of the schizotype reduce his "animal faith" in consensual validation, as well as his motivation (and communicative skills!) for checking his ideation against what others believe. It may be that the main factor in constructing and maintaining a paranoid delusion is the abnormal social communication, with the primary slippage playing a very minor role. I incline to think that the hypokrisia plays a larger part in generating hallucinations than it does in generating delusions. I think this partly on "logical" grounds, in the sense that hallucinations require a brain that can cook up false <u>data</u> (perceptions), whereas delusions involve subtle errors in applying inductive logic. Inductive logic is not a "formal" affair, since, while deductive inference occurs in it, the formal inference is from theory <u>to</u> data, whereas the "backward" inference from data (perceptions) to a paranoid theory is a matter of methodology or epistemic strategy. That is, in criticizing an inductive inference, we rely upon meta-considerations such as prior-probability, the adequacy of one's deliberate searching for critical refutation-opportunities, readiness to concoct or examine available alternative theories, and so forth. These "rules" are not formalized, as the Bayesians and "personalistic-probability" statisticians have recently emphasized. So that the importance of social orientation to oneself and the interpersonal community is much greater in matters of inductive inference. Example: A paranoid patient thinks that her boss is slandering her to make her quit the job without firing her. I can't really <u>refute</u> such a "theory" — after all, such things have been done by cowardly administrators. My low prior probabilities for such theories are based on ticklish questions like, "What are the base-rates for this event," and "why would this particular boss want to do this?" But the patient's prior probability is based upon her interpersonal aversiveness, whereby she feels (hence, <u>thinks</u>) that many people do such things, and that this particular boss has malicious intent toward her. Being a schizoid non-communicator, she then fails to consensually validate her theory by checking with the peer group, and so it goes.

Nevertheless, I still believe that the primary cognitive slippage at least helps to maintain such screwed-up ideation. When you force such patients to detail their inferential processes, you detect many subtle slippages in the transitions, such as verbal equivocations that contribute to semantic fuzziness, and which are probably made easier — given the motivation to distort — if the convergent linkage-relations are out of kilter. It's like someone playing solitaire with a badly smudged deck — he wants to win, which determines the <u>direction</u> of his errors; but the possibility of certain mistakes is partly contributed by the smudging itself. Of course it is wrong to conceive this conceptual

slippage as wholly verbal, but our ignorance of the nature of non-verbal [yet "abstract"] concepts makes it easier to use verbal slippage as the paradigm case.

178. In the schizoid speech domain, a Bleulerian "big fact" which he expends many pages exemplifying because it is a "specific kind of alteration of thinking . . . that occurs nowhere else" (Textbook of Psychiatry, p. 373), I cannot buy the pure social-communication theory as an adequate explanation, nor could Bleuler. In part, yes: There is a normal "set-to-communicate" which is no doubt often impaired by the schizoid interpersonal foul-up. But letters to the superintendent (or lawyer, or kindred) urgently pleading for release or for certain privileges, and emphasizing one's sanity, must be presumed to involve a set-to-communicate, unless we have strong counter-evidence. Thousands of such earnest, deeply-involved missives have been produced by schizophrenics, and they typically contain the unique Bleulerian associative loosening. Besides, most of us don't manifest schizoid slippage even in non-communicative writing (e.g., diaries, reminders, problem-solving ideation, spontaneous literary creation) — but the schizophrenic does it there too. I think it is unparsimonious, reflecting the usual "dynamic" prejudice of American psychiatry, to dismiss the very common presenting complaint of incipient (pre-psychotic) schizophrenics that "I can't concentrate," "My mind won't work," "The thoughts are confused," or, as one of the Piotrowski-Lewis patients put it with pitiful insight, "I can't think, this is serious because it never happened before." [It is worth noting that in their follow-up study of verifiably misdiagnosed cases, the chart-sign: "Spontaneous complaint of inability to concentrate, unattended by evidence of interfering emotional pressure," appeared in 10/24 of schizophrenic patients originally misdiagnosed psychoneurotic, but in only 1/28 of neurotics who were correctly diagnosed.] If a concerned, intelligent, introspective patient tells me that one of his main complaints is that his thoughts are vague, mixed-up, or "go their own way," when he is trying to add up a customer's bill, I believe him, since I have no cogent reason not to!

My view of the schizoid associative loosening is, I think, quite close to Bleuler's. He postulated an associative loosening of unknown etiology as part of the primary disease process — the most "core" part, hence his naming of the disease. But he concluded that the content of the aberrated associations, and (often, maybe always?) the occasions of their irruption was "katathymic" [= affectively determined]. Hence schizophrenic thought-disorder is like the dream (where the cognitive ego-functions are impaired by sleep) or the delirium (impairment by toxins or pyrexia). That we think crazily and perceive hallucinatorily is due to "organic" Factor X, but what we think and perceive is "complex-selected."

My conception of katathymic association-disturbances therefore involves both primary ("neurological") and secondary ("socio-psychological") cognitive slippage, as seen in the developed schizotaxic [= schizotype]. Synaptic slippage [= hypokrisia] — in itself neither "cognitive" or "affective," being a micro-concept to which such molar categories are inapplicable — underlies the primary cognitive slippage in a very direct fashion, which almost any reasonable parametric assumptions would presumably show. Hypokrisia also leads, given the (universally) mixed social-reinforcement schedule, to aversive drift, anhedonia, and ambivalence. On any theory of learning, motives and affects must act selectively upon available habits, including "cognitive" linkages. We have an associative network which is statistically aberrated (in its multifarious intercon-

nections) because of primary cognitive slippage; we then potentiate this by differential activation from aberrated affective-motivational selectors. Result: Katathymic thinking.

I admit the above is pretty general, so let me spell out an example. There is plenty of experimental evidence that "families" of verbal-conceptual dispositions become linked together so that we can "soup up" a formal or thematic group in a variety of ways. Whether such a generic souping-up involves a kind of super-ordinate "monitor" assembly, or merely multiple inter-assembly control linkages, we need not decide for present purposes. I conceive this souping-up as either

(a) Maintenance of a low but non-zero level of activation, i.e., the assembly is actually discharging but not at a level (number and frequency of its elements spiking) sufficient to gain control of an instrumental output channel; or

(b) The elements are not firing (or, better, not firing more than we assume needed to get non-specific activity of EEG-type) but they are individually being souped-up by sub-spike inputs to the dendritic tree; or

(c) The related assemblies are all fully activated, but a super-ordinate selector-system determines which of them controls the instrumental output. (This could be inhibitory, but it doesn't have to be.)

Now we know that ordinary verbal processes involve very complicated multiple connections, which (if Jenkins is right, and I assume he is) need not each be reinforced, but are "passively" acquired by the child via the huge mass of auditory input he receives — rather like the building up of visual pattern perception in Hebb's theory. An element in an assembly comes under partial control of elements converging from many other assemblies through the accumulation of Hebbian increments at the synaptic locus on coactivation occasions. Ditto one level up, via this single-element control linkage, an assembly comes under partial control of many other assemblies. Ditto two levels up, an assembly-system (representing, say, a word or phoneme) comes under partial control of converging inputs from related assembly-systems. The "selection" of a word to occupy a particular position in a sentence must involve, among other things, the activation of the neurons-in-assemblies-in-assembly-systems for that word. We can conceive that the members of a related word-family have various momentary probabilities due to the momentary confluent drivings from previously tokened words + motivational-affective selectors and so on. All this is normal functioning.

The hypokrisic brain will function similarly, but with an important parametric difference. Elements will fire from time to time on the basis of inputs from other assemblies which would not suffice to get the orthotaxic cell over threshold. Hence there will be, over the long haul, a slow accumulation of Hebbian increments from other assemblies which are more "remote" in the molar sense (i.e., correspond to verbal operants less closely associated by the verbal community). We need not assume that the schizotaxic child actually emits more "far-out" verbalizations, at least to begin with.

Now take the adult schizotype who is decompensating. Most of the stream of speech is normal. But from time to time, the momentary social input + previous verbalizations + sub-instrumental assembly-activations, all taken together, "happen" to involve activation of convergent assemblies each of whose long-accumulated Hebbian increments are considerably exaggerated over the normal. Singly, they might not suffice to "fire" the controllee assembly (or, on another view of souping-up, they might not activate it at a

level which is effectively competitive for the instrumental output channel). So on such a "confluent" occasion, a word with low-probability in the orthokrisic brain gets "in the running" with more normal associations. (Why don't the latter also increase, due to hypokrisia? They do, but they are in the linear or decelerated region of the control function. That's part of what makes them "normal," of course.)

We need, not assume that this (almost "accidental") convergence of exaggerated control sources suffices to displace the "normal" tokenings by "aberrant" ones. All we require is that, from time to time, such a process puts an aberrant tokening "in the running," giving it an associative strength close enough to that of the "normal" tokening so that any selective potentiator favoring the aberrant tokening will push it momentarily ahead. And of course we often have such potentiators operating, namely Bleuler's "affective complexes." It seems to me that this is a very plausible explanation of katathymic speech (and, by extrapolation, katathymic thought).

