PERCEPTUAL INDICES OF ORGANICITY AS RELATED TO PROCESS AND REACTIVE SCHIZOPHRENIA

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by

Joseph Manning McDonough

AN ABSTRACT

Submitted to the School for Advanced Graduate Studies of
Michigan State University of Agriculture and
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The purpose of this experiment was to obtain evidence about schizophrenia, particularly in relation to possible organic components. Much theory and some experimental findings suggested that at least certain schizophrenics suffer from a disease that is primarily organic in nature, perhaps of a central nervous system locus. Recent publications have sought to tie this variety of the disorder to the concept of process schizophrenia.

The two perceptual tasks chosen to investigate the problem were critical flicker frequency (cff) and the Archimedes Spiral aftereffect. The selection of these two tasks was based on the considerable amount of evidence that brain damaged individuals show gross defects in the perception of both.

Four groups of 20 subjects each were chosen. All subjects were male veterans under 45 years of age and with normal visual acuity in at least one eye. The organic group consisted of cases of known brain damage, as determined by case history and neurological examination. Twenty surgical cases from a non neuropsychiatric hospital comprised the normal group. Seventy-six patients with hospital diagnoses of schizophrenia, less than one year of current hospitalization, and no history of head injury were rated on the Elgin Prognostic Scale by two independent raters using case history material. A correlation coefficient of .89 was obtained between the two sets of ratings. The 20 patients with the highest Elgin scores were designated as the process group while the 20 individuals with the lowest scores made up the reactive group. All 80 subjects were then given both perceptual tasks.

Relative to cff it was hypothesized that: (1) brain damaged individuals would have lower cff thresholds than would normals; (2) process schizophrenics would have lower cff thresholds than would reactives; and (3) reactives would show cff levels not significantly different from normals. Hypothesis One was confirmed (.001) but Hypotheses Two and Three were not. In the latter case, reactives were significantly higher in cff than normals (.05).

Relative to the Archimedes Spiral aftereffect it was hypothesized that: (4) brain damaged subjects would perceive the effect less often than normals; and (5) process schizophrenics would not experience the aftereffect as often as either reactives or normals. Hypothesis Four was confirmed while Hypothesis Five was not.

A discussion of the results of this investigation considered the effectiveness of the cff technique in discriminating brain damaged from normal and schizophrenic individuals, and the relative inability of the spiral method to do likewise; the possibility that affective components of reactive schizophrenia contribute to the differences between them and other schizophrenics and normals; and the belief that process schizophrenia, while showing some physiological differences from reactive schizophrenia in other investigations, probably is not due to defects of a cortical nature.

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INTRODUCTION

The purpose of the present investigation was to obtain evidence concerning the etiology of schizophrenia, with emphasis on a possible relationship between it and central nervous system pathology. In addition, it was hoped that further verification of the validity and practicability of the concepts of process and reactive schizophrenia would arise out of the study, especially when viewed as the extremes on a continuum. And finally, it was hoped that additional meaningful data would come from the investigation relative to the performance of brain damaged, normal and schizophrenic subjects with critical flicker frequency and the Archimedes Spiral aftereffect.

The two perceptual tasks were chosen because of considerable evidence that brain damaged individuals show gross defects on both. Much theory and some experimental verification lend credence to the possibility that at least certain schizophrenics suffer from a disease that is primarly organic in nature, and which has perhaps a locus in the central nervous system. Recent publications have sought to tie this variety of the disorder to the concept of process schizophrenia.

Critical Flicker Frequency in Brain Damaged Individuals

When a stimulus is presented to the retina in the form of a pulsating intermittent light, the experience of flicker occurs to the subject. If the off-on cycles are presented at an increasingly rapid rate, a point will be reached when the viewer will report that he sees a steady light. This subjective point is called the fusion point, and the number of cycles per second (cps) necessary to produce fusion is critical flicker frequency (cff). This phenomenon was first reported in a publication by Segner in 1740. Since that time, more than 1300 publications have appeared on the subject of flicker or on closely related topics (83).

For some years it has been observed that, in general, brain damaged persons have significantly lower cff thresholds than do non-brain injured individuals. Numerous studies have been carried out in this area, especially in the decade following World War II, and generally high agreement is found concerning lowered cff for the organic subjects. Phillips (105) noted that the individuals with cerebral lesions showed lower flicker thresholds than normal subjects. In a study involving brain injured children, and using a group of non brain damaged mental defectives as controls, Werner and Thuma (137) obtained significant differences in cff between the two groups. With prolonged exposure in 15 organic subjects, Christian *et al* (31) noted that marked decreases in threshold appeared, while

normal, experimental subjects were not affected. Enzer, Simonson, and Blankstein (41) found that organics were much less liable to perceive the usual phenomenon of a second and third flicker after the initial cff point had been reached. Normal subjects did not display this difficulty. Miles (96,97) found cff defects in cases of multiple sclerosis, brain trauma and cases suffering from the effects of cerebral accidents such as aneurysm, occlusion or thrombosis. Further evidence of lowered cff in multiple sclerotic patients has been contributed by Ross and Reitan (112), and Parsons and Miller (104).

A good deal of careful experimentation with brain damaged subjects using the flicker fusion technique has been conducted by the group at New York University under the guidance of Teuber, Bender, and Battersby. Using large samples of Naval casualties with penetrating gunshot and shrapnel wounds of the brain, these investigators have published many articles showing that cff is significantly lower for these subjects than for normal controls (18,19, 131,132,133).

An interesting contradictory finding in the literature involves the importance of the frontal lobes in perception of cff. Halstead (54) reported that lowered cff is characteristic of patients with frontal lobe damage. This has not been borne out in subsequent studies by Battersby, Bender and Teuber (8, 9, 10, 128, 129, 130).

When 40 frontal penetrations were compared with 40 occipital-parietal gun shot cases and 60 controls with peripheral nerve injuries (but without head injury), Teuber <u>et al</u> (68) discovered that greater deficit appeared in the occipital-parietal group. For these investigators, one of the crucial factors seemed to be length of time elapsed since original damage. Lowered cff did not appear to hold in individuals where frontal damage occurred five or six years prior to testing. They concluded, that lasting defects in cff threshold apparently must involve higher brain paths than frontal areas alone.

Supporting evidence for this position appears to come from experiments in cff with lobotomized psychiatric patients. The Columbia-Greystone Project conducted in the late 1940's and early 1950's reported that no lasting defects of any significance were noted in patients who underwent lobotomy or topectomy of the frontal areas. In some cases lowered cff occurred post-operatively, but in general, the effects were transient and explained as due, in most part, to a dulling or lack of vigilance on the part of the operatee. Usual recovery of flicker threshold was accomplished within three months (71, 80, 82, 85,149).

Two studies contradict the usual finding of lowered cff in brain damaged subjects. Dean (36), found that cff did not discriminate between the organic and normal groups. Leiberman, Jlenig, and Hacker (88) were unable to obtain significant correlations between differing types of threshold responses and various diagnostic groups (including brain damaged individuals).

Many investigators have sought to explain the phenomenon of critical flicker. In general, two theoretical approaches have been utilized. The most popular view is based on the premise that neural events act in a chainlike manner, over restricted paths in the central nervous system. On the other hand, a minority of investigators lean toward a holistic or Gestalt view, where it is assumed that some sort of collective action takes place in the brain. Proponents of the latter position include Kohler, Koffka, Lashley, Goldstein, Kluver, Teuber, and Ogle (20, 21, 101).

Experts are also divided as to the neural locus of flicker. Is it primarily a retinal phenomenon or does it occur some time after the original impulse of light stimulates the retina, e.g. in the optic nerve, or in the visual cortex? Hecht and Verrijp (55) maintain that flicker is basically a photochemical event, but stress the fact that the retina is merely the "controlling influence," or the start of a complex process. Miles (97) on the basis of his investigations, makes the statement that "Flicker fusion is a retinal phenomenon in the normal person." Ross (113) sees cff as a function of the physio-chemical situation in the single receptor in addition to the total number of effective receptors in the retina.

For most investigators, however, non retinal factors are believed to be primary in the experiencing of fusion. Bartley (6) after numerous experiments, suggests that the "visual pathways further along are responsible and that the determination of cff is neural." Halstead (53) believes that cff is not due to any retinal process, but instead is the result of a fusion mechanism in the brain, probably the cortex. Simonsen, Enzer, and Blankstein (120) conclude from their study that a central process is primary in the perception of flicker, and that fatigue is probably the important factor. In a subsequent investigation, Werner and Thuma (137) agree, since brain injured subjects showed significantly lower cff thresholds than normals, yet no retinal deficiencies were observed. Bender and Teuber and their co-workers (17, 19,131, 132) adopt a "field theory," similar in many ways to Kohler's theory of figural after-effects (76) where "satiation" or lowered excitation is postulated as occurring in the occipital brain fields. Ogle (101) prefers to explain flicker and fusion in phenomenological terms until such time as considerably more is known about the neuroanatomy of the brain. It is the opinion of Landis (83), however, that

> . . . in spite of the fact it has entered into such a wide variety of investigations there is no comprehensive theory of flicker. The "general theory" of Plateau (1834) is as complete as that of any of his successors. Even such a simple point as whether the flicker-fusion threshold is dependent on retinal functional limitations or on limitations imposed by the central nervous system has never been clearly answered.

In a similar fashion, explanations are quite varied as to what causes lowered cff thresholds in brain damaged subjects in particular. Landis and Clausen (84) hypothesize that it is the duration of the dark interval which is the "link in the chain which is altered by brain damage," suggesting that the perception of a steady light persists only if the dark interval is of short enough duration to permit a second impulse to reactivate the neural pathway. Bender, Wortis, and Cramer (21) speculate that brain damaged patients undergo permanent "disequilibrium in the cortical field forces. Defects in sensory pathways, or even in regions outside the sensory pathways proper, lead to a re-organization and redistribution of the remaining forces. . . [but] the new field is no longer as stable as in the normal state." In a later article, Bender and Teuber (20) suggest that "intermittence of cerebral activities" is probably a mechanism which exists even in normal vision, but with cerebral lesions an "abnormal" intermittence occurs. Klein and Krech (73) feel that lowered flicker thresholds are due to the generalized lowering of brain conductivity, which is permanent, especially in instances of massive damage.

Summarizing the experiments which deal with brain damage and cff, we may say that the overwhelming amount of evidence indicates that:

1. brain damaged patients have significantly lower flicker fusion thresholds than do normals.

2. brain damaged patients with frontal lesions appear to suffer transient lowering only.
3. patients who have undergone psychosurgery (in the main frontal lobotomies and topectomies) have only a temporary defect in their post-operative fusion level.

Critical Flicker Frequency with Non Brain Damaged Psychiatric Patients

Although cff has been the subject of considerable scientific investigation for many years, it is only recently that the technique has been used to any great extent with persons suffering from psychiatric conditions of a functional or non organic nature. The earliest study involving mental patients and cff was done in 1906 by Wiersma (140) who reported group differences in fusion levels using manics, paranoids, melancholics and normals. He found that 11 manic patients had a mean score of 27.7 cycles per second (cps); 18 paranoids and melancholics, 12.2 cps; and nine normals, 14.7 cps. It was also noted that manics tended to have higher scores when in a manic state and lower fusion points when depressed. A replication of the experiment by Jones (61,62), some 23 years later, failed to confirm Wiersma's results in that melancholic patients obtained higher flicker rates than did manics. Ricciuti (109,110) attempted to differentiate between various diagnostic groups. His sample consisted of 15 manics, 13 depressives, 42 neurotics, 46 schizophrenics, and 54 normals. Mean cff scores were not significantly different between the groups with the single exception of normals when compared with manics. In this latter case, differences at the .05 level of significance were obtained. In an experiment involving 10 "psychiatric" patients and 10 controls, Klein and Schlesinger (74) obtained significant differences between the two groups after they had been divided into "form bound" and "form labile" by means of the Rorschach. Their hypothesis, that the "form bound" group would endeavor to "stabilize the unstable field," was born out, but no attempt to differentiate the groups on the basis of psychiatric diagnosis was undertaken. Ulett _et al_ (135), with a group of five patients, two of whom showed "symptoms of schizophrenia," and three with "depression and anxiety," report that two of their subjects underwent increased sensitivity to flicker during treatment with ACTH. In a second study, Ulett and Gleser (134) used a total of 96 individuals divided into three groups: Group 1 consisted of subjects judged least likely to develop symptoms of anxiety under stress; Group 2 was made up of psychiatric patients diagnosed neurotic or character disorder (i.e. all non psychotic); and Group 3 was a control group consisting of medical students. Groups 1 and 2 were subjected to stress (threat of shock) while the control group was not. Photic "driving," as recorded by electroencephalogram, was lowered in response to stress conditions. Psychiatric patients, however, did not return to higher "driving" on cessation of stress, while the non patient group did. Ostrow (102) tested 302 psychiatric patients on their susceptibility to photic driving. His results showed that 88 per cent of his sample exhibited susceptibility, but that the clinical diagnosis _per_ _se_ could not be related to the phenomenon. Bracken and Weidman (28), investigated cff with 13 schizophrenics and three depressives, before, during and after shock treatment (electric and insulin). Results showed that generally higher cff thresholds were obtained with improvement in the subjects' "mental condition." Fifty-eight "healthy" subjects and 51 patients (including 20 schizophrenics, and 11 ideopathic epileptics) were subjected to a number of experimental techniques including cff by Doust _et_ _al_ (38). Oxygen levels were found to vary with the rates of stimulation. Irvine(60) compared 26 paretics with 26 schizophrenics (matched on age, degree of psychosis, and length of hospitalization) and reported a significant difference between the groups (.01 level). A recent investigation by Ax and Colley (5) compared 22 brain damaged cases, most of whom had cortical lesions, with 21 psychiatric cases selected at random. Included in this latter group were 11 psychoneurotics, seven psychotics (five of whom were schizophrenic) and three character disorders. The sample ranged in age from 22 to 62 years. Flicker score differences, significant at the .01 level, were obtained between the two groups. Gravely (52) was unable to find differences in cff in comparisons involving hysterical, anxious, and normal soldiers. Krugman (78), on the other hand, found that fifty Air Force anxiety reaction cases differed significantly in cff from 50 normal controls.

It is apparent from this review of the literature that many of the studies using mental patients as subjects for flicker experiments are open to serious methodological criticism. These defects would include the small number of patients and controls often involved; the frequent combining of psychotic, neurotic and brain damaged individuals within the same group; and the wide differences in experimental technique. When these are added to the usual differences from one study to the next (e.g. dissimilarities in apparatus, amount of light adaptation, size of test patch, ages of subjects, intensity of the stimulus, binocular vs. monocular viewing, etc.), a tremendously complex situation results. As Granger (51) points out, these differences make comparisons between studies extremely difficult, if not impossible, and probably account for some of the contradictory findings.

Of the studies reviewed above, those by Ricciuti (109, 114) and Irvine (60) appear to be sufficiently well designed from a methodological standpoint to allow the following tentative conclusions to be drawn:

1. Schizophrenic patients have significantly higher cff thresholds than do brain damaged patients.
2. Schizophrenic patients do not appear to differ in cff thresholds as compared with normal subjects.

The Archimedes Spiral Aftereffect with Brain Damaged and Non Brain Damaged Psychiatric Patients

Visual fixation by the subject upon a slowly rotating Archimedes Spiral produces the illusion of expansion or contraction of the disk depending on the direction of rotation. When the spiral is brought to a sudden stop, the perceiver experiences an illusion just the reverse of that during rotation, i.e. a negative after effect. This phenomenon, sometimes referred to as the Plateau Spiral after effect, was first described by Plateau in 1850. His findings were confirmed by Oppel in 1856, Dvorak in 1870, and Bowditch and Hall some dozen years later (25).

