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Auditory attentional deficit in schizophrenia

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AUDITORY ATTENTIONAL DEFICIT
IN SCHIZOPHRENIA

A Thesis
Presented to
the Faculty of the Department of Psychology
The University of the Pacific

In Partial Fulfillment
of the Requirements for the Degree
Master of Arts

by
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This thesis, written and submitted by

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Abstract

Differences in auditory detection performance between schizophrenics and normals were examined in terms of the attentional processes involved. Each of 40 Ss (20 schizophrenics categorized along the dimensions of paranoia, premorbidity, and acuteness; and 20 hospital technical staff) were presented with 30 50-trial blocks of a tone detection task using 6 auditory ensembles consisting of 2 tones apiece separated by varying frequency bands. Tones were masked by white noise and presented in a free-running trial manner. The commonly found decrement in detection performance with normal subjects as the tones in the ensembles become more widely separated was replicated. But the differing frequency separations between the tones in the ensembles also yielded performance differences within the various schizophrenic subclasses (except the chronic/acute subclass), as well as between schizophrenics and normals. These differences can be attributed to the attentional mechanisms of scanning and beam width as there were no cognitive components involved in the experimental task.
Schizophrenics have composed a small but definite subset of society ever since the beginning of recorded history. They have been noted for their bizarre behavior in diverse sources, from the linear B script left by ancient Minoans (Edmonson, 1962), to records left by Medieval monks (Powell, 1963). However, the first efforts to categorize mental problems did not come until the middle of the 19th century when Rousseau described a particular condition characterized by its early onset and progressive deterioration. He called this condition dementia praecox (McGhie, 1969). Bleuler's classic 1911 work (English translation, 1950) changed the classificatory emphasis from the outcome of disorders to the principal symptoms of each; and it was from his work that the earlier term, dementia praecox, came to be replaced by the term schizophrenia. Bleuler observed that progressive mental deterioration was not inevitable in all cases, and therefore that schizophrenia — meaning literally "a splitting off of psychic functions" — better described what he felt to be the basic disease process in this type of mental disturbance. Although schizophrenia has been subdivided and resubdivided into differential classifications from Bleuler's time to our own, his generic title has stayed with us, mainly for want of a better overall term descriptive of the phenomenon of this particular genre of mental illness.

Not all of the early psychiatrists and psychologists spent their research hours in an attempt to classify types of mental problems. Many of these researchers were actively engaged in discovering what each hoped to be the cause of schizophrenia. All of these early theories tended first to look at the overall symptom pattern presented by each patient, and then to try to pull these symptoms together within a single unifying theoretical system, thereby elucidating causatory factors
contributing to the schizophrenic syndrome. Holistic theories of this nature ranged from Alzheimer's (in Dastur, 1959) notion that schizophrenia was characterized by "severe cerebral cortex changes, with disorganization of ganglion cells and extensive glial reactions", to the futuristic, as yet unsubstantiated, hope that a "schizococcus", acting as a psycho-toxic agent, would one day be discovered (Mandell, Segal, Kuczynski, & Knapp, 1972). Other, broader-based holistic theories have seen schizophrenia as a product of multiple factors, and have thus incorporated both somatic and psychogenic factors within their frameworks (Bellak, 1949; Freedman, 1958).

These and many other holistic theories have created nothing but dissention within schizophrenic research because of their failure to discover the one or two unifying causes of this disorder. Solare (1956) probably comes closest to summing up the reason for the failure of holistic theories in his statement: "No single school of thought is capable of producing a complete answer to the problem [of schizophrenia]. It would appear that a modern, global concept of schizophrenia depends upon accepting a principle of multiple causality marked by the interaction of various factors."

Along the lines suggested by Solare, a more promising approach to take in discovering reasons for the behavior manifested by schizophrenics would appear to lie in dividing the schizophrenic's actions into both cognitive and noncognitive processes, and then looking at the specific deficits he demonstrates in each area. This multiple causation approach involves examining several inter-related areas in which schizophrenic deficit may be found. Memory, motivation, learning and attention are
only a few of the possibilities.