[You may feel I over-use the deceleration or ogival concept, and I hope I don't. In the present case, I suspect that one could do without it. Consider an assembly-system S_1 which would effectively control the "normal" tokening-system S_n because the associatively "remote" competitors S_2, S_3, . . . S_k, do not, even in the aggregate (when simultaneously activated at levels x_2, x_3, . . . x_r) bring an aberrant tokening-system S_a to a level intense enough to pre-empt the output channel. Now assume a fixed increment Δx_i in control linkage due to previous hyper-accumulated Hebbian increments. The "normal" controller exerts increased driving power $(x_1 + \Delta x_1)$, putting the activity in S_n ahead of where it would normally be. But the remote controllers exert net influences $k(x_i + \Delta x_i)$ upon aberrant system S_a, which is therefore hyper-activated relatively more than S_n is. Even if we assume some "pattern shrinkage" (as in Hull's use of Perkins Equation), this would have to be very great to wash out the net advantage given by such convergencies.]

179. The psychoanalytic theory of schizophrenia emphasizes the "withdrawal of cathexis from objects," and while this is really a theoretical notion rather than a big fact, I should be able to say something about it, since I believe some such process to occur. I am not prepared to offer more than a sketch of how this molar construct fits into my theory, but it would run along the following lines. In experimental studies of reinforcement, we select some instrumental act for study, or we differentially strengthen the secondary reinforcing power of a particular stimulus. I think this orientation, arising from the scientist's procedure of gaining experimental control of selected aspects of a situation, leads us to neglect the fact that many concurrent inputs which we do not manipulate, which provide mere constant (or randomly fluctuating) "background" stimulation, also possess reinforcing power. But these stimulus properties are not being manipulated; they are not being discontinuously varied by onset-offset operations, nor are their several influences upon response strengths being examined. [Example: A constant input such as a faint odor of wintergreen or a "pleasant room" has been shown to influence problem-solving or rote-learning, even though no differential cue-value can operate here.] I take it for granted that almost all objects, including persons, acquire some linkage control over Olds(+) and Olds(–) centers, so that the acculturated organism tends to maintain a kind of "average" or "steady-state" hedonic tone (also of arousal) supported by such ordinary exteroceptive inputs. And I believe that part of schizotypic misery is that this average hedonic tone is chronically shifted toward the aversive side of the continuum, due to aversive drift. So it isn't always a matter of offset/onset of specific or intense rewards,

threats, punishments, etc. The continuous (or repetitively-scanned) sights and sounds of daily life, the objects of furniture in a room, the forms of other persons, etc., are continuously driving many Olds centers at low-to-moderate levels even when "nothing special is happening," and when these inputs are not functioning as discriminative stimuli to control specific operants, nor as reinforcers with important onset/offset contingencies.

Now I take Freud's "investment of libido" and "cathexis of energy" to involve, among other things, the linking of perceptual-cognitive assemblies [the neural bases of inner object-representations] to hedonic centers. "I positively cathect an object" means, neuro-physiologically, that the cortical system which embodies my previous perceptions and expectancies of the object is intimately linked to certain Olds(+) centers. Phenomenologically, we say "It is pleasurable to see what we love." Now the aversive drift will tend, on the average, to retard growth of such linkages. Furthermore, the secondary development of phobic-like avoidance patterns, with the corresponding preference for autistic thinking, shifts the objective schedule so that the primary reinforcements are infrequently paired with the secondary ones. Eating alone rather than with others, attending to fantasy instead of social input, autoerotic rather than alloerotic sexual pleasure, minimize the kind of reconditionings that normally refurbish the common secondary reinforcers. I also assume a law of disuse, which is <u>not</u> refuted by short-term experiments on nonsense syllables. Skinner's pigeons retained the lever-pressing operant after several years, which is the fact he chose to emphasize, and rightly. But the size of the extinction curves was reduced. I think there are several distinct kinds of "forgetting," and that <u>one</u> kind is sheer disuse. If an assembly-linkage is rarely activated over many years, the ordinary metabolic processes and micro-displacements will result in "engram-attrition." I think this may be one of the long-term factors leading to the terminal dementia of burnt-out schizophrenics, some of whom seem, as you know, almost "organic" in their cognitive functions.

So I theorize that the primary aversive drift, abetted by the secondary avoidant patterns, results in a reduction of the linkages between Olds(+) centers and perceptual-cognitive systems representing external objects. Suppose that Freud is right (and I'll bet he is) in theorizing that the <u>normal</u> state of early infancy is "hallucinatory wish-fulfillment," e.g., the primary-process perception of mother's breast when hunger is aroused. My theory is then consonant with another aspect of psychoanalytic theory of schizophrenia, to wit, that it consists of a regression to infantile, primary-process modes of gratification — a regression more profound and widespread than occurs in neurotics. As you know, I do not admit that <u>all</u> of the accessory-symptoms of schizophrenia are "wish-fulfilling." Phenotypically, they don't always seem to be; and I see no cogent theoretical necessity to insist that they nevertheless really <u>are</u>, if we could completely fathom their thematic content. But one must admit that <u>many</u> of them are, and in those instances I suspect Freud is substantially correct. Those perceptual-cognitive activations which arise endogenously will tend to be freer of aversive components than those arising exteroceptively, because the "mixed" external regime yields more aversive drift, given the hypokrisic bias. Example: The schizoid preference for masturbation is pretty clearly due to the fact (also true qualitatively for normals and neurotics) that the content of a masturbation-fantasy is completely specifiable by the patient, whereas the social-sexual behavior of a real sexual partner is not, and will often include numerous aversive components.

180. The clinical phenomena of "derealization," so pathognomonic as to constitute another big fact, fit easily into this interpretation. Incipient schizophrenia very often includes spontaneous complaints that objects and persons do not "seem real," the exact phenomenal quality being hard to empathize by non-schizoid therapists. I take it that at least part of the "feeling of reality," which is on the borderline between cognitive and affective-motivational properties of our experience, is (as Freud says) the cathexis of the object. A person or thing "seems more real" when its perception is attended by linked Olds(+) activity. Another component is the family of instrumental-act dispositions aroused by the percept. [Wapner has shown, for example, that the visual perception of movement in a schematic human stick-figure is normally enhanced by the subject's concurrent muscular activity.] On both counts, the schizophrenic should tend to perceive external objects as "less real." He has a reduced cathexis (lowered Olds(+) activation elicited by the percept); and his phobic-avoidant withdrawal from instrumental manipulation in favor of fantasy leaves him with attenuated dispositions to behave objectively toward the object.

It may be pushing theory too far, but I suggest that even the "sheer perceptual" distortions that occur in some (not all) schizophrenic derealizations may be explainable on this basis. The mere "unreality-feeling" is only quasi-perceptual, but we also get reports that the perception itself is changed, sometimes that objects seem smaller or farther away, sometimes that they appear larger. Recalling the Bruner-Postman research on how the "value" of an object (coin for poor kids, swastika during Nazi period) influences its perceived size, it might be that the attenuated Olds(+) feedback (abetted by exaggerated Olds(–) feedback) results in a reduced cortical "spread" in the perceptual assemblies, so that de-cathected external objects literally appear reduced. Such a reduction could be explained by the patient in two ways, either by attributing size-reduction or greater distance to the object.

Apparent size is only one correlate of cathexis, where I happen to recall Bruner's experimental material. Anecdotally, we are all familiar with a hard-to-name "reality"-quality similar to vividness, which is enhanced in normal persons by positive affective states. The fact that things look more vivid — I mean in the perceptual quality — when the perceiver is in a state of "joy," or "in love," or "euphoric," is part of the (valid) folklore. For some individuals (not all) it seems that the attraction of drugs lies partly in this heightened awareness of everyday objects. (E.g., when Wilfrid Funk was collecting data for his book If You Drink, several respondents gave this sort of explanation for why they liked alcohol. The most succinct statement of it was by Sturges Schley who, in answering Funk's query "Why do you drink?" replied "To make red redder.") That the sense du real is somehow closely tied in with Olds(+) activity level is most dramatically evidenced by the mystical experience, in which ecstasy [an affective state of pleasure to the nth] and cognitive certainty that "this is reality" coexist, the second component being rather puzzling to us non-mystics because the experience as reported sounds to us as if it were devoid of what is normally considered "cognitive content." Skeptics needle mystics about their inability to specify what is "known," but the mystic's inarticulateness doesn't bother him in the least. The positivistic critic argues: "You felt something special, I will admit; but you didn't cognize anything. It's evidently a hard-to-describe affective state, but knowledge is not involved. What is "real" is your special feeling, nothing objective.

So you shouldn't use words like 'certainty' or 'truth' or 'insight,' which are all cognitive words, involving a knowledge claim. The only knowledge you possess is introspective knowledge, i.e., knowledge by acquaintance of a unique emotional state." This reasoning strikes me, a mundane non-mystic, as perfectly sound. The fact that all higher mystics (including philosophically sophisticated ones) confidently reject it I take to be excellent anecdotal evidence of the intimate connection between Olds(+) activity and the sense-of-reality.

The opposite side of this picture is also anecdotally observed in normal subjects, although short of schizophrenia I know of nothing comparable in degree to the mystical. Most of us can report that in moods of "negative" quality (boredom, mild depression, fatigue, disappointment, worry) the external world actually looks a bit faded, drab, colorless, and somehow "non-vivid." It is noteworthy that sleepiness has the same effect, although here the origin is presumably a reduced RAS facilitation rather than decreased Olds(+) or increased Olds(–) activity.