In 1949, Freeman and Josey (43) administered the spiral to 50 normal patients and 85 psychotic patients from a state institution, the over-all age range for both groups being 8 to 76 years. Diagnoses in the psychotic sample included schizophrenia, manic depressive psychosis, dementia paralytica, epilipsy, cerebral arteriosclerosis and Korsakoff's syndrome. After being rated on degree of memory impairment, the patients were given trials with the rotating spiral. Results suggested to the investigators that a "strong probability" of correlation existed between negative after effect and impairment of memory. Standlee (121) took issue with this explanation. Using 25 psychotic and 16 normals, he concluded from his data that memory ability and the ability to experience the spiral after effect were not related. Working on Freeman's original assumption however, Price and Deabler (107) hypothesized that non-organic patients would perceive the illusion while brain damaged individuals either would fail to perceive the effect or would experience an incomplete effect. Forty adult males employed at the institution were used as controls, none of whom had a history of organic injury. The experimental groups consisted of 40 non-organic mental patients including schizophrenics, manic depressives and psychoneurotics, and 120 subjects with known cortical involvement. Diagnoses in this latter group included central nervous system syphilis, cerebral vascular accident, chronic brain syndrome associated with alcoholism, cerebral arteriosclerosis, post traumatic encephalopthy, and advanced stages of Parkinson's disease. Significant differences at the .01 level were obtained between normals and functionally psychotic patients on the one hand, and brain damaged subjects on the other. Results indicated that whereas approximately 92.5 per cent of the normals and psychotics experienced the after effect on every trial, two per cent of the organics were successful in obtaining perfect scores in perceiving the illusion. The experimenters concluded that failure to experience the effect was almost certainly to be considered an indication of cortical involvement. These findings appear to have set off a rash of experiments to verify the experiment since the obvious diagnostic possibilities of the spiral technique were readily apparent. In a published replication of the experiment Gallese (48) used known organic cases, mild or suspected organics, lobotomized schizophremics, and schizophrenics considered to be free of organic complications. He found that only 66 per cent of his organics were differentiated from the normal and non lobotomized schizophrenic group. Lobotomies and the mild organic patients did not differ from the schizophrenics and normals to any significant degree. Aaronson (1) using an epileptic sample, reported that only 14 of 65 failed to perceive the after effect. Seeking to corroborate the findings of Price and Deabler, but with a "carefully matched control group" on such factors as age, intelligence, and length of hospitalization, Page _et al_ (103) found that significant differences between the brain damaged and emotionally disturbed groups did exist. They concluded however, that Price and Deabler's theoretical implications were supported by their results, the value of the spiral technique in diagnosis was less evident due to overlap in the groups. Davids _et al_ (34) used the test with 15 cerebral palsied children, 29 emotionally disturbed non-organic youngsters, and 15 normals, and found significant differences between the groups. Whereas most of the organics failed to see the after effect, and most of the normals perceived it, the psychiatric group intended to be more variable in the response.

Theories as to what occurs in the perception of the Archimedes Spiral illusion vary. Shapiro (117, 118) feels that cortical irradiation suffers from additional inhibitory effect due to the brain damage itself Saucer (114,115) believes that patients undergo varying degress of loss in the ability to synthesize and integrate motion perception, a higher central nervous system function. Bender and Teuber (19) prefer a Gestalt field theory for descriptive, if not explanatory purposes. They propose that in organic patients, normal resistance to displacement in the cortical field is weakened, resulting in an "abnormal yielding" to the stress set up by the perception. Wertheimer and his co-workers (138,139) suggest that "satiation" (as used in Kohler's theory of figural after effects) "could be interpreted as implying metabolic changes," and therefore the size of an after effect may be thought of as a measure of the "metabolic efficiency" of the brain. Gibson (50) hypothesizes that the stimulus for motion is ordinal (i.e., the retina responds to adjacent and successive order) and relational (i.e. the perception of motion is related to and cannot be separated from the stable environment within which it occurs). Several mathematical classes of effective stimulation may be differentiated. Rotation in any object such as the Plateau spiral may be described as a type of "rigid" motion, or motion in which the figure after displacement is identical to the figure prior to displacement. These classes in Gibson's opinion are in turn "neatly correlated with types of physical events . . . which may prove to be psychophysically correlated with modes of kinetic experience."

In summary, although theories attempting to explain the phenomenon vary widely, most experiments involved with the spiral after effect appear to show that brain damaged individuals display some degree of difficulty in perceiving the illusion, while normals and functionally disturbed patients generally do not have this difficulty. The ability of the technique to serve as a diagnostic tool however, has not lived up to the original hopes of Price and Deabler (107) with subsequent studies unable to reach the heights of differential diagnosis reported by these experimentsrs.

Organic Factors in Schizophrenia

Traditionally, all mental aberrations including what is now known as schizophrenia were thought to be non-organic in nature, i.e. due to the work of the devil, possession of the afflicted soul by evil spirits, et cetera. However, with the rise of natural science in the 18th and

and 19th centuries, more and more emphasis was put into the attempt to find a physical basis for the disease. Success on the part of researchers in finding the microorganism responsible for paresis gave added incentive to investigators and theorists with a materialistic metasystem. The subsequent achievements of science in the past 50 years, and their overwhelming effect on the medical arts,continued to strengthen the position of those who postulate a physical basis for schizophrenia.

In 1896 Kraepelin (100) formulated the concept of dementia praecox (a term he borrowed from Morel), characterizing the disorder as a metabolic disturbance with an early onset, a gradual worsening of the patient's clinical condition, and an almost irrevocably poor prognosis. The disease was usually accompanied by such symptoms as hallucinations, delusions, withdrawal tendencies, bizarre thought processes, and inappropriate or dull affect. These symptoms appeared in varying combinations and with varying degrees of severity. In addition, Kraepelin suggested that clinical subtypes could be differentiated. These he designated hebephrenic, catatonic, paranoid and simple, each distinguishable on the basis of its presenting symptomatology. Bleuler (22,23,24), while largely agreeing with Kraeplein, observed that not all cases appeared to have early onset and a poor prognosis. Because he believed the "most important" characteristic of the disease to be the "splitting " of the different psychic functions, he suggested that the name be changed to "schizophrenia." Bleuler believed it was conceivable that the entire symptomatology could be psychically determined, but that the functional view had too many limitations to be regarded very seriously. These drawbacks included the usual absence of psychic trauma, the often present organic symptoms, and the fact that primitive civilizations had as high a rate of the disorder as did our own society. He concluded therefore(as had Kraepelin before him) that "Complete justice to all these factors [could] only be done by a concept of the disease which [assumed] the presence of [anatomic or chemical] disturbances of the brain." Even such "dynamically" oriented theorists as Freud (42) and Jung (63) postulate an organic basis for the psychoses. Jung for example, considers emotional upset as leading to metabolic abnormalities, which result ultimately in physical damage to the brain.

Numerous researchers feel that brain damage is <u>the</u> important factor in schizophrenia. Bateman and Papez (7) state that diseased cells of the thalamus may be at fault, or that changes in nerve cells of the prefrontal cortex occur as a result of pathogenic organisms. The latter finding was based on 70 biopsies of patients with dementia praecox. Anderson (2), expressing disillusionment with the Freudian psychodynamic approach to the problem, speculates that failures in interpersonal relations are "brought about by a very specific type of organic brain deficit," probably located in the most superficial layers of the cortex. Lauretta Bender (15, 16) with a wealth of statistics obtained at Bellevue, believes that "no child can develop schizophrenia unless predisposed by heredity" with the most important etiological factors being organic events such as birth injuries, severe illnesses, accidents and the like. Bychowski (29) in a discussion of one patient, hypothesizes that a "dynamic disturbance of the frontal apparatus" occurs in the schizophrenic, a situation not unlike that found in certain cases with brain lesions. For Eickoff (40), the arrested development of the schizophrenic child's abstract ability is due to general defects in acquisition of sensation. Again, one case is reported, in detail. Hoskins (58) in his "Biology of Schizophrenia," sees the disease as the product of defective maturation on both somatic and psychic levels, but concludes that "the next and most fundamental research in schizophrenia should be made not in the mental hospitals but in the biology labs."

From an examination of 1,087 records taken from the Berlin hospital between 1893 and 1902, Kallman (64,65) concludes that "the organic process of schizophrenia in the brain . . . attacks the cortex most exclusively and usually leaves the internal parts of the brain intact." This investigator reports that a "startling correspondence between expectation of schizophrenia and mortality from *tuberculosis* [*exists*]." "This statistical result is so unequivocal that it excludes all possibilities of coincidence and can only be interpreted as a genuine gene-coupling of the tendency to schizophrenia and the heredito-constitutional susceptibility to tuberculosis infection." He suggests that the locus of the hereditary weakness may be in the reticulo-endothelial cells. Landis (79) concurs with Kallman in the belief that schizophrenia occurs only in individuals predisposed to it through genetic background. Major (89) considers the primary cause of schizophrenia to be in the altered matabolism of the diencephalic neurons. Neilson (99) agrees that "the basic pathology must be diencephalic in location" since schizophrenic symptoms exist prior to any discoverable cortical signs. He believes that the condition is probably inborn. Roizin *et al* (111) in a discussion of a single case, reported finding considerable demyelinization of subcortical white matter. Davis and Davis (35) compared EEG records of 52 schizophrenic patients with 500 normal controls and were unable to observe and correlation between diagnosis and wave record, although a large number of the patients showed abnormalities outside the range of normal variability, and in some respects resembled closely the patterns of epileptic individuals taken between seizures. Kennard and Levy (69) found abnormal EEG's (often indicative of brain lesions) significantly more often in the more severe cases, especially those resembling *dementia* praecox. Winkelman and Book (142) concluded on the basis of ten autopsies of schizophrenics that gross brain appearance did not differ from normals, but that microscopic evidence did suggest the diagnosis. Dunlap (39), however, was unable to distinguish schizophrenic from non schizophrenic brains. Brackbill (26), summarizing the situation, points out that it seems "fairly well established" that structural damage appears in some schizophrenics, but that inadequate investigation of non psychotic subjects make evaluation difficult.

Some research has attempted to relate physique and susceptibility to schizophrenia. Kretschmer (76) believed that the disorder was most prevalent in asthenic or dysplactic body types. Kline and Tenney (75), with 1000 patients independently rated by Sheldon, discovered a positive correlation between mesomorphs and good prognosis, and a tendency for endomorphs to have a poor one. However, Wittman, Sheldon, and Katz (147), reported poor prognosis associated with ectomorphic individuals.

Evidence for organicity in schizophrenia based on the patient's performance on psychological tests was presented by Meadow and Funkenstein (92). After classifying their subjects according to physiological reactivity to epinephrine and mecholyl, they found a marked parallel between impairment in abstract thinking and autonomic nervous system response. The similarity in this respect between brain damaged and schizophrenic subjects suggested to them the possibility of cortical defect. The relationship between organicity and schizophrenia on the basis of psychological and physiological measures has also been reported by other experimenters, whose findings will be reported in a later section (27, 44,45,46,47,49,68,70).

In summary, a number of organic correlates for schizophrenia have been found as a result of biological, histological and genetic studies. Some support for the relationship between constitutional body type and susceptibility to the disease has also been reported. In addition, differences in physiological reactivity to drugs, and performance on psychological tests in a manner similar to that of brain damaged subjects has been reported by some experimenters to be present in schizophrenics. However, attempts to find organic involvement in schizophrenia specifically in the form of brain damage, have been less successful for the most part. One difficulty is that many, if not most of the studies, are inadequately designed from the standpoint of good experimental methodology, often lacking non psychotic controls. More than a few publications are based on a sample of one case, while others are merely the clinical impressions of the investigator arising out of his or her experience. Brackbill (26) identifies another source of confusion in his exceptionally fine review of the literature in this area. While concluding from the evidence that organicity is possible in some types of schizophrenia, he points out that much of the contradiction in findings probably can be blamed on the heterogeneity of the concept of schizophrenia itself.

Development of the Concepts of Process and Reactive Schizophrenia

The theorizing which has led to the concepts of process and reactive schizophrenia is a natural consequence of numerous studies which have appeared in the literature, plus a widespread dissatisfaction with the traditional Kraepelinian notion of dementia praecox since clinical experience has not been entirely supportive. Bleuler (22) recognized that the outcome of the diesase was not irrevocably poor, noting that almost complete remission was possible in some instances, especially where a relatively sudden and late onset occurred. Adolph Meyer (95) suggested that a psychobiological approach to mental disease was more profitable than being bound to the "nosological obsession" of premature classification. He advocated replacing the term dementia praecox with schizophrenia, recognizing the transitional forms of the disease, and adopting a conceptual system which would allow for both constitutional and environmental facts seen as interacting agents.

Excluding the traditional Kraepelinian categories, the most popular criterion used in experimental studies for the classification of schizophrenic patients has been that of prognosis. It has been observed for many years that some patients have relatively severe, stormy and acute schizophrenic "breaks," while others develop the disease at an early age and in an insidious manner. It has been noted also that a startling correlation appears to exist between stormy onset and good prognosis, while the patient who undergoes a slow, gradual deterioration is much more likely to have extremely poor chances of recovery.

The following variables have been listed by various investigators as being associated with the poor prognosis groups: early insidious onset (33, 67, 86, 98, 143,144); asethenic, dysplastic and pyknic body type (33, 77,90,98); introverted personality prior to illness (33,59,66,67,90, 124,146); inappropriate or dull affect (59,90); unmarried status (90); absence of precipitating stress (90,124); and family history of schizophrenia (33,98).

Good prognosis has been found to be correlated with the following factors: acute onset (23,33,67,90,98,124); precipitating stress (33,67,124,127); better pre-illness personality adjustment (59); neurotic and affective features (33,87,90,94,98,124); married status (90); clouded sensorium (23,59,66,67); cyclothymic personality (90); catatonic features (32,125); and short hospitalization (116,122).

Hoch (57) as early as 1921, described a few cases in which "spontaneous recoveries " had occurred. These he preferred to call "schizophrenic reactions" rather than instances of dementia praecox, since the latter disease was supposedly immune to remission. Harry Stack Sullivan (125), writing in 1924, pointed out that at least some cases of schizophrenia were not without hope of recovery, and that this pertained to catatonic individuals especially. In 1928 he listed "tentative criteria for malignancy" in schizophrenia,reiterating that not all cases have a poor prognosis (126). His later formulations conceived of dementia praecox as an "organic, degenerative disease" with insidious onset and poor prognosis, but schizophrenia as primarily a "disorder of living" with an acute onset due to situational stress. Prognosis here was seen as good (127).

Langfeldt (86) labeled the two groups "process" and "schizophreniform." In a longitudinal study of 100 schizophrenics over a period of six to ten years, he found recovery rates for the latter group to be much higher. In later papers, he has preferred to speak of "indefinite" schizophrenia as having a favorable prognosis, while "definite" schizophrenia does not (87).

Strecker and Wiley (124), stated that "good affect" was the best index of favorable prognosis in schizophrenia, along with stormy onset and significant precipitating stress. Strecker et al (123) in a more recent paper suggested that the stormy onset represents "the struggle of the personality against the acceptance of psychotic material." Cameron (30) also came to the conclusion that two main groups of early symptoms could be differentiated on the basis of type of onset. Symptoms of hypoactivity appeared to be correlated with insidious onset while hyperactivity was more characteristic of the schizophrenic with a shorter pre-illness history.

Wittman and Steinberg (143) studied the records of 434 neuropsychiatric cases of all types. Of the 59 who had histories of school referral problems, 35 were schizophrenic and none were manic depressive. More than 50 per cent of the schizophrenics in their sample had school records, and were described in these as being "shut in" personality types. The writers concluded that their study provided objective verification of Meyer's concept of the "shut in" personality type, a syndrome quite similar to Langfeldt's "process" schizophrenic. Wittman theorized that a personality continuum, in the form of a bell shaped distribution, best described the situation. This continuum ranged from "process" at one extreme to "manic-depressive" at the other, with the categories "schizophrenic," "normal," and "hypomanic and depressive" being intermediate.