Attention appears to be an important component of all basic psychological processes because it is the selective aspect of perception and response. Therefore, it provides the foundation for relationships both with other people and with the world in general. If schizophrenic deficits can be shown to exist in attention, it will strengthen the hypothesis that more specific pathological processes are active in schizophrenia than holistic theories imply.

Three basic theories of selective attention can be found in the current literature. Broadbent's original theory (in Moray, 1969a) is perhaps the best known and is schematized in Figure 1. He hypothesizes that "information enters the [human perceptual] system through a number of different parallel input lines [vision, hearing, and somaesthesis]". These input lines have a distinct neural representation in the brain, allowing messages to be selected on the basis of characteristics such as loudness, pitch and spatial position. A limited capacity channel is found later in the perceptual system which is capable of handling only a small amount of sensory input. Between this limited capacity channel and the initial sensory input lines, Broadbent postulates the existence of a filter with the ability to select sensory information serially from the input lines. This serialized input is then passed on to the limited capacity channel. Input not selected by the filter for immediate attention is held in short term memory where it undergoes rapid decay. The filter is believed to switch from one input line to another upon arrival of new signals on an unoccupied line, upon arrival of contextually highly probable signals, or upon arrival of input crucial to
homeostasis or survival. Switching time from one input line to another is thought to be roughly 0.25 seconds (Moray, 1969b).

Fig. 1: Broadbent's Attentional Model

Treisman's (1964a) model of attention is based on Broadbent's work, but is more explicit as to the precise functioning of the filter. She believes that the filter not only selects sensory input, but that it analyzes this input for its crude physical properties, and, on the basis of this analysis, selects messages and passes them on to the cerebral or motor cortex. Messages not selected in this way are then attenuated. Treisman has also developed the idea of a pattern recognition network (within the cortex) made up of units with varying stimulation thresholds. Thus, the stronger a message is the greater chance it has of firing a unit in the pattern recognition network thus initiating a response. Biological and emotional units have low thresholds, while most sensory and intellectual units have thresholds that can vary depending on the task engaging the individual. Deutsch and Deutsch (1963) felt Treisman's theory was redundant, as the filter and pattern recognition network performed similar tasks, so they developed their own theory which, in essence, is Treisman's theory without the initial filter.
Seen in terms of schizophrenic deficit, breakdown in attentional processes is postulated to occur at the filter level in Broadbent's and Treisman's theories and within the pattern recognition network in Deutsch and Deutsch's hypothesis. In his review article Shakow (1962) states most definitely that schizophrenic deficit is not evident in reflex latencies or sensory thresholds. Thus it would seem the schizophrenic is not disturbed by a malfunction at Broadbent's initial sensory input level. Shakow goes on to say that schizophrenics are unable to select out sensory material relevant for optimal situational responses and that schizophrenics are very susceptible to peripheral influences which keep them from attending to the task at hand. This deficit would appear to indicate some abnormalities within the attentional filter postulated by both Broadbent and Treisman. Apparently this filter in some way loses its capacity to sort relevant sensory input from that which is irrelevant.

Silverman's research (1964) led him to split attention into extensive and selective factors. Extensiveness (also termed scanning) refers to the degree to which stimuli are sampled from the environment, while selectiveness (also termed focussing) involves division of the stimulus field into "salient and irrelevant cues". From his studies Silverman concludes that extremes of the extensive attentional dimension and difficulty in selecting relevant cues "characterize the attention-response disposition of most schizophrenics". Responsibility for these extreme forms of scanning and cue selection probably can be traced to the schizophrenic's over or under active sensory filtering mechanism.

Along the same lines, Wachtel (1967) has defined attention as a "selectivity in perception and cognition". He notes that both the
pattern and the degree of organization of attentional scanning are very important in pathology — which suggests that different classes of schizophrenics might be typified by a certain type of scanning or lack of it. Wachtel also postulates that the perceptual field is restricted to a certain range of incoming stimuli at any one time. He represents attention as a beam of light, the width of which can illuminate only a limited field at any given moment. The width of this beam is synonymous with the ability to focus one’s attention and may be seen as analogous to the information transmitting capacity of Broadbent’s filter and limited capacity channel. However, the beam is mobile and movement of this limited-width beam around the perceptual field is termed scanning.