181. Some schizophrenes experience a de-realization phenomenon of the opposite kind, in that objects appear larger, "enhanced," or even "closer," the last quality often being correlated with hard-to-convey body-image aberrations (e.g., "I sort of felt melted into the room," "I got scared because it somehow seemed like I was mixed with the other person") — the kind of thing that has led analysts to talk about the schizophrene's "breakdown of ego boundaries." My theory has more trouble with the oversize or over-intense percept phenomenon than with its opposite. I would like to neurologize the so-called "restitutional tendency," which psychoanalytic theory views in terms of the patient's sporadic attempts to re-invest cathexes, to "get reality back," to "form some sort of relationship with objects, however distorted." But I do not see any plausible way to put this in neurologese. It is not clear, I think, whether the de-realization effects involving lessened size, greater distance, or reduced vividness are on the same continuum with this second kind, because the latter often involves other features as well (e.g., attention-disturbance, trouble in "focusing") which may be psychologically orthogonal. But I must admit that, prima facie, "appearing closer" or "seeming larger" do sound, phenotypically, like the opposite end of a dimension. One thing we would like to know is whether the "enlarging" versus "shrinking" (or "removing" versus "closening") aberration in visual perception show a secular trend in longitudinal studies. [Here again, there is too little factual knowledge about the natural history of the disease. The more I think about schizophrenia, the more important this neglected area becomes!] My prediction would be that in the early stages of decompensation (if it is disintegrative in extent, even transitorily), there will be an oscillation between the "close" and "far" experiences; whereas in the chronic disintegrate, the "far" (and the "faint, washed-out, non-vivid") will predominate. I say this because I have seen both phenomena treating early cases, and I think I have seen them oscillate, but am not at all sure of this. My hunch is that we deal here with the well-known oscillatory dyscontrol of servomechanism theory (the overshooting ship-rudder is the classic case), where parametric deviations — and they may be very slight — prevent the mutual feedback controls from moving to stability (either steady-state or small excursions) but instead the system undergoes extreme excursions about some middle but unstable value, which itself may also display secular trend. I realize this is somewhat abstract, and that is why I say that my theory is weak in this area of facts. But there are so many examples of extreme excursions in the decompensating schizotype that I feel rather confident that some such servomechanism

model must be at least part of the truth. Of course in the disintegrated case (especially the catatonic) the "extreme values" business is well known since Bleuler. The alternation of catatonic immobility with catatonic raptus; of "automatized obedience" with negativism; of verbigeration, echolalia and logorrhea with mutism; of mimosa-like withdrawal with aggressive acting-out — all of these are well-attested clinical phenomena in catatonics. The "gear-shifting" mood change is, when very marked in intensity and sufficiently rapid (i.e., seconds or minutes) almost pathognomic of schizophrenia. Pre-Bleuler, ole Kraepelin (Lectures on Clinical Psychiatry) pointed out that in differential diagnosis between manic-depression and dementia praecox, such rapid-fire mood shift strongly supports the latter diagnosis. No case of manic-depressive depression or agitated melancholia will ever shift from black despair to euphoria or unconcern in a five-minute interval; whereas schizophrenics often do this.

Pursuing the oscillatory thing a bit further, I would conceive of several variables as acting in concert upon the perceptual assembly-systems involved in "seeing an external object." These would be:

(a) Olds(+) feedback (tends to enhance, enlarge, vivify the perceived object)

(b) Olds(–) feedback (tends to wash out, diminish, deaden the perceived object)

(c) RAS feedback (tends to enhance, except perhaps at very high values, where the percept may be "confused" by disorganized firing, hence appear unreal but on a different basis). I assume that corticopetal RAS levels are like Hullian "drive," in that their influence upon discrimination, differentiation, "resolving power," etc., is not monotonic. So that over a wide range of values (the "normal range") increased arousal improves resolving power — a generalization I take to be pretty well supported by the experimental evidence. Is there evidence for impairment at the extreme high end? I have usually viewed the old Yerkes-Dodson law in this way, but that's maybe far-fetched.

(d) Driving from regnant need-variables, wherever they are represented in CNS.

I realize I have spent too little time on the class (d) factors, and that they would be the main component in the "restitutional" mechanisms of psychoanalysis. [Where are the Murray needs "located" in the brain?] If we assume that as a result of long-continued avoidant patterns the schizotype builds up non-gratified id-impulses, there will occur (perhaps largely "accidentally," as when he becomes sexually aroused by a movie, affectionally deprived by his pet dog's death, or deflated in self-esteem by his brother's earning a Ph.D.) periods of unusually heightened need-regnancies. Even partially de-cathected objects now exert more attraction, but the resulting impulses to adient instrumental acts [= "attempts to relate"] are of course opposed by blips of strongly-conditioned anxiety. I gather that both Olds(+) and Olds(–) centers tend to drive the reticular system, so RAS feedback will be heightened. It is pretty easy to see, in a general qualitative way, how these four components could be engaged in ballistic swings of overshooting. But each such "swing" will meanwhile alter some of the inter-assembly control linkages, however slightly, so that the mutual feedback parameters [a molar long-term effect] are themselves being altered over time. The system as a whole will keep oscillating (and shifting the zero-point) until a steady state (or one with damped excursions) is reached. This is chronic, burnt-out schizophrenia. There is no guarantee that a steady-state will always be reached, and of course there are a few schizophrenes

who become "chronic" without becoming "burnt-out," i.e., they are still oscillating in activity, withdrawal, aggression, sexual display, delusion-formation, etc., after many years of hospitalization. Anyhow, the best I can do with the two opposite kinds of visual-percept derealization is to say that whether objects appear enlarged or diminished, near or far, intense or washed-out, must depend upon the momentary net resultant of the opposing feedback factors (a)–(d). I should add that hypokrisia per se may contribute to the "enlarging" effect if learned corrections for it are momentarily impaired, since phenomenal size-or-nearness should theoretically be somewhat increased by over-recruitment of neural elements, which hypokrisia produces directly. [Note added 6/18/66: Since writing the draft I have re-read more carefully your Chapman-McGhie reprints on disordered attention in early schizophrenia, in which the "enhancing" effect is claimed to be much more typical, almost to the exclusion of the "reducing." Since they systematically tallied complaints, I must presume their mental status data are more trustworthy than my anecdotal impressions. But I insist that both phenomena do occur, and I cannot explain the great preponderance reported by them.]

Some Major Objections

182. As a good Popperian I should ideally devote myself to refuting all theories, including my own, but who except a philosopher can really feel that way? I think it's permissible to hope one's theory survives tests, but the hard part is cooking up decent tests. Fortunately, as Popper says, we can rely upon others to discern weaknesses and advance criticisms! But there are some pretty strong objections that I do not presently see any satisfactory way of answering, so I shall go over them briefly.

183. The first objection is metatheoretical, namely, the vagueness of the theory as to its molar predictions. I shall not say any more about this here, because it was discussed earlier in Sections 41-45 and 120, in connection with open concepts and probabilistic indicators. I have had no new ideas about this problem since writing those sections. At the micro-level, the theory is pretty specific; but to utilize the hypokrisia concept in deriving strict consequences for, say, the stabilized retinal image situation, we would have to possess more detailed information about how the cell-assemblies in the visual system normally work.

184. I suspect I am going too lightly over the immediate consequences of hypokrisia for linkage growth, and if I am, you needle me about it, make me spell it out. It has an "obviousness" to me which is perhaps familiarity rather than rigor! We start with what, I believe, is well-founded synaptic neurology, namely, the single neuron rarely or never spikes as a result of a single input to its surface, but rather a confluence of inputs is required. Now obviously "confluence" means here both spacing and timing, since if the inputs were "well-located" (e.g., adjacent regions) but timed 10 seconds apart, no "confluence" exists. We also know that sub-spike disruptions of the post-synaptic cell membrane consist of local depolarizations which quickly fade out when not facilitated by others (properly timed). Now I conceive 'hypokrisia' as being an exaggeration in either the spatial spread or the temporal persistence of such local sub-spike wavelets [the "persistence" could be a negative thing, namely, slowness of the re-consolidation processes that restore the membrane and its local polarization], such that the spacing-and-timing of pre-synaptic spike arrivals at specific synaptic loci necessary to give a spike-probability of value $\geq p$ is more broadly specified; or, putting the same thing the other

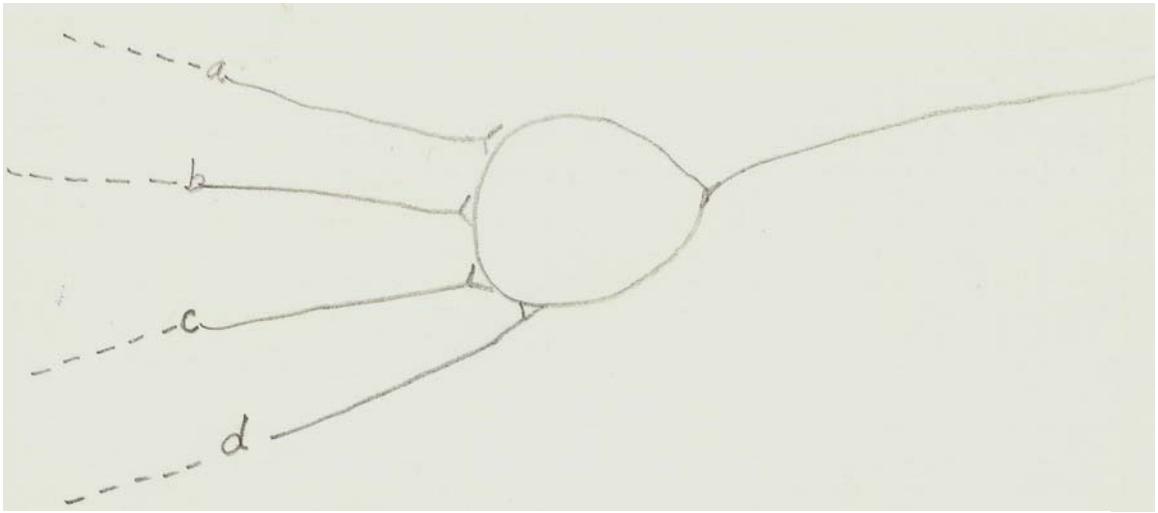
way around, a specified input pattern yields a higher p-value than in a normal brain. This is what I mean by saying that the hypokrisic neuron has a higher "signal acceptancy."