Phyllis Wittman (146) took 66 adult schizophrenics who had been examined as children, and compared them to a randomly selected control group of 66 schizophrenics with no childhood referral record. She found that her experimental group tended to have an insidious onset and little or no improvement after shock treatment, while the controls had a somewhat better recovery rate. She concluded that Meyer's concept of the "shut in" personality type did differentiate the process from the schizophreniform and "thus [had] diagnostic and prognostic significance."

Ausubel (3,4) preferred to label two distinct forms of schizophrenia as "evolutionary" and "reactive," explaining them not in terms of possible organic factors, but in terms of ego deficiencies.

A series of studies has been carried out by Funkenstein and his co-workers (44,45,46,47), who have sought to investigate the relationship between physiological reactivity and prognosis. They found that schizophrenic patients varied in the degree of autonomic nervous system response when given epinephrine and mecholyl. After forming seven subgroups on the basis of blood pressure and reestablishment of homeostasis, it was found that subjects who showed greatest reaction to the drugs had better recovery rates after shock treatment. Low physiological reactivity was most often related to little or no improvement after EST. The writers concluded that their autonomic test was a better prognostic indicator than the clinical diagnosis of the patient. Confirmation of the relationship between prognosis and autonomic reactivity has been reported by Hirschstein (56) and Geocaris and Kociker (49).

Experiments by Meadow and Funkenstein (92,93) sought to investigate the relationship between autonomic reactivity to the adrenergic and cholinergic drugs and the patient's ability to think abstractly. Low reactivity groups were most impaired when tested on block designs, proverbs, and sorting tasks. From their findings they speculate that since "it is well known that an impairment in abstraction is also associated with organic brain disease, . . . the similarity of the loss of abstract ability in these patients and in those with organic brain disease would suggest a defect in the cortex."

Kantor, Wallner, and Winder (68) were the first to label the process-reactive dichotomy as such, and study it experimentally. A sample of 203 schizophrenics was subdivided into one of the categories or the other on the basis of 22 case history criteria. Patients were then designated as psychotic or non-psychotic according to their Rorschach responses. Results indicated that individuals classified as "reactive" from case history material tended to be called non-psychotic from their Rorschachs. However, cases judged to be "process" from their histories, tended to have a greater number of psychotic-like responses on the test. Kantor concluded from the study that it was legitimate to classify schizophrenics as being either "process" or "reactive," and that there was evidence that the two groups differed as to their "psychological function characteristics." Strong indications of reactive schizophrenia were felt to be: (1) a "relatively normal" pre-psychotic personality; (2) sudden onset with the presence of logical precipitating facts; and (3) loss of a clear sensorium. The process syndrome showed just the opposite characteristics. Brackbill and Fine (27) administered Rorschachs to schizophrenic patients classified as process and reactive, and to a third group with "known central nervous system pathology." Although it was impossible to differentiate the process from the organic group on the basis of Rorschach indices of brain damage, it was observed that both of these groups had significantly more individuals with five or more of the "organic signs" than did the reactive group. Brackbill and Fine suggest therefore that "the difficulty in differential diagnosis of some kinds of schizophrenics and organics results from the involvement of central nervous system pathology in process schizophrenia."

Devault (37), in an unpublished doctoral dissertation, sought to link the Kantor findings with those of Funkenstein, et al (44,45,46,47). Eighteen process and 18 reactive schizophrenics were chosen on the basis of pre-psychotic personality, precipitating stress, type of onset and clinical picture. A control group of 18 normals was used. All three groups were presented with four pictures (one "neutral" and three "conflict" situations representing hostility, dependency and sex). Physiological measures taken on each patient included base level and amplitude of heart rate, galvanic skin response changes, and GSR arousal and recovery time. An additional stimulus in the form of a loud bell was preceded by a verbal warning. Devault found that reactive schizophrenics responded in a fashion similar to controls in amplitude of GSR and heart rate change, but that they tended to a slower homeostatic recovery than process or normal groups. Patients classified as process had lower amplitude of GSR and the other two groups, and mean heart rate changes were all negative and significantly different from those of normals and reactives. Devault interpreted these results as being "strong evidence for at least two polar types [of schizophrenics] with possible intermediate mixtures."

The belief that schizophrenia may best be conceptualized as being a continuum from "almost complete psychogenicity" to "almost complete organicity" at the other extreme has been expounded by Bellak (13,14) who considers most cases as being mixtures of the two components in varying degrees. Becker (11,12) also adheres to the continuum position. In recent experiments he criticizes the usual dichotomous approach on the ground that there is usually great overlap in the groups, not to mention Bellak's evidence in favor of multiple causation, after reviewing 3,500 papers on schizophrenia. However, Becker prefers to view his continuum not in terms of psychogenicity-organicity, but rather as "levels of personality organization" with the process syndrome reflecting a "very primitive undifferentiated personality structure, and the reactive syndrome a more highly organized one." This, he believes, is a more fruitful point of departure. In his study, patients were classified by means of a prognostic scale as being on a process-reactive continuum. Hypothesizing that process schizophrenics would show more regressive and immature thinking than would reactives, he gave each subject a Rorschach and a proverbs test. Significant relationships between place on the continuum and Rorschach "genetic" scores were obtained for both male and female patients, but only for male subjects on the proverbs task. The feasibility of the continuum approach was given further support in two recent experiments by King (70). This investigator was successful in predicting the patient's autonomic responsiveness according to their relative position on the process-reactive continuum. Subjects were rated on the basis of case history criteria suggested by Kantor _et al_ (68) and, in addition, 22 cases were further rated with high reliability on the Elgin Prognostic Scale of Wittman (144,145). King reported that reactive schizophrenics showed a significantly greater fall in blood pressure level after mecholyl than did the more process patients, and that a significant rank order correlation existed between the degree of autonomic response and the subject's position on the continuum.

The advantages of a classification system based on the concepts of process and reactive schizophrenia appeared to be be promising enough to this investigator to have encouraged its use in the present experiment. The concepts seem to have both practical and theoretical points in their favor, since as Becker (11) notes, in the clinical situation it may act as a better guide in treatment programs than traditional Kraepelinian diagnosis. They have the further advantage of providing a possibly more exact psychiatric description than the presently used categories allow. As far as psychological research is concerned, it may open the way to additional insights into the etiology of schizophrenia, insights which may now be obscured by the use of outmoded diagnostic labels in current experimentation.

THE PROBLEM

From the foregoing discussion of the literature, several conclusions may be drawn with some justification. With regard to critical flicker frequency in brain damaged individuals, the overwhelming evidence seems to indicate that brain damaged patients have significantly lower cff thresholds than to non organic subjects. This has been substantially the finding with a wide variety of brain damage diagnoses, and appears to be true of children as well as adults. Exceptions apparently exist however when the injury is fairly well confined to the frontal area, including most cases of psychosurgery. Investigations using the flicker fusion techniques with psychiatric patients of a non brain damaged type have been fewer in number, and usually much inferior to the brain damaged studies both in experimental design and in the selection of experimental subjects. However, evidence from the better controlled investigations seems to suggest that in the case of schizophrenics, cff is significantly higher than with organic patients, and that the former generally show flicker fusion thresholds within the normal range.

Experiments with the Archimedes Spiral after effect have been concerned largely with brain damage. Price and Deabler's study showed extremely wide differences between brain damaged patients on the one hand and normal and mixed psychiatric groups on the other (107). Studies subsequent to this have been considerably less successful in replicating their original findings although most have confirmed the report that organics are inferior in some measure to both normal and psychiatric patients in the perception of the after effect (48,103). There is some evidence also, that schizophrenic patients are more variable in their response than are normals and organics.

For the better part of a century, the search for organic correlates for schizophrenia has been of major concern to a number of investigators in many branches of science. Biologists, chemists, physiologists, psychologists and neurologists have all sought to compile evidence that would point to some specific materialistic factor as being basic in the etiology of the disease. The results of these studies have been rather unrewarding in the long run. But the outstanding successes of 20th century science, especially in the fields of physics and chemistry, when coupled with the occasional suggestive finding in schizophrenia, combine to keep alive the hope that the disorder, in the long run like all other diseases, will prove to be basically organic in nature.

Although many of the investigations involving the use of schizophrenic patients as subjects are poorly designed and lacking in adequate experimental controls, it is felt by some (26,108) that the heterogeneity of the concept of schizophrenia itself is the primary culprit in obscuring any factors which otherwise might show themselves. As a result, increasing experimentation has been done in attempting to classify schizophrenics, not in terms of the traditional Kraepelinian categories, but for example, according to physiological responsiveness, constitutional body types, estimate of prognosis, and responses to psychological tests. One of the most recent suggestions is to classify patients in terms of a process-reactive dichotomy, or on a continuum with these categories as end points on a scale. Actually, this notion has had a rather long history, and is really an outgrowth of much prior experimentation and theorizing as to the nature of the disorder. Nevertheless, recent studies based on a process-reactive formulation of schizophrenia have resulted in some support for the speculation that process schizophrenia may be of organic etiology, while reactive forms may be due primarily to psychogenic factors. If this were true, many of the inconsistent findings in experiments using groups diagnosed simply as "schizophrenic," would be attributable to the heterogeneous nature of the concept.

Definitions

A process schizophrenic, for the purposes of this study, is an individual who has been given a clinical diagnosis of schizophrenia by the hospital staff and who, on the basis of his Elgin Prognostic Scale score (from a total of 76 schizophrenics) has been ranked among the top 20 subjects at the "high" end of the continuum. A person fitting this category could best be described as having an early and insidious onset of psychosis with an absence of precipitating stress, an inadequate prepsychotic personality with a tendency to withdraw from all human contacts, and presenting clinical symptoms showing a flat or dull affect and a relative absence of confusion.

A reactive schizophrenic is defined in this study as a person with a hospital diagnosis of schizophrenia and who, on the basis of his Elgin Score, has been ranked among the lowest 20 individuals in the entire schizophrenic sample (N = 76). A typical reactive patient is one with a relatively abrupt and stormy onset of psychosis, usually attributable to a logical and significant stress situation. The pre illness personality may be described as normal or neurotic, rather than schizoid, with perhaps some degree of outgoingness present. The clinical picture is likely to include severe confusion and many affective components.

Purpose of the Investigation

The present study was undertaken in an attempt to:

1. Gain further knowledge concerning the etiology of schizophrenia with emphasis on a possible relationship between it and central nervous system pathology.

2. Obtain additional evidence for the validity and practicability of the concepts process and reactive schizophrenia, particularly with regard to a continuum approach.

3. Secure additional data relative to the performance of brain damaged, normal, and schizophrenic subjects with critical flicker frequency and the Archimedes Spiral aftereffect, and in particular check on the effectiveness of the latter as a diagnostic technique in organicity.

Hypotheses

The following hypotheses were formulated:

1. Brain damage individuals will have lower mean cff thresholds than normal controls.
2. Patients rated as process schizophrenics will have a lower mean cff threshold than will reactive schizophrenics, and this score will tend to approach the score of brain damaged subjects, i.e. fall between reactive and organic patients.
3. Patients rated as reactive schizophrenics will perceive fusion at levels close to, or not significantly different from normal subjects.
4. Brain damaged individuals will perceive the aftereffect of the Archimedes Spiral less often than normal controls.
5. Patients rated as process schizophrenics will not perceive the spiral aftereffect as often as normals and reactive schizophrenics.

METHODOLOGY

Apparatus

Two pieces of apparatus were used in the experiment.

1. The Archimedes Spiral was made from a cardboard disc, off-white in color, and eight inches in diameter. A black India ink stripe, one-half inch wide, was painted on the cardboard so that it made one and three quarter turns from the center until it tapered completely off the outside edge of the disc. The spiral was mounted on a Stoelting color wheel whose speed was calibrated at the beginning of each morning and afternoon testing session, and set at 100 rpm's. The apparatus permitted rotation in one direction only. When rotated, an illusion of the disc contracting, or drawing into the center, was created in the typical viewer. On stopping the disc suddenly, the "normal" experience was to perceive the spiral as expanding.

2. Critical Flicker Frequency was produced by means of an episcotister. The apparatus consisted of a rectangular box, 24 inches wide, 16 inches high, and 10 inches deep which was open at the back permitting the experimenter to manipulate the necessary controls. It was painted a flat black color, both inside and out, in order to keep reflected light at a minimum. A black cardboard shield, 8 x 10-1/2 inches, was mounted on an arm extending 14-1/2 inches from

the front of the box. A "peep-sight," 3/8ths of an inch wide, was bored in the middle of the cardboard from which the subject viewed the flickering light monocularly. The cardboard was wide enough that monocular viewing was assured even while both of the subject's eyes were kept open. The test patch or target consisted of a circular aperture 5/8ths of an inch in diameter, cut into the fact of the box. Visual angle of the target was $2^{\circ}28'$ degrees. The light source was an ordinary 40 watt frosted bulb which was housed in a small box inside the apparatus. A small hole was cut in the lamp housing which allowed a beam of light to escape in the direction of the test patch. Three pieces of milk glass were placed between the bulb and the target: one directly in front of the bulb on the inside of the lamp housing box; one against the outside of the housing box; and a third placed immediately behind the target aperture. Intensity of the test patch was .085 candles per square foot, as measured by a Macbeth Illuminometer. A shutter panel which could be raised and lowered over the target aperture, allowed the experimenter to cut off the stimulus immediately at the end of each flicker trial.

Power for the episcotister was supplied by a Cenco Stirrer, a 115 volt, 60 cycle motor (type NS 1-12), built by the Central Scientific Company of Chicago. A light-weight, black cardboard disc was mounted on the motor, and fixed so that it cut across the beam of light at a point between the front panel of the apparatus and the lamp housing. Four equal sized sectors were cut in the disc in a manner to produce a light dark ratio (LDR) of 1.0. An ordinary automobile speedometer was used to measure the speed of the spinning disc. This was set to one side of the motor and driven by a rubber machine belt. Dial illumination was provided by a 7-1/2 watt white bulb, shielded in such a way that only enough light to read the speedometer was emitted. Calibration of the speedometer was done by means of a Strobotac and proved to be linear (1 mph = .5663 cps). The rate of rotation for the disc was controlled manually by the experimenter by turning a small knob on the motor. The speed of the motor at its slowest was 15 mph, at which a very marked flicker was produced. In a two day session prior to beginning the experiment, a voltage regulator indicated that voltages were approximately constant during the hours in which the experiment was to be run.

Subjects

Eighty hospitalized male veterans between 19 and 45 years of age made up the sample for this experiment (Table I). All subjects with extremely poor visual acuity or structural defects in both eyes were eliminated. However, patients with defects in one eye only, were retained in the group. The subjects in all groups were naive as to the real purpose of the experiment, having been told only that

TABLE I

CHARACTERISTICS OF SAMPLE

	Organic	Normal	Process	Reactive
Number of cases	20	20	20	20
Age				
Mean	35.20	31.15	26.25	33.60
S.D.	6.56	6.06	2.51	5.68
Education (grade completed)				
Mean	9.85	10.70	10.75	10.85
S.D.	2.76	2.45	2.20	1.31
Race				
Negro	8	8	4	5
White	12	12	16	15
Marital Status				
Single	8	8	18	7
Married	5	11	1	11
Divorced	7	1	1	2

they were to undergo two "special eye tests." Occasionally it was found that a patient who had been accepted for testing was so incapacitated or in such a confused (i.e. psychotic) state that he was unable to follow the fairly complicated instructions. This occurred with four organic patients, all of whom were eliminated from the experiment. In the schizophrenic group, although a good percentage actively hallucinated in the test room, only one proved to be totally unable to carry out the task as directed. However, among the 40 schizophrenics whose extreme Elgin scores qualified them for inclusion in either the process or reactive group, a total of four were not used in the experiment. Two patients in the reactive classification left the hospital before they could be tested. In the process group, one patient was transferred and the other was the above mentioned individual who was too confused to follow the experimental procedure. Selection of the experimental subjects was continued, however, until a total of 20 persons was obtained for each of the four groups. In the case of the process and reactive samples, the next two ranked patients on either end of the continuum were substituted for the patients who were dropped (Table II).