All three theorists (Shakow, Silverman, and Wachtel), despite slight definitional discrepancies, appear to view the schizophrenic’s attentional deficit as arising from some malfunction within both the hypothetical sensory filter postulated by Broadbent, and the schizophrenic’s attentional scanning mechanism.

Despite the apparent promise of both the attentional filter theory and the scanning deficit theory, it became apparent several years ago that perceptually oriented schizophrenic research, which did not differentiate among the different subclasses of the disorder, yielded widely varying results at best (McGhie, 1969; Silverman, 1964). Cromwell and Dokiecki (1968) have suggested that more coherent results are obtained when the performance of certain subclasses of schizophrenics is examined separately. Shakow (1962) has suggested several overlapping dichotomies for describing these subclasses. His dichotomies include five major dimensions along which schizophrenic disorders can vary. These
include typical/atypical, dementia praecox/schizophrenia, good premorbid/poor premorbid, chronic/acute, and paranoid/nonparanoid. Research has shown that the latter three dichotomies are marked by striking attentional differences within each.

Paranoid schizophrenia is characterized by delusions of persecution, omnipotence or grandeur, ideas of reference (believing oneself a topic of strangers' discussions), a hostile or aggressive attitude, excessive religiosity, and a systematized hypochondriacal state (Shakow, 1962). Of course, not every patient manifests all of the foregoing symptoms. For the purposes of the present study, patients not evidencing a delusional symptom pattern are classified as nonparanoid.

The paranoid-nonparanoid attentional comparisons (McGhie, 1969; Payne, 1961; Silverman, 1964) suggest that nonparanoid schizophrenics underscan the perceptual field. This hyposcanning leads to a lessened stimulus input which contributes to a breakdown in relevant contact and communication with others. On the other hand, the paranoid schizophrenic's highly systematized delusional system causes him to overscan the environment in a flurried search for threatening people or events. This heightened sensitivity leads to the input of more stimuli than could possibly be integrated, thus causing confusion and an even greater sense of impending threat (Silverman, 1964). It would appear that in his efforts to protect himself from the environment, the paranoid schizophrenic is actually adding to the cause of his own fears (Shakow, 1962). His perceptual filter seems to be working "overtime" in scanning from one input line to another.

A second distinction has been made between chronic and acute schizophrenics (McGhie, 1969; Shakow, 1962; Silverman, 1964). This dichotomy
is based on the actual amount of time spent in a mental hospital. Patients with less than two years hospitalization are considered acute, while those with more than six years are considered chronic. It has been hypothesized that the chronic patient becomes less sensitive to his environment as the years pass (McGhie, 1969). Field scanning and utilization of relevant cues are considerably lessened in paranoids and nonparanoids alike as a result of lengthy (e.g., more than two years) hospitalization (Silverman, 1964). These perceptual changes appear to arise from the chronic patient's gradually acquired reductions in initial high scanning rate, reductions reinforced by lessened anxiety and confusion (Cromwell & Dokecki, 1968).

The third distinction has been made between good premorbid and poor premorbid schizophrenics. Good premorbid are those who showed adequate sexual and social adjustment prior to hospitalization, while poor premorbid are those who showed inadequate sexual and social adjustment. Generally, poor premorbid tend to underscan while good premorbid show extensive scanning. Several reviewers (Cromwell & Dokecki, 1968; Shakow, 1962; Silverman, 1964) have indicated that good premorbid are highly anxious individuals. This anxiety, precipitated by the schizophrenic's confusion and uncertainty at the sudden onset of his symptoms, leads to hyperscanning the perceptual field. Both premorbid groups show deficiencies in attentional focusing and are thus unable to separate relevant from irrelevant cues (Shakow, 1962). However, the poor premorbid focuses on too few cues, causing him to perform in a field dependent manner; while the good premorbid attends to all possible cues, without differentiating relevant field dimensions from irrelevant ones (Silverman,
1964). It can be seen, at least in attentional processes, that schizophrenics differ greatly from each other, and that different extremes of the same category often differ in opposite directions from normal control subjects.