Consider a specified micro-anatomic configuration, defined by the distribution of placements of synaptic knobs on a neuron of specified shape and size. It is, I understand, accepted doctrine that certain input patterns [= spike-arrival times = coordinates in the "synaptic signal space"] yield probabilities $\simeq 1.00$ of the post-synaptic cell's spiking. Similarly there are other input patterns which yield a near-zero spike probability. I argue that at these extremes hypokrisia makes no difference. For overdetermined spikes, the hypokrisia is superfluous — because there's no place for the probability $p \simeq$ to go. At the low end, say an input consisting of only two spike-arrivals at knobs far apart and with the two arrival times separated by a millisecond, the input signal isn't even in the running, it can't bring the post-synaptic cell anywhere near spike threshold. Because a spatial hyperspread from either locus will still not give any "interaction" of the membrane disturbances, their centers of origin being too remote.

But there are many other inputs yielding optional transmission probabilities in the intermediate region; and it is these which the hypokrisia boosts a bit, from p to $p + \Delta p$. The increment Δp is "synaptic slippage."

The micro-details must surely involve Gestalt-type criteria for signal acceptancy. If any physical process is strongly "configural," this one must be. The spike-probability will be a complex configural function of signal-space coordinates, where the pattern of arrival-times in relation to their pattern of distances, angles, and topographic orderings is the functional law.

A matrix of times/against/positions could be set up for each micro-anatomic pattern, and the argument to follow employed mutatis mutandis. But without loss of generality, let me exemplify by considering spatial factors only, and, in particular, the composite criterion of number and adjacency of synaptic knob spike-arrivals. Imagine a synapse with four termini, from four pre-synaptic neurons, thus:



We will ignore time except as the defining interval, i.e., we ask simply which knobs are active during a time-interval Δt starting from an arbitrary zero (say, from arrival of the first impulse after the cell is out of refractory phase). Let us assume that three adjacent knob-arrivals will fire either a normal or hypokrisic cell of this micro-anatomic configuration. If no adjacent knobs are active during Δt, we are subthreshold for either a normal or a hypokrisic cell. But we suppose that two adjacent-knob arrivals will fire a hypokrisic cell, whereas this will not suffice to fire a normal cell.

Now imagine the set of knob-arrival patterns possible in an interval Δt. There are 16 such, counting the null-pattern. The possibilities, with the firing results in normal and hypokrisic case, are these:

Pre-synaptic Pattern	Normal Result	Hypokrisic Result
abcd	Fires	Fires
abc	Fires	Fires
abd	0	Fires
acd	0	Fires
ab	0	Fires
ac	0	0
ad	0	0
bcd	Fires	Fires
bc	0	Fires
bd	0	0
cd	0	Fires
a	0	0
b	0	0
c	0	0
d	0	0
Null	0	0

It is a direct consequence of the hypokrisic signal-acceptancy that, among the total disjunctive set of possible input-patterns, there are more of them which can get the cell over spike threshold. Now imagine a longer interval during which the assembly of origin (assembly containing cell-bodies whose axons are the termini a,b,c,d) is cooking at a certain activity-level. This controller-assembly activity generates a distribution of frequencies of occurrence of the 16 input patterns. Whatever that distribution may be, it is clear that it will yield more spikes in the controllee neuron, because some of the inputs will be of the too-adjacent type, and on such occasions the hypokrisic controllee cell will fire, but the orthokrisic will not. Hence, over such an interval, the mean spike-frequency of the controllee cell will be higher than is normal. The activity-level of the controllee assembly is measured [optimal metric known to Omniscient Jones!] by some increasing function of the discharge-frequencies of its component neurons. [Milsum, in his treatise on biological control systems, suggests that on evidence to date, a pretty adequate measure of "nervous activity" for a transmission line would be the average reciprocal of inter-spike times, i.e., a composite "pulse frequency."] Whatever the optimal metric, it seems pretty clear that any plausible index of controllee activation, increasing as the

number and rate of neural elements active increases, will show an exaggerated controllee level for any intermediate controller level. Only at extremely low or high controller levels will this fail to obtain (e.g., if controller levels never provide adjacent pairs in our example, or if they are continuously providing adjacent triads or tetrads, so that all elements in the controllee assembly are being "driven" as fast as their refractory-periods permit).

A possibility I have not explored rigorously is that the statistical effect of hypokrisia also yields an attenuated modulation (i.e$_e$, that dy/dx is reduced, while y_x is increased, for any given x). I don't know whether any general statement can be made along these lines without additional parametric assumptions. My intuition resonates to the idea, since in molar terms I would like to say that schizotaxic linkages are both exaggerated and poorly-modulated. Suppose, for example, that the "average" controllee cell is receiving mostly one-locus inputs. Then it will, on the "average" occasion, fail to spike, in either the normal or hypokrisic brain. If we increase the controller activation slightly, so that now the average controllee cell is receiving mostly two-locus (adjacent) inputs, it will "usually" spike in the hypokrisic brain, but still not in the normal. Hence the finite differential ratio $\Delta y/\Delta x$ will be larger in the hypokrisic case. But if we are operating at an average level of two-locus arrivals, a shift to three will yield an increment in the normal case, but not in the hypokrisic. This (rough) reasoning suggests that the derivative of the inter-assembly control function is higher for hypokrisics in the lower activity-region, but lower for hypokrisics in the higher activity-region, where the division of activity-regions into "lower" and "higher" is in terms of the pulse-frequency averages around some critical (intermediate) region. This line of thought is certainly worth pursuing, especially with regard to the derivation of aversive drift. You recall that we made use there of the notion of deceleration (or ogival acceleration-then-deceleration) in the linkage function. A reversal in the relative positions of dy/dx would, I intuit, tend to strengthen the quantitative core of that argument. But I have an uneasy feeling that it might work against the derivation, when details are pressed. Actually, this business of average value of y for a given x versus rate of change of y with x is a weakness in my thinking about hypokrisia that badly needs clearing up.

185. As for genetics, I suppose the main objection to a dominant gene theory is why schizophrenia occurs with such high life-risk incidence (1 per cent being very "high" when compared with other dominant mutations that reduce fecundity). I do not fully understand the force of this one, but I have to take Sheldon Reed's word for it. A recessive theory would, I gather, have less trouble here. Of course we know that neither a dominant nor a recessive theory fits the available genetic data, if one were to accept (recorded) psychiatric diagnosis as the indicator. But I consider this indicator so unsatisfactory that I don't take it very seriously. It is reliable enough to infer "something genetic is important," given the MZ-DZ differences found. There is plenty of non-genetic evidence to show, for example, that the false negative rate is greatly in excess of the false positive rate; but how much greater cannot be estimated precisely enough to provide a quantitative test of any specific genetic model. Furthermore, the system of genetic equations would have to be made more elaborate, by including coefficients for the differential effect of schizotypy upon the fecundity of males and females; and also for the differential effect of schizotypy in mothers versus fathers as social-learning factors influencing clinical decompensation in schizotaxic offspring. I have, surprisingly, not come across any equations in human genetics that include coefficients for such

influences. In order to prevent such corrections from being ad hoc "fudge factors," one must have more equations than unknowns, starting blind. Alternatively, if we postulate a dominant gene, the theory fixing this value to start with, we can test the model with one less equation. If there were two coefficients for the social-learning potentiating effect (by papa and by mama), and two for the fecundities, and one variable for the gene-frequency, we have a system with 5 unknowns, which is (in any empirical case) always soluble, so provides no test. A sixth equation is therefore needed. The published data on father → child, mother → child, child → father, child → mother, sib → sib, twin → twin might be adequate for this purpose, but the equations would get pretty complicated. I am still working on this, but it's tricky for a genetic novice like me to fool around with. So I am shelving this problem until I learn more about the mathematics of human genetics.

My choice of a dominant model is partly based (context of discovery!) upon my clinical impression that at least one parent of my schizotypic patients was pretty clearly "crazy," as retrospectively described by the patient; and that this crazy parent was more often mother than father (but not invariably by a long shot). The published data by Lidz et al., fit my experience, although they prefer to give an environmental interpretation. The McConaghy stuff on psychometric thought-disorder adds a little weight, as does the Phillips-Turner study of the same thing. However, the "big fact" to which I pay most heed in picking a dominant model is the Kallmann figure for schizophrenia incidence in offspring of two-schizophrenic parent-pairs. In this prospective study, it seems reasonable to assume that the potentiation of the offspring's schizophrenia must be near its maximum. Because each parent having disintegrated, we assume the polygenic potentiators to be loaded on both sides; ditto the offspring's schizophrenogenic environment. If the child of such a mating receives the specific schizogenic genotype, his chances of getting schizophrenia should be close to 100%. [Reasoning thus: People do develop schizophrenia. On a genetic theory, those who do develop it do so because (a) Their specific talent is potentiated by (b) Other genes and (c) Malignant family environment. Most of those who develop it are not from matings as bad as this one, on either (b) or (c); because most schizophrenics do not have schizophrenic parents; hence the parents of most schizophrenics do not have as much (b) as this, else they would have decompensated. I am also assuming that, on the average, (c) is worse in a two-schizophrenic parent home than in a home where one parent is merely schizotypic.] On a dominant model, we expect 3/4 of children of a two-schizophrenic (both heterozygous) mating to be schizotaxic; so the schizophrenia-rate among such offspring should be a little less than 75%, which agrees well with the empirical 68% found by Kallmann. Whereas a recessive theory should give us somewhere between 100% and the 86% he finds concordant in MZ twins. [Reasoning: Monozygotic twins reared together share (a) schizogene + (b) polygenic potentiators + (c) nurturing environment. Any failure of such pairs to be diagnostically concordant is attributable to life factors other than these three; hence, presumably, to extra-familial and adult environment "contingency factors" (mean boss, infectious disease, battle-axe wife, etc.). The confluent factors (a)–(b)–(c) which do produce decompensation in the proband fail to produce it in 14% of the cotwins. So I infer that "given the adverse factors (a)–(b)–(c) in amounts found in the average schizophrenic, the probability that a schizotype decompensates is .86." This logic needs some cleaning up, but you get the idea.]