<u>Group I</u>. <u>Organics</u>. This group consisted of 20 individuals with <u>known</u> brain damage, who were chosen on the basis of their diagnosis (as determined by neurological findings of the medical staff), their case history material,

TABLE II

CLASSIFICATION OF 76 SCHIZOPHRENIC PATIENTS ACCORDING TO ELGIN PROGNOSTIC SCALE SCORES

Patient	Rater #1 Elgin Score	Rater #2 Elgin Score	Average Score for 2 Raters	Classification or Disposition
1. HG	17	19	18	Reactive (R-1)
2. SV	29	23	26	Reactive (R-2)
3. PC	25	29	27	Reactive (R-3)
4. PJ	30	26	28	Reactive (R-4)
5. KG	28	30	29	Reactive (R-5)
6. HD	32	27	29.5	Reactive (R-6)
7. HF	32	29	30.5	Reactive (R-7)
8. MJ	34	27	30.5	Reactive (R-8)
9. SJ	28	33	30.5	Reactive (R-9)
10. AR	30	32	31	Reactive (R-10)
11. KJ	35	27	31	Reactive (R-11)
12. BA	34	32	33	Reactive (R-12)
13. CL	32	37	34.5	Reactive (R-13)
14. NR	37	34	35.5	Reactive (R-14)
15. HJ	30	43	36.5	Reactive (R-15)
16. RC	39	34	36.5	(AWOL)
17. DC	33	41	37	Reactive (R-16)
18. AD	38	37	37.5	Reactive (R-17)
19. GW	37	39	38	Reactive (R-18)
20. PR	39	37	38	Reactive (R-19)
21. FD	28	48	38	(Discharged)
22. MG	40	37	38.5	Reactive (R-20)
23. CL	38	39	38.5	Middle Group(Not Used)
24. MI	37	42	39.5	"
25. MG	42	39	40.5	"
26. BR	45	37	41	"
27. UP	39	44	41.5	"
28. SC	36	47	41.5	"
29. MJ	46	40	43	"
30. CS	41	46	43.5	"
31. RJ	55	32	43.5	"
32. TL	47	41	44	"
33. PR	44	45	44.5	"
34. BA	44	45	44.5	"
35. NF	50	40	45	"
36. WW	49	41	45	"
37. DD	51	42	46.5	"
38. UG	44	50	47	"
39. WC	46	48	47	"

TABLE II (continued)

Patient	Rater #1 Elgin Score	Rater #2 Elgin Score	Average Score for 2 Raters	Classification or Disposition
40. WR	45	49	47	Middle Group(Not Used)
41. IH	47	48	47.5	"
42. BJ	48	48	48	"
43. KB	43	53	48	"
44. GJ	55	43	49	"
45. HH	49	49	49	"
46. KS	55	43	49	"
47. BD	55	45	50	"
48. HC	52	48	50	"
49. FW	49	54	51.5	"
50. SL	52	53	52.5	"
51. NP	58	48	53	"
52. KG	54	53	53.5	"
53. EM	53	55	54	"
54. SJ	50	58	54	"
55. DJ	52	57	54.5	Process (P-20)
56. PJ	56	54	55	Process (P-19)
57. PL	56	54	55	Process (P-18)
58. LN	52	58	55	Process (P-17)
59. RW	50	60	55	(Transferred)
60. BD	56	55	55.5	(Too confused)
61. WJ	52	59	55.5	Process (P-16)
62. SE	56	57	56.5	Process (P-15)
63. LR	60	55	57.5	Process (P-14)
64. MW	60	56	58	Process (P-13)
65. GR	58	58	58	Process (P-12)
66. HS	57	59	58	Process (P-11)
67. RG	57	61	59	Process (P-10)
68. ZB	63	55	59	Process (P-9)
69. BK	55	65	60	Process (P-8)
70. BW	62	58	60	Process (P-7)
71. DW	61	60	60.5	Process (P-6)
72. KC	59	62	60.5	Process (P-5)
73. RL	62	59	60.5	Process (P-4)
74. HJ	58	67	62.5	Process (P-3)
75. MJ	63	64	63.5	Process (P-2)
76. LF	61	66	63.5	Process (P-1)

and their presenting symptomatology. An effort to control for age factors (by not considering any individual over 45 for inclusion in any of the groups) severely restricted the number of brain damaged patients that were available at the Veterans Administration neuropsychiatric hospital where the bulk of the experimental data was collected. Over an 11 month period, only 14 patients could qualify for inclusion in this group. For this reason, the remaining six organic subjects were taken from the population of a general medical and surgical (GM & S) hospital of the Veterans Administration. The organic group ranged in age from 23 to 45 years with a mean age of 35.2. It consisted of eight Negroes and 12 whites, of whom five were married, seven divorced, and eight unmarried. Mean educational level was 9.35 years. Diagnoses included three multiple sclerosis; six brain trauma; four brain tumor; three Huntington's Chorea; two Parkinson's disease; one cerebrovascular accident; and one intercranial infection case.

Group III. "Normal" Controls. This group was made up of 20 individuals from two surgical wards of the GM & S hospital. It included every man in the two wards who was under 45 years of age and who had neither a history of emotional difficulties necessitating hospitalization or out patient treatment, nor a past record of head injury or long period of unconsciousness. Each patient was a volunteer since hospital rules required that subjects give their permission prior to testing. The patients were naive as to the purposes of the experiment however, since they were told only that they were to undergo an eye examination which would be compared with similar examinations done on mental patients at another VA hospital. Only one patient who was asked to volunteer, refused. This normal control group ranged in age from 21 to 43 years with a mean age of 31.15. It consisted of eight Negroes and 12 whites, of whom 11 were married, one divorced and eight single. Mean educational level was 10.7 years. All patients in the group were hospitalized for medical-surgical reasons, and diagnoses included varicose veins, appendicitis, torn cartilege, hemorrhoids, sacroiliac strain, and post operative foot, wrist, and hip cases.

The third and fourth groups were made up of patients diagnosed as schizophrenic. All of these individuals were chosen from patients assigned to the Acute and Intensive Treatment Service of the NP hospital. One hundred and sixty-one schizophrenic case histories were examined. From these, a total of 76 were chosen on the basis of their having a case folder with sufficient material to allow rating; no evidence, either behaviorally or from the record, of any head injury or long period of unconsciousness; a present hospitalization of not more than one year; a history free of repeated hospitalization over the past ten or 12 years for neuropsychiatric reasons; and definite evidence from the history of the presence of gross schizophrenic symptomatology, e.g. hallucinations, delusions, ideas of reference, and bizarre behavior. A brief description of the two schizophrenic groups follows.

Group III. Process Schizophrenics. This group of 20 individuals ranged in age from 19 to 36 years with a mean age of 26.25. It consisted of four Negroes and 16 whites, of whom only one was married, one divorced, and 18 single. Mean educational level was 10.75 years. Diagnoses included 16 schizophrenic reactions of the unclassified type, three paranoid type, and one catatonic type (Table III).

Group IV. Reactive Schizophrenics. These 20 patients ranged in age from 25 to 44 years with a mean age of 33.6 years. The group consisted of five Negroes and 15 whites, of whom 11 were married, two divorced and seven unmarried. Mean educational level was 10.85 years. Diagnoses included 13 schizophrenic reactions unclassified type, three paranoid type, two catatonic type, and two schizo-affective type (Table III).

An examination of the characteristics of the four groups (Table I) discloses that there were no differences between the groups in terms of level of education. However, since variances for the four groups were heterogeneous, a nonparametric test of significance was in order. The Kruskal-Wallis H test, using ranked data, was computed and found to be not significant (Table IV).

TABLE III

OFFICIAL HOSPITAL DIAGNOSIS OF SCHIZOPHRENIC PATIENTS RATED IN THIS STUDY AS PROCESS AND REACTIVE

Diagnosis	Process	% of Total	Reactive	% of Total
Schizophrenic reaction, unclassified type	16	80	13	65
Schizophrenic reaction, paranoid type	3	15	3	15
Schizophrenic reaction, catatonic type	1	5	2	10
Schizophrenic reaction, schizo-affective type	0	0	2	10
Total	20	100	20	100

TABLE IV

KRUSKAL-WALLIS H TEST FOR MEAN EDUCATION LEVEL DIFFERENCES BETWEEN GROUPS

	Organic	Normal	Process	Reactive
Mean grades completed (in years)	9.85	10.70	10.75	10.85
Sum of Ranks	700.5	808.5	845.0	856.5

$H^{(1)}$ = 3.10 (df = 3) Not significant.
(1) H is distributed approximately as Chi Square with df=k-1.

The same test of significance was applied to the mean ages for the four groups since again it was noted that homogeneity of variance was not present. In this case, a very significant H value was obtained, indicating that the four groups were different (Table V). A further analysis by means of another nonparametric technique, the Mann-Whitney U test, showed significant differences between various combinations of the groups when compared two at a time (Table VI). Process schizophrenics as a group were significantly younger than each of the other three groups. This finding may be accounted for in part by the very nature of the concept of process schizophrenia which is typically of "early, insidious onset." In addition, the selection device used (i.e. the Elgin Scale) reflects the younger age of process schizophrenics in Item B (Appendix). The inclusion of older process schizophrenics in the same sample which would have tended to equate this age factor, was impossible since length of current hospitalization was held to a maximum of one year. The only other significant difference between any two groups in terms of mean age level, occurred between organics and normals (.05 level). This is somewhat harder to account for. It may possibly be explained as due to the fact that organic subjects were not restricted to a maximum of one year's hospitalization since extreme difficulty was encountered in obtaining brain damaged patients for the experiment. In addition, the progressive nature of the

TABLE V

KRUSKAL-WALLIS H TEST FOR MEAN AGE DIFFERENCES BETWEEN GROUPS

	Organic	Normal	Process	Reactive
Mean age (in years)	35.20	31.15	26.25	33.60
Sum of Ranks	1051.5	782.0	444.0	953.5

$H^{(1)} = 18.45$** (df = 3)
** significant at the .001 level (two tail test)
(1) H is distributed approximately as Chi Square with df=k-1.

TABLE VI

MANN-WHITNEY U TEST FOR MEAN AGE DIFFERENCES BETWEEN PAIRED GROUPS

Groups	Mean Age 1st Group	Mean Age 2nd Group	Sum of Ranks 1st Group	Sum of Ranks 2nd Group	U	z
Organic vs Normal	35.20	31.15	480.0	337.5	272.5	1.96*
Organic vs Process	35.20	26.25	552.5	265.0	345.0	3.93***
Organic vs Reactive	35.20	33.60	439.0	381.0	229.0	.78
Normal vs Process	31.15	26.25	500.5	317.0	293.0	2.52*
Normal vs Reactive	31.15	33.60	362.5	453.5	247.5	1.29
Process vs Reactive	26.25	33.60	275.0	543.5	335.0	3.66**

* significant at the .05 level (two tail test)
** significant at the .01 level (two tail test)
*** significant at the .0001 level (two tail test)

pathology in some of the organic cases (e.g. multiple sclerosis, Huntington's Chorea) meant that patients with such diseases were generally able to remain out of the hospital until somewhat later in life. However, as was stated previously, no subjects in the experiment, including brain damaged, were allowed to exceed 45 years of age.

Relative to marital status (Table I), it may be noted that 90 per cent of the process group had never married while only 40 per cent of the organics and normals, and 35 per cent of the reactive schizophrenics had never married. Again, the large proportion of single individuals in the process group is tied closely to the concept itself. This is reflected in the Elgin Scale in Items A, D, and I (Appendix).

In the organic group, the highest incidence of divorce (58 per cent of those married) deserves some comment. In most cases, the divorce took place after the veteran's illness or accident occurred. From this it may be inferred that the divorce was attributable, at least in part, to the presence of the disability itself.

Classification of Schizophrenic Patients on a Process-Reactive Continuum

As has been mentioned previously, although most investigators at first conceived of process and reactive schizophrenia as a dichotomy, recent investigations have shown an inclination toward using a continuum approach. It would appear that the concept of a continuum has a number of advantages. In the first place, it would seem to best fit clinical observations since it is rare to find a case of schizophrenia where the history and symptomatology completely fall within the usual criteria for one classification as opposed to the other. Second, the idea of a continuum would appear to adapt itself well to a greater number of theoretical considerations than would a dichtomous approach. The theory, for example, could be along the lines of Bellak's (14) psychogenicity-organicity concept, or a purely psychological one such as Becker's (12), or again in terms of psychobiological interaction as suggested by Meyer (95,120). In the third place, the continuum is better suited for experimental purposes than is the dichotomy. Because of the relatively crude nature of our measuring instruments (e.g. the prognostic scales), a great deal of overlap occurs between the two groups. A continuum permits the use of many patients who would rightfully fall in the middle or mixed category. At the same time, it avoids a "forced" choice situation where borderline individuals must be placed in one group or the other, thereby increasing the chances of obscuring the results.

After deciding on the use of a process-reactive continuum in rating our schizophrenics, the next step was to choose a method of doing so. Becker's (11) revision of the Elgin Prognostic Scale was employed to rate the 76 schizophrenic patients originally selected. The Elgin Scale was devised and first used by Wittman (144) at the state hospital of the same name. Becker's revision (see Appendix) consists of 20 statements, each subdivided into a number of items describing more or less specifically, the degree to which the statement is applicable to the person being rated. Each of these sub items is assigned a numerical value. Whereas Wittman used only end points to rate each statement, Becker added the intermediate values in order to "add to the precision in rating" (11). In the present study, ratings on each of the 76 patients was done by two advanced clinical psychology trainees, one of whom was the writer. Prior to rating, ten case histories were chosen at random from the rejected cases and rated by each trainee. The results were compared and discussed in detail and by this means a considerable amount of agreement was reached as to what each sub item meant subjectively to each rater. Rating then began on the 76 case histories and was continued for a period of approximately six months as they became available. The raters' individual point scores for each subject are shown in Table II along with the average point score for both raters combined. A Pearson product moment correlation coefficient of .893 was found to exist between the two sets of ratings. This is significant at the .01 per cent level of confidence. The 76 cases were then ranked on a continuum, and the resulting distribution appeared unimodal and essentially normal in shape (Figure 1). The 20 patients

Figure 1. Distribution of Elgin Prognostic Scale Averaged Scores for 76 Schizophrenic Patients

N = 76
x = 1 subject

					x	x			
				x	x	x	x	x	
				x	x	x	x	x	
				x	x	x	x	x	
				x	x	x	x	x	
			x	x	x	x	x	x	
			x	x	x	x	x	x	x
		x	x	x	x	x	x	x	x
		x	x	x	x	x	x	x	x
		x	x	x	x	x	x	x	x
		x	x	x	x	x	x	x	x
x		x	x	x	x	x	x	x	x
15.1-20	20.1-25	25.1-30	30.1-35	35.1-40	40.1-45	45.1-50	50.1-55	55.1-60.	60.1-65

Good Prognosis — Poor Prognosis

receiving the lowest point totals were then selected as being most "process," and in a similar fashion, the 20 patients at the upper end of the distribution were chosen as being most "reactive" (Table II).