So the dominant model fits this extreme case of two-parent schizophrenia better than a recessive model would. The dominant model predicts a value in the region from

(.86)(.75) = .64 to (1.00)(.75) = .75 for offspring of two schizophrenics, and if we arbitrarily split the difference at .69 we come very close to Kallmann's figure.

Reed points out that one way in which a selection pressure can be counteracted is by pleiotropic effects that are favorable to survival and fecundity in sub-clinical individuals, such as in incomplete penetrance for the "adverse" trait and higher penetrance for some other "favorable" trait, or as in a favorable phenotypic manifestation in the heterozygote. The classic example is the sickle-cell mutation, where the homozygotes succumb early to sickle-cell anemia (very low reproductive fitness) but the heterozygotes (in whom anemia behaves as a recessive) have a greater resistance to malaria than the normal homozygote does. (Hence the sickle-cell gene-frequency is much higher in malarial regions.) It could be that the well-compensated schizotype reverses the fecundity-trend seen in diagnosed cases, e.g., the female overbreeds as one of many manifestations of schizotypic social incompetence. But there are no data to support this directly, and it seems rather unplausible (unless mediated by the factor of social class?).

Theoretically it could be that the mutation-rate replaces the schizogenes lost by selection pressure in each generation; but I gather that a strong objection to this is that the required mutation-rate would be "too high" (i.e., mutations just don't happen that often).

So we must record the combination of two big facts, disease-incidence and selection-pressure, as an unsolved difficulty for the theory.

186. Apart from the very long-term question of why a dominant gene which in our society reduces fertility has not been eliminated, there is a short-terra objection from those who believe that the incidence of schizophrenia has actually increased within recent years. Needless to say, nobody has any sufficiently accurate data to prove this one way or the other. But supposing that it could be shown to be true, one can think of some rather obvious explanations of such a trend. The first explanation is a combination of genetic and social, namely, it might be that the fecundity figures, which are lowered in diagnosed cases, would be somewhat boosted in compensated cases. (Reminder: Kallmann and others report much greater influence in males than in females.) I can recall, without even checking my records, five of my own female schizotypic patients who became pregnant "unintentionally." They were all educated individuals, thoroughly acquainted with the best available modern techniques of contraception, and none of them had ethical, esthetic, or religious scruples against their use. Three of them actually became pregnant in the course of psychotherapy. In one instance, the pregnancy occurred after the patient had already made up her mind to obtain a divorce! The dynamics in these women included self-destructiveness, desire to "prove a point" about the feminine role, and some mixture of the irrational, inefficient decision-making that is associated with even mild forms of schizoid cognitive slippage. Now I can hardly make a case that this sort of thing would be sufficient by itself to counteract the reduced fertility percentages, but if, as I suppose, the "clinical penetrance" of the schizogene, i.e., the incidence of psychotic decompensation among schizotypic individuals, runs perhaps around 20%, i.e., that the schizotype-rate in the population is 5% as compared with the 1% incidence of diagnosed schizophrenia; then a considerably smaller positive fertility bias (associated with carelessness about contraception and with lower class status) would theoretically be enough to counter-balance the fertility reduction among decompensated cases. In diagnosed schizophrenia the reduction in birth rate is very considerable, amounting to around 50%. But Kallmann's data show that this reduction is very much greater in what he calls the

"nuclear" group than it is in what he calls the "peripheral" group. In the general population the birth-rate he gives is 3.3. For nuclear schizophrenia, the birth-rate is 1.4, whereas for the peripheral schizophrenic group, the birth rate is 3.1; so that for the peripheral group the percentage is really very close to that for the general population. If one were to extrapolate in a linear fashion the second order difference between the nuclear and the peripheral group and then move into the compensated group for which he has no data, it might be plausibly argued that the birth rate among them would be slightly higher than among the general population. If my mathematics is correct, on the assumption that there are four times as many compensated schizotypes as diagnosed schizophrenias in the population, an increase of the order of only about 1/12 in the birth rate among compensated cases would be sufficient to counteract the decrease of 1/2 in diagnosed cases. I have no positive statistical evidence to support this notion, but I can't see that we could rule it out as fantastic, especially if I'm right in believing that the main source of the schizogene for schizophrenic probands is the mother rather than the father.

There are sociological factors at work which would, I think, tend to increase the probability of decompensation in contemporary American culture if the gene-frequency were being maintained at a stable level, and quite possibly would be able to increase it even if the gene-frequency were slowly on the decline via selection pressure. The first of these sociological factors is the marked increase in ambivalence toward the maternal role in the American female, which is so obvious from common observation and clinical experience that I do not really feel much need to document it statistically. If, as I assume with Garmezy, the schizophrenogenic mother is an important potentiator of decompensation in those who carry the gene, this social learning factor would tend to counteract a loss of the gene from the genetic pool as reflected in our statistics on the frequency of diagnosed cases. A second sociological factor is the marked increase in the probability of case-finding which has been commented upon by many writers so I will not say any more about it here. Many mildly or intermittently decompensated cases come to formal diagnosis and hospitalization who two generations back would have been socially protected in a simpler, rural environment and especially in the context of the "extended family." Finally, as a follower of Spengler, I am convinced that modern Western society, with the decline of fellow-feeling and the dominance of the megalopolis (in which nothing really rules, says Spengler, except money and intellect) tends to increase whatever tendencies for social alienation a person has on the basis of his genetic background and his early nurturant environment. In today's large cities, it requires only a tiny dash of social aversiveriess in order for a person to become, and remain, permanently "alone."

While none of these factors can be accurately quantified, I believe that in the aggregate they provide an adequate basis for thinking that the proportion of schizotypes who decompensate is very likely on the increase, and that this could be more than enough to make up for any elimination of the schizogene from the genetic pool during the short interval for which we possess statistics on schizophrenia-rate.

187. Another difficulty is the kind of linkage-growth assumed in my speculative neurophysiology. I make repeated use of a linkage-growth-by-coactivation in the derivation of aversive drift. This gives me what I want for schizophrenia, but it maybe gives us consequences we don't want for learning theory generally. We dare not allow all kinds of assembly or assembly- system coactivations to yield strong linkage-growth and linkage-maintenance, because this would imply a powerful Law of Exercise (and would,

even worse, render operant extinction impossible). Refreshing you on what was said in those sections, what we assumed was that

a. Anatomically close assemblies readily become internally consolidated and mutually linked by mere coactivation, without "reinforcement." Example: Hebb's theory of learning to perceive visual objects.

b. Linkages from perceptual-cognitive systems to Olds(+) centers grow by "mere" coactivation, even though these are anatomically remote. But,

c. Anatomically remote linkages other than (b) grow weakly, if at all, by mere coactivation. To be of quantitative importance, these remote-system coactivations require facilitation (especially after-reverberatory?) from

   (1) Olds(+) feedback ("reward" reinforcement, typically drive-related)

   (2) Ascending RAS ("stimulus-onset" reinforcement, not typically drive-related)

I confess I rely heavily on armchair block-diagram reasoning in distinguishing these cases, although we know that the three kinds do occur, and that they do differ in molar properties. (E.g., "incidental learning" or "pure Law of Exercise learning" is very feeble when anatomically remote activations like a visual perception and an instrumental act are involved. The fact that the latent learning controversy went on so long is sufficient testimony to this generalization.)

Armchairing the block diagram, it has to be thus, doesn't it? Given that everything is potentially linkable with everything else (and the anatomical pathways must exist ab initio, otherwise no amount of training could link an arbitrarily chosen cue to a performable act; but we know that we can achieve such by conditioning), if it were the case that mere coactivation of such remote stimulus-response assembly-systems yielded strong linkage-growth, the process would "grow on what it feeds on," and all sorts of random, dysteleological learnings would be taking place to the detriment of the organism. And this is just not what happens.

However, there is one kind of remote linkage which should grow by mere coactivation, without Olds(+) facilitation, namely, the perceptual → Olds(+) linkage itself. Because in order for "reinforcement" to be available as a feedback-facilitator of inherently "neutral" coactivations, we have to start with something to get a foot in the door. My hunch is that this precondition involves some special micro-anatomy of the Olds(+) wiring, such that the initial quasi-random convergence of fibers between perceptual assemblies and Olds(+) centers gives rise to quantitatively unique growth-potential. The Olds(+) centers must have a privileged status as feedback providers, which two remote perceptual systems (or a perceptual and instrumental system) lack ab initio. I can't see how it could be otherwise, assuming God or evolution designed the machine for survival.

But my efforts to examine possibilities here became bogged down, because I realized I was, in effect, concocting a theory of learning! So if I am ever to bring this memo to a close, I must simply record the comment that my theory of schizophrenia has a major problem, how to avoid a powerful Law of Exercise as an undesired consequence of my neurophysiological assumptions. I note that the kind of linkage which grows by mere coactivation and tends to be self-maintaining on my assumptions is that between perceptual assemblies and Olds(+) or Olds(–) centers. This is, of course, the kind of

linkage which at the molar level has usually given most trouble to learning theories of the S–R reinforcement type (e.g., why anxiety-conditioning is so stable in neurosis, or what is the explanation of Allport's "functional autonomy of motives"). So, if it's any consolation, I reflect that I am in good company, and on an old belly-ache of an issue much broader than the etiology of schizophrenia. I suppose you assume, as I do, that Solomon and Wynne have a piece of truth. But that won't quite fix me up, because I do not have a place for extinction. Once a perceptual assembly gains control over an Olds(+) or Olds(–) center, we get repeated high-intensity coactivations, so the linkage would be expected to "grow on what it feeds on". Some neurotic behavior is admittedly like this (e.g., stage-fright). But it won't do as a general implication, so here I am in real difficulties.

188. Whatever the micro-details may be, the Solomon-Wynne concepts of "anxiety-conservation" and "partial irreversibility" surely have some molar truth to them; and I find it hard to suppose that their distinction between "traumatic anxiety" and less extreme amounts represents a qualitative difference. Neal Miller argues (against Allport) that if you carry out the experiment long enough, the apparent "functional autonomy" is shown to be only very high resistance to extinction. His main ground for this is the appearance of an extinction curve in the shuttle-box shock situation. But the graph (in Stevens Handbook) shows the behavior of a rat whose latency has risen to 5 seconds after 600 no-shock trials, i.e., the animal is still responding, however "weakly." Suppose we rashly assign a similar parameter to the schizotype in relation to a social-aversive event, e.g., girl refusing a date. If the patient's avoidant patterns lead him to try for dates an average of once a week, he would still be having an anxiety-blip after 10 years!

189. Freud's view of early anxiety-learning emphasizes the notion of a kind of "massive flooding" due to the child's undeveloped ego-functions — related to the "sense of helplessness" when aversive stimulation (or intense id-pressures) cannot be "handled" because the cognitive and executive ego-functions are not yet adequate to the task. It occurs to me that this "flooding" concept might be related to hypokrisia rather directly, in a way perhaps similar to the Chapman-McGhie findings on inability to suppress (or "selectively inattend") to inputs. A combination of id-impulses and learned external danger-signals which would be only moderately anxiety-arousing in normals would, due to the hypokrisic exaggeration in Olds(–) center activity, yield a "traumatic" level of anxiety in the schizotaxic brain. The same would be tentatively predicted for the arousal component, so that hypokrisic slippage in the reticular formation produces larger RAS feedback. Here again the parametric problem makes it impossible to say what the net (resultant) effect on linkage-growths would be. Hypokrisic heightening of Olds(–) activity will tend to suppress concurrent perceptual-cognitive activity (as in "denial", "repression", and "perceptual defense"); but the parallel, heightening of RAS activity will tend to excite these same perceptual-cognitive systems (as in "alertness," "perceptual vigilance," "sensitization"). Since we have anecdotal and experimental evidence that both phenomena occur, and differently in different types of subjects (e.g., selected by MMPI-profile), it is not purely ad hoc to speculate that some sort of balancing out occurs in the schizotaxic. The net algebraic sign of the Olds(–) inhibitory feedback and RAS excitatory feedback is critical for the long-term trend. One plausible suggestion, lacking parametric information, is that the arousal effect of aversive cues is, on the average, somewhat greater than the arousal effect of appetitive cues. If this is true in a general way, we might argue that the hypokrisia will influence the facilitative/inhibitory feedback balance

differentially, in that RAS-feedback will counteract Olds(–) inhibition relatively more than RAS-feedback will supplement Olds(+) facilitation. This might be an additional contributor to aversive drift. (I rather like this speculation, because it seems to me that, net hedonic tone aside, schizotypes do tend to become more "excited" by threats than by gratification-opportunities.)

190. It is not clear to me whether my theory is in difficulty about the paranoid/non-paranoid dichotomy among schizophrenics. If there are two diseases [= two distinct etiologies], we are in trouble, of course. I am betting on a single gene, once we exclude a few possible "phenocopies" (e.g., extreme introversion + high anxiety parameter + schizophrenogenic mother, combining to yield a pseudo-schizotype). The family studies show that there is some correlation of symptomatology; but they also show that exceptions do occur. It is also well-known that shifts in clinical picture occur in the natural history of the disease (e.g., a 50-year old state hospital chronic may have presented classical catatonic symptoms on first admission at age 20, then shown a "social recovery" for some years, then a paranoid picture upon re-admission at age 35, then a deterioration to something very like hebephrenic silliness and scattering, and finally a terminal picture which, had it occurred earlier, would have been labeled "dementia simplex"). The fact that late-developing paranoid cases are more common requires theoretical explanation, of course; but the existence of atypical cases is equally important etiologically. We must somehow reconcile the psychometric finding that most paranoid schizophrenics are relatively free of tested "thought-disorder" with the clinical fact that some of them also display Bleuler's associative loosening, both intra-verbal and in affect/content inappropriateness. In the textbook criteria for differential diagnosis between "paranoid state" and "paranoid schizophrenia," the affective inappropriateness and the associative loosening are usually emphasized. I of course hold that paranoid delusions (and, even more, hallucinations) are strong prima facie evidence of cognitive slippage. If the currently available psychometric measures of "thought-disorder" do not pick this slippage up, I incline to think that the tests are (a) Insufficiently sensitive [an ad hoc hypothesis] and that they are (b) Not idiographically built for katathymic potentiation [an obvious fact]. Nor is ad hoc hypothesis (a) entirely ad hoc, because the non-paranoid schizophrenics in all studies I have seen do show overlap with the non-schizophrenic controls, a psychometric finding that would seem to demonstrate less sensitivity than would be required in order to validly argue: "No psychometric thought-disorder, ergo no cognitive slippage present."

But the trends are consistent enough, even using ordinary intelligence tests, to constitute a "big fact" which any theory must explain. I will only sketch out the general lines of my tentative explanation. I start with the clinical fact that the paranoid form tends to have a later age of first admission. While many of these late-blooming cases do have (on careful anamnesis) a long history of schizoid maladaptation, the clinical fact is their ability to remain socially compensated for a long period. If my former patient "Miss X" [you know who] is ultimately hospitalized (as she probably will be when her savings run out, since she now can't work even in a sheltered environment) she will appear, on the record, as a late-blooming paranoid case. When I first saw her therapeutically she was a Hoch-Polatin pseudoneurotic, characterized mainly by pan-anxiety and anhedonic depression. Her first micropsychotic episode took place [after a couple of years]. Genuine delusion-formation did not occur until a year or two later. My guess is that she will be in a state hospital before she reaches age 45. As a beginning graduate student, she had an

8′27 Multiphasic, and I saw her as "schizoid" when I rated her for admission. What are we to think of a case like this?

191. My hunch is that the polygenic potentiators play a critical role in the paranoid/non-paranoid differences. The main ones I think of are "energy-level" and "aggressiveness." In Erik Erikson's refurbishing of Freud, the late anal and phallic stages are viewed as involving the development of autonomy and of motor, manipulative skills and attitudes, the learning to do things, to act upon the environment "aggressively," to refuse to submit, to "work one's will," to seek gratification by achieving mastery, etc. Perhaps the constitutional factor of "mesomorphic toughness" should also be included, along with energy-level and the aggression-parameter, among the polygenic potentiators tending to the paranoid form. Some of the body-type studies indicate that paranoid schizophrenics are more often mesomorphs; and in Lewis' classic study of post-mortem cases it was reported that paranoid schizophrenics had much larger heart-weight, aortic diameter, biceps muscle (i.e., mesodermal tissue-development) than the other sub-types. Psychoanalytic study of schizophrenics has, in general, stressed the element of "anal rage" in paranoid schizophrenia, while seeing the disorder generically as oral-fixated. Surely there is no type of patient whose volcanic anger is so frightening to behold as that of the paranoid schizophrenic in an extrapunitive mood (excluding epileptic equivalents).

So my guess is that if a schizotaxic has these three genetic dispositions (high energy, high aggression, high mesomorphy — and perhaps better intelligence?) he will tend to develop more adequate ego-function, both cognitive and executive, than one who is at the low and on these dimensions. Efforts to manipulate and understand the external world will be stronger, more persistent, and even more socially adequate than in the other type. The Murray need-system will be shifted toward a profile with greater elevations on n Cognizance, n Autonomy, n Dominance, n Counteraction, n Order, n Achievement, n Recognition (all of which are broadly "anal" and "phallic-intrusive" derivatives). Such persons will handle their conflicts by thinking, working, studying, struggling, competing — in spite of the concomitant tendencies to dereism that they share with the more withdrawn, scattered, mimosa-like schizotypes. They will use fantasy to excess, but will attempt to realize fantasy and to act it out. Hence the pronounced role played by "failure to achieve" when they finally decompensate, the so-typical mental content of unrecognized superiority, of vocational failure attributed to others' machinations, the grandiose perpetual-motion inventors and frustrated world-improvers which abound in state hospitals. Perhaps the later age of onset is partly a simple matter of "life finally catching up with dereism," in that the patient has been able to deceive himself for a long time by semi-psychotic mechanisms, always expecting things to take a turn for the better, but finally being forced to adopt more heroic mechanisms when the world fails to deliver? Precisely when this point comes would be a matter of many forces, including the "accidents" of life. We would view the age of first admission as a very crude statistic, reflecting a mainly "social-administrative" cutting score on a complex continuum, analogous to the multiple "resistance" factors determining at what age a diabetic or senile dement becomes diagnosably symptomatic.