The decision to use Becker's modification of the Elgin Scale was arrived at after a consideration of possible advantages and disadvantages connected with the instrument. Disadvantages included certain violations of good scale construction (e.g. different ranges in sub scale numerical values from item to item), and probably unequal discriminatory ability along the entire length of the scale. Becker's (11) own criticism (i.e. that the scale is curtailed and not discriminating enough at the "normal" end), was thought to be a justified one. However, the advantages seemed to outweigh the disadvantages. In the first place, the method of selection used by Kantor et al (68) seemed less appropriate in a continuum frame of reference. Also, Wittman's original use of the scale (144) showed a very high two-rater reliability of .87. Subsequent validity studies by the same and other investigators (70, 145) showed the scale capable of predicting prognosis in a large percentage of the cases. Becker's modification (i.e. a numerical, weighted scale value for each of the 20 criteria) seemed to offer an even greater degree of exactness in the ratings, and at the same time permit a grading along the entire length of the continuum.

Procedure

The experiment was conducted in a room nine feet by 12 feet in which the sole illumination came from a 150 watt bulb directly in the center of the ceiling, 12 feet above floor level. A heavy canvas "blackout" curtain totally eliminated all light from the room's only window. All testing was done by a single experimenter (the writer) between the hours of 9:00 A.M. and 4:00 P.M. Subjects in all four groups were tested at various hours of the day in an effort to randomize the effects of time of day on cff.

Both the Archimedes Spiral apparatus and the episcotister were placed on a standard size office desk which served as the experimental table. In the case of the episcotister, a small wooden stand supported the box, raising the target aperture to eye level. Each subject was tested on the Archimedes Spiral first, followed immediately by the cff trials.

Procedure for the administration of the Archimedes Spiral. Each subject was seated at a distance of eight feet from the face of the spiral, and with the room under ordinary illumination. The experimenter, while pointing to the spiral, asked the subject to describe the color, shape, and design of what he saw. This was done in order to get an additional estimate of the visual acuity and degree of confusion in the patient. The experimenter then proceeded with the following instructions, after those of Gallese(48).

"This is a special eye test. Look at the center black spot and don't take your eyes away until I tell you to." After the disc had been rotating for ten seconds, the experimenter reminding the subject to keep staring at the center, asked "What does it appear to be doing?". If the patient's answer indicated that he saw contraction of the disc, no further questions were asked. If this did not elicit the correct response, the experimenter then said, "Does it seem to be changing in any way?" If this also was unsuccessful in bringing forth the correct answer, the experimenter asked the final question. "Does it seem to be getting bigger, smaller, or going away from you, or coming toward you, or anything else?" The subject's response was then recorded. At this point, the patient was again reminded to keep his eyes on the rotating spiral. At the end of 30 seconds of rotation the disc was stopped abruptly. The patient was then asked, "Now what does it appear to be doing?" A scoring system similar to that of Price and Deabler (107) was used in recording the subject's response. If his answer suggested that he perceived the aftereffect, he was given a score of one. If he did not report the illusion of expansion, a score of zero was recorded. In cases where the examiner was uncertain as to the subject's answer, a score of one-half was given for the trial. Four 30 second trials were administered to each subject with a one minute interval between each trial. The patient's final score was based on all four trials. A point of total of four therefore, would represent perception of the aftereffect on each trial, while a score of zero would indicate that he had failed to perceive the effect on any one of his trials.

Procedure followed in obtaining Critical Flicker Frequency. Immediately on completion of his fourth Archimedes Trial, the patient was asked to take a chair in front of the episcotister where he could view the flicker target through the peepsight. The following directions were then read to him. "This is another special eye test. In order for you to make a good score on this eye test it is very important that you follow my instructions carefully. Please keep your head from moving and do not blink your eyes. Face the box directly. Look through the peepsight with your best eye, and keep both eyes open at all times." (The patient was then checked as to his viewing technique, and this was corrected if necessary.) "The spot you see there (experimenter points to test patch) is going to flicker off and on. I am going to turn out the lights in the room and, after about a minute, you will see the flickering light in front of you. Stare at it steadily and don't blink. After a little while the light will no longer appear to be flickering. Instead, as you stare at it, it will gradually appear to become a steady light. As soon as it looks as though it is no longer flickering, you say NOW! . . . and I will turn if off. Then we will do exactly the same thing over again for a few times. Do you understand? (The main instructions were again repeated rapidly.) Remember, keep your head still, stare directly at the light, and don't blink."

The overhead light was then turned off and the only illumination came from the well-shielded 7-1/2 watt bulb in the rear of the apparatus which permitted dial readings. After one minute of dark adaptation, the shutter which covered the target was raised, allowing the subject to view the test patch. After ascertaining that he saw the flickering light, and that he was holding his head in the correct position, the experimenter manually increased the speed of the disc (at approximately one mph per second[1]) until the subject reported the appearance of a steady light. A total of ten successive ascending trials were taken with about 30 seconds between each trial. In order to control for possible auditory cues arising from the episcotister, an electric fan, placed adjacent to the flicker appratus, was used to mask the sound of the motor. This was done quite effectively by placing a small piece of cardboard on the fan in such a way that the blades would strike it, creating a rather loud, clattering noise. A regular sequence of varied starting speeds was used. The first trial opened at a dial reading of 15 mph. The remaining trials began as follows: Trial #2--20 mph; #3--25 mph; #4--20 mph; #5--25 mph; #6--25 mph;

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[1]Thus, for example, if the flicker trial was begun at a dial reading of 20 mph, and the S indicated fusion at a dial reading of 30 mph, a period of about 10 seconds would have elapsed.

#7--20 mph; #8--25 mph; #9--15 mph; and #10--20 mph. In this way it was hoped that, since a constant rate of acceleration was maintained, the patient would be unable to use subjective time cues in judging fusion without it becoming known to the examiner. After considerable practice the experimenter considered himself capable of reading the speedometer within one-quarter of a mile per hour at the instant when the subject reported that he perceived a steady light. In many cases where the examiner was unsure as to the realibility of the subject's reports, additional readings were given in the following manner, as a check. Various levels of flicker were set in a random fashion below, above and at the point of fusion. When the shutter was raised, revealing the test patch, the subject was instructed to report immediately whether it was flickering or steady. As many trials were administered as was felt necessary, in order to obtain an accurate estimate of the individual's fusion threshold.

Statistical Treatment

Critical Flicker Frequency. Of the 10 flicker trials on each subject, only the last seven were used in determining the mean score for each individual. The first three trials were omitted as practice trials after the observation that in the majority of cases, subjects used only in pilot experimentation were unable to report fusion thresholds on initial trials with full accuracy because of the novelty of the task. On the basis of the last seven trials mean flicker scores and standard deviations were computed for all 80 patients (Table VII). Generally, it was found that the subjects were quite reliable in their reporting of fusion as an examination of the individual S.D.'s will show. Only 12 subjects of a total of 80 (i.e. 15 per cent of the entire sample) had a flicker S.D. exceeding three quarters of a cycle per second, while only one subject had a S.D. of more than one cps.

The original design of the experiment called for a statistical treatment of the data by the analysis of variance technique. Specifically, an analysis of variance involving repeated measures on each individual (df =559) was to be done. However, when mean cff scores and their variances were computed for each of the diagnostic groups (Table VII), it was noted that the variances between groups were quite dissimilar. A Hartley F max test (136) revealed that variances differed significantly between the four groups Table VIII). Since one of the assumptions of the F test was not met, namely that of homogeneity of variance, another method of analyzing the data had to be found. It was decided that a nonparametric technique which did not rest on the assumption of homogeneous variances between groups, would be preferable. The Kruskal-Wallis H test (119), a nonparametric test analogous to the F test for more than two groups, was selected. The H test involved ranking of all subjects in the sample, regardless of group, according to their mean flicker scores with a corection for tied scores applied to the formula. Siegel (119) is of the opinion that the Kruskal-Wallis test is the most efficient nonparametric test for more than two independent samples, with a power efficiency of 95.5 as compared to the most powerful parametric test, the F test.

In order to get at possible differences in means between various combinations of the four diagnostic groups compared two at a time, another nonparametric test of independent samples was used. The test decided upon was the Mann-Whitney U test, which like the Kruskal-Wallis, makes no assumption relative to the homogeneity of the variances for the two groups involved. The U test requires that the mean scores for the 40 individuals being compared (i. e. 20 subjects in each of the groups) be ranked, with the person obtaining the lowest flicker score given the rank of one, and so on until all 40 are ranked as a single group. A correction for tied ranks is applied in this case also. Siegel states that in instances where the Mann-Whitney is used properly, its power efficiency is close to 95 per cent of that of the _t_ test, "the most powerful parametric test [for two groups]. It is therefore an excellent alternative to the _t_ test, and, of course, it does have the restrictive assumptions and requirements associated with the _t_ test."

He states further that instances have been reported in the literature in which the U test is superior to its parametric alternative in rejecting the null hypothesis (119).

To determine the relationship between any schizophrenic's rank according to his rating on the Elgin Scale and his rank on cff, Kendall's _tau_ or correlation coefficient was computed. Lastly, the discriminability of cff scores for the brain damaged group as opposed to the other three groups was determined and discrimination indices reported in percentages (Table XV). Finally, in view of the fact that the hypotheses of this investigation were not based on extremely well established theoretical grounds, it was decided that all levels of significance would be determined on the basis of two tailed tests.

Archimedes Spiral. Individual spiral aftereffect scores were determined on the basis of scoring system devised by Price and Deabler (107), a method which is comparable to other recent studies done with this technique (34,48). Individual scores are reported in Table XVI. Chi Square was used to compare the organics with the other groups.

RESULTS

Critical Flicker Frequency

Mean cff scores together with variances for each of the four groups are summarized in Table VIII. Group means in cycles per second were as follows: organics 13.20; normals 16.38; process schizophrenics 16.43; and reactive schizophrenics 16.67. However, a Hartley F max test (136) revealed that variances for the groups could not be considered homogeneous, thus necessitating the use of nonparametric tests of significance. The mean cff scores for each individual were then ranked (Table IX). A Kruskal-Wallis H value, significant at the .001 level of confidence was obtained, indicating that the four groups differed in mean flicker threshold beyond chance expectations (Table X). In order to discover exactly where the differences existed, Mann-Whitney U tests were run between all combinations of the groups in pairs. Table XI summarizes these results. It was found that the organic group differed very significantly (.0001) from all of the other groups. A significant difference in flicker fusion mean scores at the .05 level was also found to exist between normals and reactive schizophrenics.

Hypothesis 1. Brain damaged individuals will have lower mean cff thresholds than normal controls.

TABLE VII

INDIVIDUAL MEAN CRITICAL FLICKER FREQUENCIES[1]

Organic			Normal			Process			Reactive		
Subject	Mean	SD	Subject	Mean	SD	Subject	Mean	SD	Subject	Mean	SD
O1	11.79	.22	N1	16.58	.39	P1*	16.26	.57	R1**	17.88	.43
O2	11.45	.34	N2	16.18	.57	P2	15.96	.58	R2	17.98	.45
O3	13.45	.86	N3	16.32	.47	P3	17.84	.74	R3	17.21	.57
O4	14.72	.29	N4	16.91	.55	P4	16.12	.24	R4	17.09	.48
O5	11.81	.51	N5	16.32	.56	P5	15.29	.76	R5	16.18	.58
O6	15.07	.28	N6	16.37	.46	P6	17.48	.09	R6	17.25	.49
O7	13.49	.32	N7	17.49	.61	P7	16.54	.56	R7	16.32	.65
O8	14.41	.74	N8	16.28	.49	P8	17.05	.34	R8	17.21	.50
O9	17.11	.82	N9	15.75	.67	P9	15.51	.61	R9	17.27	.25
O10	11.47	.26	N10	16.04	.48	P10	17.58	.48	R10	16.50	.71
O11	12.28	.38	N11	17.13	.41	P11	15.83	.74	R11	16.70	.19
O12	12.74	.37	N12	15.74	.81	P12	17.21	.67	R12	17.67	.53
O13	11.18	.31	N13	15.78	.57	P13	15.03	.87	R13	16.38	.42
O14	14.80	.35	N14	16.44	.60	P14	17.17	.83	R14	14.28	.82
O15	13.61	.71	N15	17.30	.47	P15	15.49	.50	R15	16.24	.42
O16	12.96	.36	N16	16.32	.41	P16	15.82	.34	R16	16.46	.52
O17	13.95	.81	N17	15.82	.87	P17	17.66	.44	R17	16.34	.78
O18	13.13	.38	N18	15.97	.78	P18	16.59	.58	R18	17.35	.35
O19	11.65	.44	N19	16.20	.55	P19	15.79	.47	R19	16.67	.42
O20	12.96	.15	N20	16.74	.43	P20	16.36	.39	R20	14.36	1.10
Group Mean and Average S.D.	13.20	.445		16.38	.557		16.43	.540		16.67	.533

[1]Based on seven trials per subject. * Highest Elgin score. ** Lowest Elgin score.

TABLE VIII

MEAN CRITICAL FLICKER FREQUENCIES FOR ALL GROUPS AND HARTLEY F MAX TEST FOR HOMOGENEITY OF VARIANCE

	Organic	Normal	Process	Reactive
Mean	13.20	16.38	16.43	16.67
Variance	2.30	.26	.73	.94
F max	$\frac{2.30}{.26}$ = 8.81**	df 4,19		

** Significant at the .01 level.

TABLE IX

INDIVIDUAL MEAN CFF RANKS

Organic	Normal	Process	Reactive
1.0	26.0	21.0	16.0
2.0	27.0	23.0	17.0
3.0	28.0	24.0	37.5
4.0	30.5	25.0	40.0
5.0	34.0	29.0	44.5
6.0	35.0	30.5	47.0
7.0	37.5	32.0	50.0
8.0	39.0	33.0	52.0
9.5	42.0	36.0	53.0
9.5	44.5	41.0	57.0
11.0	44.5	48.5	58.0
12.0	44.5	54.0	62.0
13.0	48.5	55.5	67.5
14.0	51.0	61.0	67.5
15.0	55.5	65.0	67.5
18.0	59.0	67.5	70.0
19.0	60.0	73.0	72.0
20.0	64.0	75.0	77.0
22.0	71.0	76.0	79.0
63.0	74.0	78.0	80.0
Sum of Ranks 262.0	915.5	947.5	1115.0

TABLE X

KRUSKAL-WALLIS H TEST FOR MEAN CFF DIFFERENCES BETWEEN GROUPS

	Organic	Normal	Process	Reactive
Mean cff	13.20	16.38	16.43	16.67
Sum of Ranks	262.0	915.5	947.5	1115.0

H = 39.01** (df = 3)
** significant at the .001 level.

TABLE XI

MANN-WHITNEY U TEST FOR MEAN CFF DIFFERENCES BETWEEN PAIRED GROUPS

Groups	Mean cff 1st Group	Mean cff 2nd Group	Sum of Ranks 1st Group	Sum of Ranks 2nd Group	U	z
Organic vs Normal	13.20	16.38	224.0	596.0	386.0	5.03**
Organic vs Process	13.20	16.43	225.0	595.0	385.0	5.00**
Organic vs Reactive	13.20	16.67	230.0	590.0	380.0	4.86**
Normal vs Process	16.38	16.43	408.5	411.5	201.5	.04
Normal vs Reactive	16.38	16.67	335.5	484.5	274.5	2.02*
Process vs Reactive	16.43	16.67	361.0	459.0	249.0	1.32

* significant at the .05 level (two tail test)
** significant at the .0001 level (two tail test)

Since the mean flicker threshold for organics (13.20) was significantly lower than that of the normal control group (16.38), hypothesis one was confirmed.

> Hypothesis 2. Patients rated as process schizophrenics will have a lower mean cff threshold than will reactive schizophrenics, and this score will tend to approach the score of brain damaged subjects, i.e. fall between reactive and organic patients.