192. How plausible this all is depends upon a statistical question in part, namely, whether the cluster of paranoid indicators is such as to show a latent taxonomy. If it should clearly show this [has it, yet?], my interpretation is a bit far-fetched, although not, I think, completely excluded. Consider the following:

193. In Section 17 on polygenic potentiators I suggested that for some of these (e.g. anxiety-proneness, basic introversion) the high end of the dimension raised the schizotype's odds of decompensating; for others (e.g., intelligence, energy-level) the low end did; but there were dimensions which theory and clinical experience suggested would be a handicap at either end (e.g., sex drive, rage-parameter). If, ceteris paribus, the middle region of the rage-parameter tends not to potentiate schizotypy into schizophrenia, whereas persons with either very weak or very strong dispositions to anger are more likely to decompensate, this would provide an easy derivation of the paranoid/non-paranoid typology. I take the rage affect to be the underlying "emotion" which becomes linked to a family of instrumental dispositions involving "attack" (as contrasted with "flight"), "activity" (as contrasted with "passivity"), manipulative coping as contrasted with withdrawal or sufferance. I view one set of Murray needs as mixed affective-motivational variables, the motivational component consisting of action-tendencies (with their associated cognitive-perceptual dispositions) that have been learned on the basis of their affect-reducing consequences. That is, "pure rage" as a relatively inchoate emergency emotion quickly becomes differentiated into a state which has cognitive and instrumental components, whereupon we classify it as a drive, employing in lay language such goal-oriented terms as 'hate,' 'vindictiveness,' and so forth. The prime motive thus formed by social learning (and even by non-social experience, such as learning to open a stuck door by kicking it vigorously) is Murray's n Aggression. But, as mentioned above, there is a family of related need-variables having at least partly the same developmental origin as n Aggression, chiefly n Autonomy, n Dominance, n Counteraction, and, in more sublimated, aim-inhibited guise, probably n Recognition, n Achievement, n Order, and even n Cognizance. The broad spectrum of controlling motives which variously exhibit the theme of "mastery" of one's environment — the urge to assert one's will, to refuse to knuckle under, to persist in the face of difficulties, to coerce and control others, to "make up for failures," to act rather than be acted upon, and to pull strings on the basis of "knowledge" — all of these are in varying degrees derivatives of the "aggressive impulse." If the non-paranoid schizotype had more of this, he would not withdraw and "give up" the battle so readily; he would give himself a fighting chance to succeed, to find at least partial gratification, to develop a self-concept of some personal worth. But at the other extreme, clinical experience suggests that the paranoid schizotype suffers in part from his exaggerated rage-readiness. After all, the world is full of Walter Mittys who fantasy greatness but docilely accept the minor bruises to pride that are the daily fare of "little people" in menial and subservient stations of life. But the paranoid is infuriated by these things. "How dare they treat me this way!" is an attitude that you can get by with if your IQ is 160 or you make a million bucks; but if you're a shoe-salesman, it becomes socially unworkable and subjectively intolerable. Taking the "fight-versus-flight" dimension seriously, it may also be that schizotypes with a sufficiently low rage-threshold are constitutionally prone to overdo the "fear → rage" sequence (Rado), which we know from Azrin's species-comparative research is either wired-in or early and universally acquired by animals. In such a case, the malignant polygenic combination of high anxiety-proneness with high rage-readiness would lead such schizotypes to an early development of the (fear → rage) transition, with the further consequence that the avoidant-withdrawal pattern is less striking in them because they "convert" fear into rage so quickly that the instrumental responses they tend to develop are more rage-guided than fear-guided. Such a schizotype would be more "effective," both in his social output and in maintaining subjective status ("hope," "success-expectation," "self-esteem") than the

person, who early learns to withdraw from seeking gratification or status by acquiring strong phobic, introverted, avoidant, passive patterns.

The statistical model of this situation would not even demand my assumption that high rage-readiness potentiates. The "insurance" against decompensation provided by aggression might be monotonically increasing, but the sub-set among paranoid schizotypes who nevertheless do finally decompensate would take longer to do so, and would be more "intact" when finally diagnosed. But my hunch is that the rage-parameter curve passes through a maximum of "disease-resistance," this maximum being shifted considerably to the right of the grand mean.

I don't think I am overdoing the importance of rage, since it is one of the two great "emergency-emotions", and since aggression occupies such a focal role in the psychodynamics of paranoid patients. Clinicians disagree about certain aspects of the paranoid pattern (e.g., Where does homosexuality fit in? Does the grandiosity precede or follow the persecutory ideation? Is there always a subtle hypochondriacal component?) but I have never read or talked to a clinician who denied the ubiquity of volcanic anger in these patients. And of course it is the paranoid schizophrenic — almost exclusively — who presents us with the most dramatic objective behavioral manifestation of extreme hatred, namely, he commits murder.

194. [Methodological note: Under what conditions does a "quantitative" (Galilean) variable give rise to a "qualitative" (Aristotelian) end-result? I have been puzzled about this for years. If Sheldon's mesomorphic component were due to a single gene, it would have to be pleiotropic for the main facets of the somatotonia cluster. So the energetic, tough, active, aggressive stuff — all part of the paranoid syndrome — could be taxonomic on this basis. Or perhaps the gene is for "energy" only, the aggression and muscular development being learned by persons with high energy-level.

[But if the mesomorphy is polygenic, how does a "type" emerge? We have the same statistical problem outside of psychopathology, of course. "The intellectual" is a type, in American culture. But one can hardly imagine an Aristotelian dichotomy as its causal origin. Ditto the "left-wing" and "right-wing" typology in American politics.

[One way to get a bimodal distribution is by autocatalysis. In B of O Skinner asks what would happen if the experimenter adjusts the schedule to match a given rat's extinction ratio [= mean responses per pellet on FI] with an imposed FR value? Result: Random fluctuations in short-term response strength lead some rats to get ahead of the matched value, hence to develop the high rates of FR; whereas others fall below and extinguish. End-result is "taxonomic" in the single output variable rate — actually not merely bimodal but non-overlapping! So even on one dimension, a taxonomy can develop from a continuum under suitable circumstances.

[Similarly, we can conceive that schizotypes above a certain critical value on mesomorphy have a probability >½ of entering upon a social-feedback cycle that strengthens their habits of paranoid defense, whereas others tend to move in the other direction. "Withdrawal" is perhaps the clearest form of this, where each socially-avoidant episode carries its increment of consequences, thus:

a. Reinforcement of withdrawal-tendency by momentary anxiety-reduction

b. Further consolidation of the self-concept "I am shy, I don't get on with people"

c. Further lagging behind in development of social skills

d. Further shift of the objective schedule (e.g., "See, there was no point in inviting him, he never accepts, he's a nut")

e. Further reinforcement of the dereistic substitute-gratification tendencies

[It is not hard to see how a bimodal distribution of "withdrawal" could gradually emerge from slight quantitative differences at the beginning. And I take it as obvious that when several different variables define the type-space, either interaction among them or environmental mold factors can result in two distinguishable populations becoming differentiated. But someone competent should go to work on the general logical and cybernetic formal properties of such systems. At least I hope to have shown you that, in principle, a statistical typology could arise from continuous variation in potentiators, both end-types having the same specific schizogenic etiology.]

Immediate Decision Problems on Strategy

195. If you and I are agreed to join our talents and defects in studying the psychophysiology and genetics of schizophrenia over the next few years [Deo volente!], the considerations in this memo give rise to certain immediate decision-problems in strategy and tactics. What general course should we plot? Which indicators, to be tried out on which kind of patient, and on which family-members, should we begin with? I think I am somewhat clearer on these issues as a result of writing the memo, but I am far from being resolved.

196. Take first the choice of indicators. Ideally, we would of course prefer an indicator-set which would function both (a) as a high-valid system of joint signs for testing a genetic model, and (b) simultaneously support or refute the neurological theory substantively. The latter requirement will be best met by indicators that are qualitatively diverse and not greatly influenced by shared nuisance variables, especially those of a "psychodynamic" nature. Thus we could not claim much support for a neurological construct as etiologically basic if the indicators were all "interpersonal" or "anxiety-based" in their manifest content, and showed sizeable correlations with psychometric or mental-status measures of affective-motivational variables. Fortunately this substantive requirement tends to mesh with the methodological requirement of high indicator-validity, in the sense that we want measures to correlate strongly with the latent taxonomy and negligibly within taxa (although the intra-taxon correlations need not be zero, as my PR-65-2 shows[4]).