An examination of Table XI discloses that although the mean cff threshold for the process group was slightly lower than that of the reactive group (16.43 vs 16.67, respectively), the difference was not significant. In order to determine whether a more rigorous dichotomization would yield differences between the two schizophrenic groups, the 10 subjects rated as most reactive (i.e. lowest Elgin scores), were compared with the 10 patients rated most process (i.e. highest Elgin ratings). Again, no significant differences were found to exist between process and reactives in terms of flicker fusion (Table XII). Thus hypothesis two which predicts that the process group would approach the organic group in mean flicker threshold was not supported by this investigation.

> Hypothesis 3. Patients rated as reactive schizophrenics will perceive fusion at levels close to, or not significantly different from normal subjects.

A mean cff threshold for the reactive group of 16.67 proved to be significantly different from the 16.38 average of the normal individuals at the .05 level of confidence

TABLE XII

MANN-WHITNEY U TEST FOR MEAN CFF DIFFERENCES BETWEEN EXTREME[1] PROCESS AND REACTIVE SUBJECTS

	Mean cff	Sum of Ranks
10 Extreme Process	16.56	86.0
10 Extreme Reactive	17.09	124.0

U = 31.0 (n_1 = 10; n_2 = 10)
Not significant

[1]10 highest and 10 lowest scores on Elgin Scale

TABLE XIII

MANN-WHITNEY U TEST FOR MEAN CFF DIFFERENCES BETWEEN 10 EXTREME[1] REACTIVES AND 20 NORMALS

	Mean cff	Sum of Ranks
10 Extreme Reactives	17.09	215.0
20 Normals	16.38	250.0

U = 40.0* (n_1 = 10; n_2 = 20)

* significant at the .02 level (two tail test)

[1]10 lowest scores on Elgin Scale.

(Table XI), thus contradicting the prediction of hypothesis three. Following the rationale used in the analysis of hypothesis 2, a test was done in which the entire normal group of 20 subjects was compared with the 10 schizophrenic patients judged to be most reactive on the basis of their Elgin rating. In this case the difference between the two groups was even more significant (.02)(Table XIII). A comparison of the remaining 10 reactive patients with the 20 normal subjects did not result in a significant difference however (Table XIV). This leads to the conclusion that differences in cff between the reactives and normals are due to the 10 reactives with the lowest Elgin scores (i.e. the 10 patients judged to be most reactive). Similar comparisons involving the 10 most process and the 10 least process subjects versus the 20 normal patients did not result in any significant differences between the groups.

Additional Findings Related to cff

In order to determine whether or not a relationship existed between a schizophrenic subject's flicker fusion rank, and the same individual's rank on the Elgin scale, Kendall's _tau_ or correlation coefficient was computed. The resulting correlation of .048 indicated that there was essentially no relationship between the magnitude of a schizophrenic's Elgin rank (or position on the process-reactive continuum) and his ranked flicker score.

TABLE XIV

MANN-WHITNEY U TEST FOR MEAN CFF DIFFERENCES BETWEEN 10 LEAST EXTREME[1] REACTIVES AND 20 NORMALS

	Mean cff	Sum of Ranks
10 Least Extreme Reactives	16.24	171.00
20 Normals	16.38	294.00

$U = 84.0$ ($n_1 = 10$; $n_2 = 20$)
Not significant

[1] 10 highest scores among reactives on Elgin Scale.

TABLE XV

DIFFERENTIATION OF ORGANICS FROM ALL OTHER GROUPS ON BASIS OF CFF

Lowest cff		No. of Organics with cff Higher than Column 1	Per Cent Differentiated
For Normals	15.74	1	95
For Process	15.03	2	90
For Reactive	14.28	5	75

The ability of cff to discriminate brain damaged from non brain damaged patients was exceptionally good however. When compared with normals, organic subjects were successfully differentiated by their flicher threshold 95 per cent of the time, since only one brain damaged individual obtained a higher cff score than that of the lowest normal. Somewhat less discrimination was present in comparisons of process and reactive groups with the organics (90 per cent and 75 per cent respectively) (Table XV).

Archimedes Spiral Aftereffect

> Hypothesis 4. Brain damaged individuals will perceive the aftereffect of the Archimedes Spiral less often than normal controls.

Archimedes Sprial aftereffect scores for all individuals in the experiment are presented in Table XVI. It will be noted that with the exception of the organic group, all subjects in the study (i.e. 20 normals, 20 process and 20 reactive schizophrenics) obtained perfect scores on the task. When the organics were compared with any of the other three groups, a Chi Square value of 9.17 was obtained ($p < .01$). Therefore hypothesis four was confirmed.

> Hypothesis 5. Patients rated as process schizophrenics will not perceive the spiral aftereffect as often as normals and reactive schizophrenics.

Since all process schizophrenics in the experiment achieved perfect scores on the Archimedes Spiral, hypothesis five was not borne out.

TABLE XVI

INDIVIDUAL ARCHIMEDES SPIRAL AFTEREFFECT SCORES[1]

Organic		Normal		Process		Reactive	
Subject	Total Score	Subject	Total Score	Subject	Total Score	Subject	Total Score
O1	4	N1	4	P1	4	R1	4
O2	4	N2	4	P2	4	R2	4
O3	0	N3	4	P3	r	R3	4
O4	0	N4	4	P4	4	R4	4
O5	1	N5	4	P5	4	R5	4
O6	0	N6	4	P6	4	R6	4
O7	4	N7	4	P7	4	R7	4
O8	3	N8	4	P8	4	R8	4
O9	4	N9	4	P9	4	R9	4
O10	4	N10	4	P10	4	R10	4
O11	4	N11	4	P11	4	R11	4
O12	0	N12	4	P12	4	R12	4
O13	3	N13	4	P13	4	R13	4
O14	0	N14	4	P14	4	R14	4
O15	4	N15	4	P15	4	R15	4
O16	3	N16	4	P16	4	R16	4
O17	4	N17	4	P17	4	R17	4
O18	4	N18	4	P18	4	R18	4
O19	4	N19	4	P19	4	R19	4
O20	4	N20	4	P20	4	R20	4

[1]See page 57 for description of scoring method.

The ability of the spiral technique to discriminate organic subjects from non brain injured individuals was relatively poor. Of 20 organics, only nine failed to obtain perfect scores on the task. This resulted in a test discrimination index of only 45 per cent in the present investigation. Five of the nine subjects were totally unable to perceive the effect on any of the four trials. Another

organic patient perceived the aftereffect only once in four trials, while the remaining three individuals not obtaining perfect scores, had an average of three points out of a possible four. Eleven brain injured patients (55 per cent) were able to report the aftereffect on each of the trials however. An examination of the data revealed no apparent relationship between those patients failing, or almost failing the test (i.e. achieving scores of zero or one) and any particular type of organic diagnosis. The six patients with scores of zero or one included one cerebrovascular accident, one brain trauma, one multiple sclerosis, one Huntington's Chorea, and two cases of brain tumor.

DISCUSSION

Brain Damaged Patients and Cff

The results of this experiment on cff in cases of known brain damage are in full agreement with most prior evidence. Only one brain damaged individual obtained a flicker threshold higher than that of the lowest normal. This single exception, a patient suffering from the effects of a gun shot head wound, was the only member of the organic group whose injury was confined exclusively to the frontal regions as far as neurological examination could ascertain. Since most evidence points to the conclusion that frontal injury, either accidental or deliberate (e.g. psychosurgery), generally affects cff only temporarily, the frontal locus of the lesion for this particular subject may even explain the single exception. In short, the present experiment provides additional support for the position that cff is a comparatively good technique for discriminating brain damaged persons with gross destruction from normals and schizophrenics. Increased utilization of cff in the ordinary hospital setting appears to be well warranted, a possibility that has been recognized by Klebanoff (72) and others (54,81,97). Its potential seems especially good when a comparison is made with the relatively poor discriminating ability shown by the usual paper and pencil tests of organicity (148).

Cff and Process and Reactive Schizophrenia

Reactive schizophrenics, while predicted to have cff thresholds similar to those of normals (in the belief that no cortical defect is present), actually showed a higher fusion level than normal patients. This result, although unexpected, is not entirely incompatible with other research findings. King (70) points out that most evidence from mecholyl studies shows normals and poor prognostic (process) groups to have relatively low autonomic response levels, while good prognostic patients (reactives) respond much more strongly to the drugs. He cites other experiments (91, 105,141) which corroborate the impression that reactive schizophrenics have marked physiological reactivity generally while process subjects do not. In this sense the process individual resembles more the normal person. King suggests that "A possible explanation lies in viewing both normals and process schizophrenics as representing relatively uniform, stable adjustments over a period of time" while the heightened homeostatic response of the réactive patient reflects the "precipitous nature" of his adjustment.

Another explanation of the cff results with reactive subjects in this experiment may be offered. Some evidence from the literature indicates that patients of a manic type show higher fusion thresholds than do other groups (109, 140). In addition, there have been studies which suggest the possibility of a relationship between manic or affective symptoms and characteristics of schizophrenics with better prognoses (i.e. reactives) (30,44, 123).Selection of reactive patients on the basis of affective components in the clinical picture (Items A, D, G, P, T, of the Elgin Scale) may account for the significantly higher flicker levels for reactives as compared with normals. It should be noted also that only two of the 76 schizophrenics rated, (subjects R_1 and R_2, Table VII) carried a hospital diagnosis of "schizo-affective." Cff scores for these two individuals (both of whom were included in the reactive group) were the highest in the entire sample. We may speculate therefore that a manic component present in the reactive subjects may have contributed to higher cff levels attained by this group. Furthermore, it is possible that the present day concept of reactive schizophrenia has much in common with the formerly popular diagnosis of manic-depressive psychosis.

The experiment failed to reveal differences in cff between patients rated process and reactive. In considering possible reasons for this, a number of alternatives suggest themselves. First, the Elgin Scale may be at fault. But the fact that other investigators (11,12,70,144,145) have found that patients rated process and reactive perform differently in experiments would speak in favor of the scale. Even allowing that certain structural defects were present in the scale, evidence from the present study suggested that the scale was functioning to a certain extent. Mean cff scores for the normal, process, and reactive groups fell on a continuum of ascending order (Table VIII). Although no differences were found to exist between normals and process, or between process and reactives, significant differences ($p < .05$) were obtained between reactives on one extreme and normals on the other. In addition, when the 10 "most" extreme reactive subjects (i.e. those with the lowest Elgin ratings) were compared with the normals, the differences were even more significant ($p < .02$). Thus, in a sense the scale was able to differentiate between two groups of schizophrenics: one that differed from normals, and one that did not.

Lack of differences in cff between the two schizophrenic groups could be due to the fault of the concepts themselves. Certainly the correlation coefficient of .048 between an individual's Elgin rating and his cff score lends little support to the process-ractive continuum notion, assuming of course that cff itself was not the source of error. But rejection of the process-reactive classification leads us back to the alternative of viewing schizophrenia as a homogeneous entity--already proven an unpromising basis for research. The paucity of results in schizophrenic experimentation to date is felt by some to be due to the very heterogeneity of the concept (26, 108). In addition, widespread clinical observation plus diversified scientific investigation further emphasize the inadequacy of any view of the disease as a homogeneous one.

It is readily conceivable that the lack of cff differences between process and reactive schizophrenics is due to the flicker phenomenon itself. This view would hold that process schizophrenia could be an organic (or even cortical) condition, but with the lesions or structural damage so slight as to evade detection by such a crude technique as cff. The ability of flicker to discriminate cases of mild organicity admittedly has not been well investigated. But to fall back on the excuse of inadequate instrumentation is merely to echo one truism of psychological experimentation generally. Furthermore, cff is relatively speaking one of the more exact techniques available to psychological research. In the present experiment, the instrument proved to be very reliable with only small variations in fusion level from one trial to the next occurring in any particular individual.

Perhaps the most parsimonious statement concerning lack of differences in cff between the two schizophrenic groups is that process schizophrenia is not organic in nature, at least in the cortical sense. Evidence in favor of there being some physiological difference between the two is fairly strong, particularly in those studies having to do with autonomic reactivity to drugs such as epinephrine and mecholyl. However, data purporting to show that schizophrenia is due to a defect of the cortex is less plentiful, and much reference to this possibility must be put down as mere speculation.

Archimedes Spiral Aftereffect

While it is true the spiral aftereffect discriminated organics from all other groups in the present study, the results were somewhat disappointing in light of earlier experiments utilizing the technique. Price and Deabler (107) were able to report that approximately 98 per cent of their organic group was correctly identified by failure to perceive the aftereffect. Subsequent publications indicated that a somewhat lower discrimination index was more typical (34,38). In the present investigation, however, only 45 per cent of the brain damaged group was identifiable through failure to perceive the effect. It is possible that better control of such variables as age and visual acuity contributed to the wide discrepancy between our results and those of previous studies. But the fact that only known organics were used in this study rather than merely cases carrying an official hospital diagnosis of some sort of organic involvement, should if anything, tend to increase the discriminability of the spiral technique. In light of this, the findings of Price and Deabler in particular, become even less understandable. In any event, on the basis of the present investigation, we must conclude that the Archimedes Spiral aftereffect appears to offer little or nothing in the way of a diagnostic aid for the practicing clinician.

SUMMARY

The purpose of this experiment was to obtain evidence about schizophrenia, particularly in relation to possible organic components. Considerable theory and a few experiments suggest that at least certain schizophrenics suffer from a disease that is primarily organic in nature, perhaps of a central nervous system locus. Recent publications have sought to tie this variety of the disorder to the concept of <u>process</u> schizophrenia.

The two perceptual tasks chosen to investigate the problem were critical flicker frequency (cff) and the Archimedes Sprial. The former consisted of a flickerling light whose rate of presentation was increased gradually to the point where the viewer reported perceiving a steady light source. Photic pulse rate at this subjective point is called the critical flicker frequency. The second task was one in which the subject steadily fixated a rotating spiral disc for 30 seconds. On sudden stopping of the spiral, the normal viewer's reaction was to experience the disc as expanding. This phenomenon is referred to as the Archimedes Spiral aftereffect. The selection of these two tasks was based on the considerable amount of evidence that brain damaged individuals show gross defects in the perception of both.

Four groups of 20 subjects each were chosen. All subjects were male veterans under 45 years of age and with normal visual acuity in at least one eye. The organic group consisted of cases of known brain damage, as determined by case history and neurological examination. Twenty surgical cases from a general medical and surgical hospital comprised the normal group. Seventy-six neuropsychiatric patients with hospital diagnoses of schizophrenia, less than one year of current hospitalization, and no history of head injury were rated on the Elgin Prognostic Scale by two independent raters using case history material. A correlation coefficient of .89 was obtained between the two sets of ratings. The 20 patients with the highest Elgin scores were designated as the process group while the 20 individuals with the lowest scores made up the reactive group. All 80 subjects were then given both perceptual tasks.

Relative to cff it was hypothesized: (1) brain damaged individuals will have lower cff thresholds than will normals; (2) process schizophrenics will have lower cff thresholds than will reactives; and (3) reactives will show cff levels not significantly different from normals. Hypothesis One was confirmed ($p < .0001$), but Hypothesis Two and Three were not. In the last case, reactives were significantly higher in cff than normals ($p. < .05$).

Relative to the Archimedes Spiral aftereffect it was hypothesized: (4) brain damaged subjects will perceive the effect less often than normals; and (5) process schizophrenics will not experience the aftereffect as often as either reactives or normals. Hypothesis Four was confirmed while Hypothesis Five was not.

A discussion of the results of this investigation considered the effectiveness of the cff technique in discriminating brain damaged from normal and schizophrenic individuals, and the relative inability of the spiral method to do likewise. Attempts to account for the findings included the possibility that affective components of reactive schizophrenia contribute to the differences between them and other schizophrenics and normals, and the belief that process schizophrenia, while showing some physiological differences from reactive schizophrenia in other investigations, probably is not due to defects of a cortical nature.

REFERENCES

1. Aaronson, B. Age, intelligence, aphasia, and the spiral after-effect in an epileptic population. _J. clin. Psychol_., 1958, _14_, 18-21.

2. Anderson, Camilla. Organic factors predisposing to schizophrenia. _Nerv. Child_., 1952, _10_, 36-42.

3. Ausubel, D. A psychopathological classification of schizophrenia. _Psychiat. Quart_., 1949, _25_, 127-144.

4. __________. Ego development and the personality disorders. New York: Grune and Stratton, 1952.

5. Ax, A. and Colley, W. Temporal acuity of vision, audition, and touch in psychogenic and neurogenic pathology. _J. consult. Psychol_., 1955, _19_,455-461.

6. Bartley, S. H. Vision. New York: Van Nostrand Co.,1941.

7. Bateman, J., and Papez, J. Significance of the thalamus in psychoses with reference to hallucinations and delusions. _J. clin. exp. Psychopath_., 1951, _12_, 89-103.

8. Battersby, W. Critical flicker frequency in patients with cerebral lesions. _Amer. Psychologist._,1950, _5_, 271-2 (abstract).

9. __________. The regional gradient of critical flicker frequency after frontal or occipital lobe injury. _J. exp.Psychol_., 1951, _42_, 59-68.

10. Battersby, W., Bender, M. B., and Teuber, H. L. Effects of total light flux on critical flicker frequency after frontal lobe lesion. _J. exp. Psychol_., 1951, _42_,135-142.

11. Becker, W. C. The relation of severity of thinking disorder to the process-reactive concept of schizophrenia. Unpubl. Ph.D. dissertation, Stanford Univ., 1955.

12. __________. A genetic approach to the interpretation and evaluation of the process-reactive distinction in schizophrenia. _J. abnorm. soc. Psychol_., 1956, _53_, 229-236.

13. Bellak, L. Dementia praecox. New York: Grune and Stratton, 1948.

14. __________. A multiple factor psychosomatic theory of schizophrenia. Psychiat. Quart.,1949, 23,738-755.

15. Bender, Lauretta. Anxiety in disturbed children. In P. Hoch and J. Zubin (eds.). Anxiety. New York: Grune and Stratton, 1950.

16. __________. Childhood schizophrenia. Psychiat. Quart., 1953, 27, 663-687.

17. Bender, M. B., and Kreiger, H. Visual function in perimetrically blind fields. Arch. Neurol. Psychiat., 1951, 65, 72-79.

18. Bender, M. B., and Teuber, H. L. Spatial organization of visual perception following injury to the brain. Arch. Neurol. Psychiat., 1947, 58, 721-739.

19. __________. Spatial organization of visual perception following injury to the brain. Arch. Nurol. Psychiat., 1948, 59, 39-26.

20. __________. Disorders in visual perception following cerebral lesions, J. Psychol., 1949, 28, 223-233.

21. Bender, M. B., Wortis, S., and Cramer, J. Organic mental syndrome with the phenomena of extinction and allesthesia. Arch. Neurol. Psychiat., 1948, 59, 273-291.

22. Bleuler, E. Dementia praecox or the group of schizophrenias. New York: International Universities Press, 1950. [Orig. publ. 1911.]

23. __________. Textbook of psychiatry. New York: Macmillan Co., 1936.

24. __________. The physiogenic and psychogenic in schizophrenia. Am. J. Psychiat., 1930, 10, 203-211.

25. Boring, E. G. Sensation and perception in the history of experimental psychology. New York: D. Appleton-Century, 1942.

26. Brackbill, G. Studies of brain dysfunction in schizophrenia. Psychol. Bull., 1956, 53, 210-226.

27. Brackbill, G. and Fine H. Schizophrenia and central nervous system pathology. J. abnorm. soc. Psychol, 1956, 3,310-313.

28. Bracken, H. V., and Weidman, J. Uber die Wirkung der psycyztrischen Schockbehardlung. auf die Flimmer-Verschemlzungs-Frequenzen. Arch. Psychiat. Nervenkr., 1952, 188, 339-344. [Abstr. from Landis (83).]

29. Bychowski, G. Physiology of schizophrenic thinking. J. nerv. ment. Dis., 1943, 98, 368-386.

30. Cameron, D. E. Early schizophrenia. Am. J. Psychiat., 1938, 95, 567-582.

31. Christian, P., and Schnitz, W. Underschungen von Sehhirnverletzten mit optischen Periodenreizen. Dtsch. Z. Nervenheilk., 1942-43, 154, 81-131. [Abstr. from Landis (83).]

32. Clow, E. The use of a prognostic index of capacity of social adjustment in psychiatric disorders. In P. Hoch and J. Zubin (eds.). Current problems in psychiatric diagnosis. New York: Grune and Stratton, 1953.

33. Darrah, L. Shall we differentiate between schizophrenia and dementia praecox? J. nerv. ment. Dis., 1940, 91, 323-328.

34. Davids, A., Goldenberg, L., and Laufer, M. The relation of the archimedes spiral aftereffect and the trail making test to brain damage in children. J.consult. Psychol., 1957, 21, 429-433.

35. Davis, Pauline, and Davis H. The electroencephalograms of psychotic patients. Am. J. Psychiat, 1939, 95, 1007-1020.

36. Dean, C. A comparison of normal and brain pathology groups on certain aspects of visual perception under conditions of intermittent illumination. Dissert. Abstr., 1955, 15, 1912-13.

37. Devault, S. Physiological responsiveness in reactive and process schizophrenia. Unpubl. Ph.D. dissertation, Michigan State Univ., 1955.

38. Doust, J. W. L., Schneider, R. A., and Harris, G. W. Studies on the physiology of awareness: the effect of rhythmic sensory bombardment on emotions, blood oxygen saturation, and the levels of consciousness. J. ment. Sci., 1952, 98, 640-653.

39. Dunlap, C. B. The pathology of the brain in schizophrenia. In Schizophrenia (dementia praecox). Vol. V. New York: Paul Hoeber, Inc., 1928.

40. Eickoff, Louise. The etiology of schizophrenia in childhood. J. ment. Sci., 1952, 98, 229-234.

41. Enzer, N., Simonsen, E., and Blankstein, S. The reappearance of flicker at high flash frequency in patients with brain pathology and in normal subjects. J. lab. clin. Med., 1944, 29, 63-73.

42. Fenichel, O. The psychoanalytic theory of neuroses. New York: Norton, 1945.

43. Freeman, E., and Josey, W. Quantitative visual index to memory impairment. Arch. Neurol. Psychiat., 1949, 62, 794-796.

44. Funkenstein, D., Greenblatt, M., and Solomon, H. Autonomic nervous system changes following electric shock treatment. J. nerv. ment. Dis., 1948, 108, 409-422.

45. __________. Psychophysiological study of mentally ill patients. Part I. Am. J. Psychiat., 1949, 106, 16-28.

47. __________. An autonomic nervous system test of prognostic significance in relation to electroschock treatment. Psychosom. Med., 1952, 14, 347-362.

48. Gallese, A. J. Spiral aftereffect as a test of organic brain damage. J. clin. Psychol., 1956, 12, 254-59.

49. Geocaris, K., and Kociker, J. Blood pressure responses of chronic schizophrenic patients to epinephrine and mecholyl. Am. J. Psychiat., 1956, 112,808-813.

50. Gibson, J. J. Visual perception of objective motion and subjective movement. Psy. Review, 1954,61,304-314.

51. Granger, G. W. Personality and visual perception. J. ment. Sci., 1953, 99, 8-43.

52. Gravely, A. M. An investigation of perceptual tests as measurements of temperment. Unpubl. Ph.D. dissertation, Univ. of London, 1950, 1-235. [Abstr. from Landis (83).]

53. Halstead, W. C. A power factor (P) in general intelligence: the effect of brain injuries. J. Psychol., 1945, 20, 57-64.

54. Halstead, W. C. Brain and intelligence. Chicago: Univ. of Chicago Press, 1947.

55. Hecht, S., and Verrijp, C. Intermittent stimulation by light. IV. A theoretical interpretation of the quantitative data of flicker. J. gen. Physiol., 1933, 17, 269-282.

56. Hirschstein, R. The significance of characteristic autonomic nervous system responses in the adjustment, change and outcome in schizophrenia. J. nerv. ment. Dis., 1955, 122, 254-262.

57. Hoch, T. A. Acute psychoses with symptoms resembling dementia praecox. Am. J. Psychiat., 1921, 78, 364-372.

58. Hoskins, R. G. The biology of schizophrenia. New York: Norton and Company, 1946.

59. Hunt, R., and Appel, K. Prognosis in psychoses lying midway between schizophrenia and the mania depressive psychoses. Am. J. Psychiat., 1936, 93,313-39.

60. Irvine, R. P. CFF for paretics and schizophrenics. J. abn. soc. Psychol., 1954, 49, 87-88.

61. Jones, L. W. Individual differences in mental inertia. J. Nat. Inst. Indust. Psychol, 1929, 4, 282-294. [Abstr. from Landis (83).]

62. __________. Temperment and threshold for flicker. Brit. J. Psychol., 1929, 29, 422-426.

63. Jung, C. G. The psychology of dementia praecox. Nerv. and Ment. Dis. Monographs, 1944.

64. Kallmann, F. The genetics of schizophrenia. New York: J. J. Augustin, 1938.

65. __________. Modern concepts of genetics in relation to mental health and abnormal personality development. Psychiat. Quart., 1947, 21, 534-553.

66. Kant, K. Differential diagnosis of schizophrenia in the light of concepts of personality stratification. Am. J. Psychiat., 1940, 97, 342-357.

67. Kant, O. Problem of psychogenic precipitation in schizophrenia. Psychiat. Quart., 1942, 16,341-50.

68. Kantor, R., Wallner, J. M. and Winder, C. L. Process and reactive schizophrenia. J. Consult.Psychol., 1953, 17, 157-162.

69. Kennard, Margaret, and Levy, S. The meaning of abnormal electroencephalograms in schizophrenia. J. nerv. ment. Dis., 1952, 116, 413-423.

70. King, G. F. Differential autonomic responsiveness in the process-reactive classification of schizophrenia. J. abn. soc. Psychol. [in press].

71. King, H. E., and Clausen, J. Ch. on psychophysiology in F. Mettler (ed.) Psychosurgical problems. (Second Graystone project). New York: Blakiston Co., 1952.

72. Klebanoff, S., Singer, J., and Wilensky, H. Psychological consequences of brain lesions and ablations. Psychol. Bull., 1954, 51, 1-41.

73. Klein, G., and Krech, D. Cortical conductivity in brain injured. J. Pers., 1952, 21, 118-148.

74. Klein, G., and Schlesinger, H. Perceptual attitudes of "form-boundedness" and "form-lability" in Rorschach responses. Amer. Psychologist., 1950, 5, 321. (Abstr.)

76. Kohler, W., and Fishback, J. The destruction of the Muller-Lyer illusion in repeated trials: an examination of two theories. J. exp. Psychol., 1950, 40, 267-281.

77. Kretschmer, E. Physique and character. New York: Harcourt Brace, 1925.

78. Krugman, E. Flicker fusion frequency as a function of anxiety reaction: an explorative study. Psychosom. Med., 1947, 9, 267-272.

79. Landis, C. Experimental methods in psychopathology. Ment. Hygiene, 1949, 33, 96-107.

80. __________. Psychology. Ch. in F. Mettler (ed). Selective partial ablation of the frontal cortex. New York: Hoeber, 1949.

81. __________. Something about flicker-fusion. Scientific Mon., 1951, 73, 308-314.

82. __________. Remarks on psychological findings attendant on psychosurgery. In The biology of mental health and disease: A symposium. New York: Paul Hoeber, 1952.

83. Landis, C. An annotated bibliography of flicker fusion phenomena. Ann Arbor: Armed Forces National Research Council, 1953. Pp. 130.

84. Landis, C., and Clausen, J. Changes in sensory and motor performances induced by active psychiatric treatment. J. Psychol., 1955, 40, 305.

85. Landis, C., and Zubin, J. Discussion of psychological investigations. In F. Mettler (ed.) Psychosurgical problems. New York: Blakiston, 1952.

86. Langfeldt, G. Prognosis in schizophrenia and the factors influencing the course of the disease. Acta. Psychiatrica and Neurologia Supplementum, 1937, 13, 218. [Abstr. from Devault (37).]

87. __________. The diagnosis of schizophrenia. Am. J. Psychiat., 1951, 108, 123-125.

88. Leiberman, D., Jlenig, H., and Hacker, M. The metrazol flicker threshold in psychiatric patients. EEG clin, neurophysiol., 1954, 6, 9-18.

89. Major, S. Schizophrenia: a diencephalic syndrome. Psychiat. Quart., 1947, 23, 83-92.

90. Malamud, W., and Render, N. Course and prognosis in schizophrenia. Am. J. Psychiat., 1935, 95, 1039-1057.

91. Malmo, R., and Shagass, C. Physiologic studies of reaction to stress in anxiety and early schizophrenia. Psychosom. Med., 1949, 11, 9-24.

92. Meadow, A. and Funkenstein, D. The relationship of abstract thinking to the autonomic nervous system in schizophrenia. In P. Hoch and J. Zubin (eds). Relation of psychological tests to psychiatry. New York: Grune and Stratton, 1952.

93. Meadow, A., Greenblatt, M., Funkenstein, D., and Solomon, H. Relationship between capacity for abstraction in schizophrenia and physiological response to autonomic drugs. J. nerv. ment. Dis., 1953, 118, 332-338.

94. Meduna, L., and McCullough, W. The modern concept of schizophrenia. Med. clin. N. Amer., 1945, Chicago number, 147-164. [Abstr. from Becker (11).]

95. Meyer, A. Constructive formulation of schizophrenia. Am. J. Psychiat., 1921, 78, 355-364.

96. Miles, P. W. Flicker fusion fields. IV. Neuro opthalmic leisions. Proc. Asso. Res. Ophthal., 1950, 19, 51-56.

97. Miles, P. W. Testing visual fields by flicker fusion. Arch. Naurol. Psychiat., 1951, 65, 39-47.

98. Muller, M. Prognosis, schizophrenic diseases. Fortschr. d. Neurol. Psychiat., 1935, 7, 447-474. [Abstr. from Becker (11).]

99. Nielson, J. M. The basic pathology of schizophrenia. J. nerv. ment. Dis., 1948, 107, 340-357.

100. Noyes, A. P. Modern clincial psychiatry. Philadelphia: W. B. Saunders, 1953.

101. Ogle, K. N. Researches in binocular vision. Philadelphia: W. B. Saunders, 1950.

102. Ostrow, M. Flickered light as a provocative test in EEG. EEG Clin. Neurophysiol., 1949, 1, 245.

103. Page, H., Rakita, G., Kaplan, H., and Smith, N. B. Another application of the spiral aftereffect in the determination of brain damage. J. consult. Psychol., 1957, 21, 89-91.

104. Parsons, O., and Miller, P. Flicker fusion thresholds in multiple sclerosis. Arch. Neurol. Psychiat., 1957, 77, 223-225.

105. Pfister, H. Disturbances in the autonomic nervous system in schizophrenia and their relations to insulin, cardiazol, and sleep treatments. Am. J. Psychiat. Suppl., 1938, 109, 94-118.

106. Phillips, G. Perception of flicker in lesions of the visual pathways. Brain., 1933, 56, 464-478.

107. Price, A., and Deabler, H. Diagnosis of organicity by means of spiral aftereffect. J. consult. Psychol., 1955, 19, 229-302.

108. Rabin, A. I. and King, G. F. Psychological studies in schizophrenia. In L. Bellak (ed.) Schizophrenia. New York: Logos Press, 1958.