197. How many indicators we need will depend upon the validity of each and their intra-taxon correlation, but for practical reasons (especially since cooperation of proband's family is required) the indicator-set should be fairly small. As mentioned in the early part of this memo, I no longer hold that the genetic model is testable only via indicators which taken jointly, are quasi-infallible. This would be nice, and I do not by

[4] Meehl, P. E. (1965). *Detecting latent clinical taxa by fallible quantitative indicators lacking an accepted criterion* (Report No. PR-65-2). Minneapolis: University of Minnesota, Research Laboratories of the Department of Psychiatry.

any means renounce all aspiration to this ideal. But I am currently satisfied that it is no absolute requirement. A set of, say, four to six indicators of moderate to strong taxonomic validity but negligible intra-taxon correlation would suffice to corroborate or refute the dominant genetic model, or any other "simple" genetic supposition. We can write the Dawes-Meehl equations for such a fallible indicator-set, and can then plug in the theoretical dominant-gene frequency-values to derive estimates of the frequency of indicator-patterns in family members of schizophrenic probands. As I now see it, a chi-square on a table of these pattern expectancies for parents and siblings provides a test of the model which is just as rigorous for validities of .70 as for validities of .99, although admittedly one's subjective conviction (and the profession's reaction) would be greater [rationally? I don't know] if the higher validities obtained.

198. Let me illustrate this by a crude example. Suppose we cooked up a set of four indicators each of which had a hit-rate of .75 in both schizotaxic and non-schizotaxic populations when cut symmetrically, i.e., with base-rate P = Q = ½, so valid-positive rate: p_s = Valid-negative rate: $q_n = {}^3/4$. As mentioned earlier in memo, with any luck at all we can do better than this, since some MMPI scales do this well against official diagnosis, whereas my souped-up MMPI index and the schizotypic checklist do considerably better against a <u>good</u> criterion [ahem: Meehl diagnosing a therapy case]. To get a hit-rate of .75 cutting symmetrically between two overlapping normal distributions would require the cut to lie at 1 P.E. above the orthotaxic mean and 1 P.E. below the schizotaxic mean, hence requiring these means to be separated by 2 P.E. = 1⅓ sigmas, approximately. On MMPI scale 8, this would require that schizotypes average around T = 63, and we can of course do a lot better than that.

Suppose that the four indicators are negligibly correlated pairwise. As I showed in PR-65-2, correlations often considered respectable in clinical work, such as in the .20's and .30's, turn out numerically to have a very small distorting influence away from independence when viewed in terms of the distribution of tallies in a 4-fold table. So as an approximation let us assume negligible intra-taxon correlation between the indicator-pairs.

The theoretical expectancies for indicator-patterns among persons carrying the schizogene and those not carrying it are of course symmetrical, cutting so as to yield a hit-rate of .75 in each group:

<u>Sign-patterns</u>	<u>Schizotypes</u>	<u>Non-schizotypes</u>
All 4 signs present	$p^4 = .3164$	$q^4 = .0039$
3 signs present	$4p^3q = .4219$	$4pq^3 = .0469$
2 signs present	${}_4C_2p^2q^2 = .2109$	${}_4C_2p^2q^2 = .2109$
1 sign present	$4pq^3 = .0469$	$4p^3q = .4219$
No sign present	$q^4 = \underline{.0039}$	$p^4 = \underline{.3164}$
	.9910	.9910

The dominant model requires every proband to have a schizotaxic parent, and a few of them may have two schizotaxic parents.

Taking the no-doublet assumption as a first approximation, we then assume that every schizophrenic proband has one, and only one, schizotaxic parent. From the preceding expectancy table, the incidence of sign-patterns 4 plus, 3 plus, etc., among parents is directly computable (multiply by ½). Further, this ½ schizotaxic is not random over parents pooled, but one schizotaxic must be found in each parental pair. So we can compute the sign-pattern expectancies for parent-pairs. Example: What is the probability that one parent has 2 signs positive and the other has 1 positive? This configuration can arise in two ways:

Schlzotaxic parent has 2 signs, non-schizotaxic parent has 1 sign

Probability = (.2109)(.4219) = .0890

Schizotaxic parent has 1 sign, non-schizotaxic parent has 2 signs

Probability = (.0469)(.2109) = .0099

So the expected frequency of a (2+, 1+) parent-pair pattern is .0890 + .0099 = .0989 or around 10%.

There are 25 distinguishable parental sign-configurations, if we ignore sex, so this provides a pretty sensitive chi-square test of the model. As argued earlier in the memo, I believe that mother is the genetic culprit in at least 75% of cases, and this asymmetric hypothesis generates a 50-cell table of parent-pair sign-patterns. But here, of course, no exact point-value is predicted. Actually I suppose one should test the gene-model, and if it fits, we then test the no-sex-difference hypothesis. If it stands refuted, we then use the equations to estimate the mother/father split. All I can predict from my theory is a preponderance of mothers over fathers (i.e., a mere "directional" trend). The 75% figure is an educated guess, based on asking "How often must mother be schizotypic to explain the nearly 100% 'schizophrenogenic mother' impression received by therapists?" The crude statistical model for this explanation is in my memo to Garmezy [Meehl to Garmezy, March 10, 1965: "Collecting MMPI profiles on parents of your schizophrenic sample," Section 11]. Of course some more direct support for the mother/father bias is provided by the fecundity data, which show that schizophrenia impedes mating and parentage more among males.

But I have wandered — the point of the numerical example is that modest construct validities with negligible intra-taxon correlations suffice to generate a well-dispersed set of theoretical frequencies against which to test the genetic model. Therefore our validity aspirations may be set realistically as regards selection of the indicator-family.

199. There is one set of indicators that will be studied, regardless, and on a larger N, with better criteria, than you and I could get, namely, the Ford Project stuff. This may yield either a set of high-valid MMPI keys, or a high-valid Self-Sort, or both. I am inclined to assume that at least one good indicator will emerge from that study, so that the "self-descriptive structured test" domain will be represented. I am reluctant to employ more than one such, partly because of the Campbell-Fiske point, and partly because this approach can hardly do much for testing a neurological theory [possible exception: Self-report on neurological complaints discussed in Section 77]. I would therefore propose that you and I set aside that whole area (self-description, whether by Q-sort or MMPI items) except for two possibilities:

a. We may want to develop a self-descriptive indicator based on item content we invent.

b. Ford data will shortly be put on tape here so that local analyses can be pursued independently of the Florida computer.

200. I favor including at least one quasi-neurological indicator, at least one measure of cognitive slippage, and perhaps at least one measure of anhedonia, since these are members of my schizotyplc tetrad. But, as mentioned previously, I do not favor working on an interpersonal aversiveness indicator, because I feel confident we cannot develop one which is even moderately free of non-schizoid nuisance variables. In the cognitive slippage domain, I favor using two indicators of highly dissimilar behavioral content (e.g., a visual-perception task and a conceptualizing task).

201. I take it as agreed that the selection of a sufficiently valid indicator-set should precede any family studies. With unlimited resources this might not be true, i.e., we could bring in relatives and throw the book at them, relying upon subsequent statistical treatment (if N is also large!) for (a) Selecting indicators (b) Cross-validating them (c) Estimating their joint taxonomic validity (d) testing several genetic models. But with limited resources such an approach — the old Lykken fantasy, which I also like — is not feasible. Hence, stage one is indicator-selection.

202. On what patient population should indicator-selection be carried out? Consider: If we show (or rely on the published research to show) that an indicator is highly valid for disintegrated schizotypy [= diagnosable psychotic schizophrenia, which is what 99% of studies have dealt with], we still need to find out if it has validity for the compensated case. Hence a study, by us, of the indicator on compensated cases is necessary. But if that is true, why not study only compensated cases from the start? Answer: Because we have no high-valid criterion for compensated schizotypy, and PR-65-2 method demands a huge N.

The only "out" I see here is to concentrate upon the one variety of compensated case which is sure-fire, i.e., "schizophrenia in remission." True, many of them are only semi-compensated; but if an indicator fails on this group, presumably it would fail to spot well-compensated cases diagnosed by Omniscient Jones.

The General Hospital Drug Clinic is the best source here. No practical means of getting drug-free cases there exists, but again, I assume that if an indicator fails when patients are medicated, it will probably fail when they are drug-free. This assumption may be more dubious, if there are any psychotropic drugs that operate directly upon the schizotaxic defect. I doubt this, for any except LSD, although Bus Glueck used to think that Stelazine did something very schizo-specific to the "cloudiness" component of schizoid thought-disorder.

The other possibility, perhaps to be pursued concurrently, is the Psychology undergraduate subject pool. We could use their SCB Multiphasic profiles as a crude initial screen (to get a decent base-rate) and run our potential indicators on them. The N here would probably be adequate, depending upon cooperation. But I suppose we would have to include a fairly detailed Mental Status interview in the criterion, and that is a pain in the neck.

203. I suppose the biggest strategy question of all is whether to begin with existing indicators or to construct new ones. It is depressing to reflect that after a half-century of

psychological testing and experimenting upon schizophrenia we do not have a clear-cut answer to this question. How do you explain this? I do not know whether there already exists a tetrad of 75%-valid indicators or not! Disgusting, isn't it?

The logistics of getting cases "in remission" from General Hospital present a problem, of course. I assume that the research-enthusiasm and general attitude of cooperation down there are adequate, but we can hardly expect to bring those patients to Diehl. This puts limitations on the apparatus (and space!) we can utilize. Whether the University Hospital OPD is a practicable source of subjects is doubtful, but I suppose we should at least look into it (sigh!).

If we relax the strictures as to compensation to include pseudoneurotic patients, the University inpatient service can probably provide subjects. It is hard to weigh the logistic problems against the disadvantages of studying semi-compensated subjects.

If you agree that the indicator-selection job must precede any family-study data, there is no point in discussing pros and cons of various types of family study at this stage (e.g., proband → offspring versus proband → sib), so with these somewhat pessimistic dilemmas before us, think I should bring the monster to a close.