109. Ricciuti, H. A comparison of critical flicker frequency in psychotics, psychoneurotics, and normals. Amer. Psychologist., 1948, 3, 276.

110. Ricciuti, H. A comparison of critical flicker frequency in psychotics, psychoneurotics, and normals. Fordham Univ. Dissertations, 1949, 16, 150-154. [Abstr. from Landis (83).]

111. Roizin, D., Moriarity, J., and Weill, A. Schizophrenic reaction syndrome in course of acute demyelinization of central nervous system. Arch. Neurol. Psychiat., 1945, 54, 202-211.

112. Ross, A., and Reitan, R. Intellectual and affective functions in multiple sclerosis: a quantitative study. Arch. Neurol. and Psychiat., 1955, 73, 663-677.

113. Ross, R. Contribution to the theory of retinal reaction to intermittent stimuli. Psychol. Bull., 1935, 32, 712 (Abstract).

114. Saucer, R. T. Processes of motion perception. Science, 1954, 120, 806-807.

115. Saucer, R. T. and Deabler, H. Perception of apparent motion in organics and schizophrenics. J.consult. Psychol., 1956, 20, 385-389.

116. Schofield, W., Hathaway, S., Hastings, D., and Bell, Dorothy. Prognostic factors in schizophrenia. J. consult. Psychol., 1954, 18, 155-166.

117. Shapiro, M. B. Experimental studies of perceptual anomaly. J. ment. Sci., 1953, 99, 304-409.

118. __________. A preliminary investigation of the effects of continuous stimulation of the perception of "apparent motion." Brit. J. Psychol., 1954, 45, 58-67.

119. Siegel, S. Nonparametric statistics. New York: McGraw-Hill Book Co., 1956.

120. Simonsen, E., Enzer, N., and Blankstein, S. S. Investigations of the state of the central nervous system by means of fusion frequency of flicker. Influence of age, fatigue, and cerebral lesions. Am. J. Physiol., 1941, 133, 449.

121. Standlee, L. The Archimedes negative after effect as an indication of memory impairment. J. consult. Psychol., 1953, 17, 317.

122. Stotsky, B. A. A comparison of remitting and non-remitting schizophrenics on psychological tests. J. abn. soc. Psychol., 1952, 47, 489-496.

123. Strecker, E., Ebaugh, F., and Ewalt, J. Practical clinical psychiatry. Philadelphia: The Blakiston Co., 1951.

124. Strecker, E., and Willey, C. Prognosis in schizophrenia. In Schizophrenia (dementia praecox). Vol. V. New York: Paul Hoeber, Inc., 1928.

125. Sullivan, H. S. Schizophrenia: its conservative and malignant features. Am. J. Psychiat., 1924, 81, 77-91.

126. __________. Tentative criteria of malignancy in schizophrenia. Am. J. Psychiat., 1928, 84,759-787.

127. __________. Conceptions of modern psychiatry. Washington, D.C.: The Wm. Alanson White Foundation, 1947.

128. Teuber, H. L. Some observations on the organization of higher functions after penetrating brain injury in man. In The biology of mental health and disease: A symposium. New York: Paul Hoeber, Inc., 1952.

129. __________. Physiological psychology. Annual Rev. Psychol, 1955, 6, 267-296.

130. Teuber, H., Battersby, W., and Bender, M. B. Effects of cerebral lesions on intellectual functioning in man. Fed. Proc., 1952, 11, 161.

131. Teuber, H., and Bender, M. B. Changes in visual perception of flicker, apparent motion, and real motion after cerebral lesion. Amer. Psychol., 1948, 3, 246-247.

132. __________. Critical flicker frequency in defective fields of vision. Fed. Proc., 1948, 3, 123-124.

133. __________. Alterations in pattern vision following trauma of occipital lobes in man. J. gen. Psychol. 1949, 40, 37-57.

134. Ulett, G., and Gleser, G. The effect of experimental stress on photocally activated EEG. Science, 1952, 115, 678-682.

135. Ulett, G., Trufant, S., and Ronzoni, E. The EEG with photic stimulation in psychiatric patients receiving ACTH. EEG clin. Neurophysiol., 1951, 3, 79-81.

136. Walker, Helen M., and Lev, J. Statistical inference. New York: Holt and Co., 1953.

137. Werner, H., and Thuma, B. Critical flicker-frequency in children with brain injury. Am. J. Psychol., 1942, 55, 394-399.

138. Wertheimer, M., and Jackson, C. W. Figural after effects, "brain modifiability" and schizophrenia: a further study. J. gen. Psychol., 1957, 57, 45-54.

139. Wertheimer, M., and Wertheimer, Nancy. A metabolic interpretation of the individual differences in figural after effects. Psychol.Rev., 1954, 61, 279-280.

140. Wiersma, E. Die Sekiudarfunktion bei Psychosen. J. Psychol. Neurol. Lpz., 1906, 8, 1-24. [Abstr. from Landis (83).]

141. Williams, M. Psychophysiological responsiveness to psychological stress in early chronic schizophrenic reactions. Psychosom. Med., 1953, 15, 456-462.

142. Winkelman, N., and Book, M. Observations on the histopathology of schizophrenia. I. The cortex. Am. J. Psychiat., 1948, 105, 889-896.

143. Wittman, Mary and Steinberg, D. A study of prodromal factors in mental illness with special reference to schizophrenia. Am. J. Psychiat., 1943, 100, 811-816.

144. Wittman, Phyllis. Scale for measuring prognosis in schizophrenia. Elgin State Hosp. papers, 1941, 4, 20-33.

145. __________. Follow up on the Elgin prognostic scale results. Ill. Psychiat. J., 144, 4, 56-59.

146. __________. Diagnostic and prognostic significance of the shut-in personality type as a prodromal factor in schizophrenia. J. clin. Psychol., 1948, 4, 211-214.

147. Wittman, Phyllis, Sheldon, W. H., and Katz, C. A study of the relationship between constitutional variations and fundamental behavior reactions. J. nerv. ment. Dis., 1948, 108, 470-478.

148. Yates, A. The validity of some psychological tests of brain damage. Psychol. Bull., 1954, 51, 359-379.

349. Young, K. M. "Critical flicker frequency." In F. Mettler (ed.) Selective partial ablation of the frontal cortex. New York: Paul Hoeber, Inc., 1949.

APPENDIX

BECKER'S REVISION OF THE ELGIN PROGNOSTIC SCALE

Items A through O are rated on the basis of case history data. Items P through T are rated on the basis of the presenting clinical symptoms. Item O was not used, however, in the present study because the necessary information was not easily ascertainable.

A. Defects of interest versus definite display of interest.

0. Keen ambitious interest in some of the following: home, family, friends, work, sports, arts, pets, gardening, social activities, music, dramatics.

2. Moderate degree in several activities including social gatherings, sports, music, opposite sex,etc.

4. Mild interest in a few things such as job, family, quiet social gatherings. The interest is barely sustaining.

6. Withdrawn and indifferent toward life interests of average individual. No deep interests of any sort.

B. Insidious versus acute onset.

0. Development over a period of 0-1 months with sudden dramatic divorcement from more or less commonplace living.

1. Development over a period of 2-4 months with marked personality changes from relatively commonplace living.

2. Development over a period of 5-7 months with moderate personality changes. May be some accenting of previous trends, but changes also.

3. Changes have taken place over a period of 8-12 months with noticeable personality modifications, but primarily an accenting of existing trends.

4. Slow development of symptoms, but possible to detect personality changes in 2 years prior to onset.

6. Very slow development of symptoms so that final disorder appears as an exaggeration of already strongly accentuated personality traits. Indications even prior to adolescence.

C. Shut-in personality.

General: The psychotic condition is simply an exaggeration of the peculiar type of personality shown all through childhood. Stormy childhood often with over-protection and anxiety, a difficult adolescence characterized by inability to get along with and mix with other children. Constitutional apparently rather than the product of specific environment.

5. Very much as described above.

3. Moderately the picture described above.

1. Only mildly this way, but some resemblance to pattern.

0. Apparently normal childhood, little evidence of shyness, unusual difficulty, or else unusual behavior is attributable to environmental factors.

D. Schizothymic versus syntonic personality.

0. Very sociable, fond of people and social gatherings; many friends, active in groups and sports, participates in life of his community.

2. Moderately sociable, likes people and social gatherings, but doesn't go far out of way to meet people.

3. Midly shy, mildly sociable. Will interact when the situation presents itself. Prefers association in family group as a rule.

4. Moderatly shy, retiring, etc. More concerned with ideas than people.

6. Very seclusive, shy, retiring, mixes little with others. Few if any close friends. Interested in ideas rather than people. Passive, an onlooker at life rather than an active participant. Poor "Bite on life."

E. Range of interests.

0. Wide and varied interests, keen bite on life, and its opportunities, forward and interested in making adaptation to daily life in many spheres.

2. Moderate breadth of interest, interested in making adaptations to daily life, but does not go out of way to seek new opportunities.

4. Moderate restriction of interests. Narrow goals, but some detectable variety of interests within a narrow orientation.

6. Inadequate interest in varied problems of life, rigid, narrow goals or interests, circumscribed activities because of the narrow range of interests.

F. Constitutional bias.

0. A healthy, strong energetic physical and mental make up that makes the interplay between heredity and environmental influence during childhood a satisfactory one.

2. Suggestions of defects in physical and mental stamina occasionally observed. Not at all marked. Perhaps proness to repeated illnesses in childhood.

4. Regarded from early childhood as different, queer, or odd; perhaps associated with some real defect or handicap-physical, such as deformity, or speech defect, but more often only an imaginary defect of personality.

G. Low energy tone.

0. Very strong drive, keen active and alert interest and ambition shown in school, social and work spheres. Good grasp on life, liked life and had energy enough to enjoy it. Outgoing and adequate in meeting life.

2. Moderately adequate drive, interest, and energy as described above.

4. Moderately inadequate energy tone. Tends toward submissive, passive ractions. Shows some potential to face life's problems, but would rather avoid them than expend the necessary energy.

6. Submissive, inadequate passive reactions, weak grasp on life, does not go out to meet life's problems, does not participate actively but passively; accepts his lot without having the energy to help himself.

H. Asthenic build.

0. Large, barrel-shaped trunk, with relatively short legs, and arms; shield-shaped face, short broad head upon a thick neck, set well down between shoulders.

2. Athletic build. Balanced weight, good musculature, head shape, etc., intermediate to 0 and 4.

4. Long, slender extremities with relatively small, narrow trunk. Egg-shaped face; elongated narrow head on a tall, slender neck.

I. Heterosexual contact.

0. Purposefully contacts the other sex, dates frequently, makes successful effort to be attractive in manner, dress, accessories, etc., so as to be popular with girls (boys).

2. Dates when situation affords. Maybe marries,but to have difficulties in compatibility. Wants to interact with other sex, has some techniques but not completely successful.

3. If married, apt to divorce or separate. Generally this is rated as a midpoint between 2 and 4.

4. Moderate lack of heterosexual contact. Tends to avoid dates, dances, but has on occasion participated in same. Might think (he) she would like to marry someday, but little enthusiasm for it.

6. No association with the opposite sex. Never had any dates. Avoides dances and social gatherings which require the intermingling of boys and girls.

J. Marked academic interests versus active interest in sports.

0. An active interest in sports, participates in baseball, basketball, tennis, football, or other sports. A solitary sport such as swimming or golf is not so important unless the patient plays or swims with others rather than self.

1. Moderate interests in sports and other interests.

2. Mild interest in sports, mild interest in study.

3. Moderate interest in study--without other interests.

4. Fond of study, worked diligently at school and excells in this field associated with inadequacy in sports and social field; a grind without the ambition or drive in work and play to equal his achievements as a student.

K. Careless, indifference versus worrying, self-conscious type.

0. Subjectively sensitive, critical of self, preoccupied with own conflicts, but shows little of the extreme, bizarre, unusual, mysterious or socially unacceptable behavior.

2. Some concern and preoccupation with difficulties--a moderate position to 0-4.

4. Withdrawal and disinterest in social surroundings, careless of social requirements, given to daydreaming, and excentricity, dirty, disheveled appearance, profane language, unacceptable habits.

L. Exclusive stubborn traits versus insecurity and inferiority feelings.

0. Timid, lacks self-confidence, feels insecure and inferior. Very sensitive and critical of self; feels certain problems in life but participates and does not accept his lot passively or without regret and struggle.

1. Moderately like 0.

2. Neither timid nor stubborn.

3. Moderately stubborn.

4. Complete withdrawal from surroundings and interest, inadequate in meeting life but stubborn and opinionated, refuses to change, even if suggested, to achieve a more adequate adjustment. Opinionated and egocentric.

M. Toxity of exhaustion.

0. History of illness, disease of exhaustion closely associated with the onset of psychotic symptoms.

1. Illness present, not severe, but related to onset. Less severe exhaustion.

2. Poor health--but not requiring bed.

3. Fair health--a little run down.

4. Excellent health history, health in no sense an etiologic factor in the development of psychosis.

N. Precipitating conditions (situational reaction).

0. A strong relationship between onset of symptoms and situational problems that would require definite and continued effort to adjust satisfactorily; i.e. death, failure, loss, interpersonal strife. The average person would definitely try to flee such a situation rather than attempt to change it.

1. Marked stresses related to onset, but not as severe as 0.

2. Moderate stresses related to onset such as financial problems, interpersonal discord, etc., which would cause considerable worry to the average individual.

3. Mild stresses that the average person would react to in some way but which would not usually lead to a breakdown.

4. Onset of psychotic symptoms not related to any disturbance or difficulty in the patient's situation--or a disturbance of such a trivial nature that it would be ignored or quickly forgotten by the average person.

O. Duration of psychosis.

0. Under 2 months
1. 2-4 months
2. 4-6 months
3. 6-8 months
4. 10-12 months
5. 1-2 years
6. 2-3 years
7. Over 3 years

<u>The following scales are rated from the presenting clinical picture</u>:

P. Inadequate affect versus emotional instability of appropriate affect.

0. Adequate or overly demonstrative affective expression. This includes appropriate expression and manic depressive aspects in which there is a facile display of emotion.

2. Moderately inadequate affect. Tends to be rigid, dull, or slightly inappropriate. Only moderate responsiveness to emotional stimulation.

4. Markedly inadequate, inappropriate, rigid, or dull affect. Emotional life expressed is at odds with behavior or strikingly inappropriate. Little reaction to stimulation of any strength.

Q. Hebephrenic symptoms: extreme indifference, complete divorce between ideas and affect; extreme carelessness in appearance and reaction with untidiness in some cases, silly behavior, often silly laughter without appropriate stimulation.

0. Not as above.
1. Mildly as above.
2. Moderately as above.
3. Markedly as above.
4. Very markedly as above.

R. Ideas of influence: Patient feels that someone or something is directing his actions, thoughts, or speech. Some outside influence forces him to do things even against his own will.

(Rate 0-4 as in scale Q)

S. Physical interpretation of delusions: The patient has certain feelings (possibly hallucinations) that are linked up with definite delusional ideas; for instance, that there is a snake in his stomach, that food passes right through his body, that someone is passing electrical currents through his body, that the food he eats is poisoned, etc.

(Rate 0-4 as in scale Q)

T. A typical symptoms: Manic or depressive feature mixed with the schizophrenic picture. Display of appropriate affect, over-talkative, distractive, facetious, display of interest in other patients, desire to help humanity in general, depression, feeling of sin or guilt, psychomotor retardation, anxiety, crying.

0. Very markedly atypical picture, shows many of the above features with considerable strength of affect.

1. Markedly atypical picture.

2. Moderately atypical picture, less intensity of features shown.

3. Mildly atypical picture, unusual features are minimal or lacking in intensity.

4. Lacking atypical features.