Research in support of the differential characterizations of attentional functioning within the subclasses of schizophrenia has been based mostly upon subject tasks involving fairly complex cognitive processes (Cameron, 1939; Chapman & Taylor, 1957; Payne & Caird, 1967; Weckowicz, 1960; Weckowicz & Whitney, 1960). As an example, Chapman and Taylor (1957) presented subjects with four stimulus cards, each with figures on the four corners, and with a pack of response cards again with a figure on each corner. Instructions were to sort the response cards on to the categories indicated by one corner picture of each stimulus card. Pictures in the other three corners were irrelevant. Thus, it was necessary to retain the concept of which corner contained the "cue" picture as well as to be able to visually match one form to another. When confronted with this task, schizophrenics made significantly more card-sorting errors than normals.

In another study, Weckowicz & Whitney (1960) demonstrated an increase of the illusory effect in the Müller-Lyer optical illusion using schizophrenics as subjects. Their results indicate that schizophrenics evidence a reduction in both size and distance constancy. Weckowicz (1960) also found that schizophrenics performed poorly on an embedded figures task. Both the Müller-Lyer illusion and the embedded figures test require that subjects not only visually perceive objects, but also that they demonstrate a perceptual selectivity based on an understanding of
the concepts underlying the task.

Recently the strategy of evaluating attentional processes using stimuli with lesser cognitive context has appeared. Via this strategy, it should be possible to eliminate cognitive factors, such as concept retention, which could easily contaminate studies designed to measure attentional processes.

As an example of this technique, Neale, McIntyre, Fox & Cromwell (1969) found that schizophrenics were clearly unable to pick relevant visual cues out of a tachistoscopic display as accurately as their matched normal controls when the cue stimulus — one specific letter — was embedded in a display of letters. However, when only one letter at a time was flashed on the screen, schizophrenics were just as capable as controls at deciding whether the letter was or was not the cue stimulus. This experiment indicates that schizophrenic deficit is found not in the schizophrenic's ability to perceive visual stimuli, but in his ability to scan the perceptual field rapidly and focus his attention on relevant cues. These results indicate promise for future research that uses psychophysical methodology to assess attentional processes and deficiencies.

It has been fairly well established that schizophrenics evidence attentional deficiencies in visual processes. However sensory input is processed through other than visual channels and it would greatly strengthen attentional deficit theories of schizophrenia if evidence for schizophrenic deficit could be supported by research in other perceptual areas. Audition is also important in contributing to one's impressions of one's surroundings. Thus if an auditory deficit could
be shown to exist in schizophrenia analogous to the visual deficit already postulated, the theory that attentional deficit is an explanatory factor in schizophrenia would be considerably strengthened.

Based on the visual research alluded to above, it is assumed that the schizophrenic's ability to perceive auditory stimuli is comparable to that of normals. Thus, it is principally within auditory focusing and scanning mechanisms that deficits would be expected to exist.

As in vision, two principal attentional components may be considered active in audition, scanning and focusing (or beam width). Several investigators have hypothesized that subjects, when listening for a cue tone, are sensitive to only those tones (other than the cue tone) which fall within a symmetrical band centered about the cue tone (Greenberg & Larkin, 1968; Greenwood, 1961; Swets, Shipley, McKey & Green, 1959; Veniar, 1958a). Beyond this band the probability of tone detection drops sharply. Thus, a critical band could be roughly equated to auditory attentional beam width. Auditory scanning could then involve sweeping the critical band across ranges of frequencies (Greenberg & Larkin, 1968; Greenwood, 1961; Veniar, 1958a). Veniar (1958a) has indicated that a subject must pass through all intervening frequencies, in shifting his attention from one tone to another. This shifting process requires a measurable amount of time. Thus at any one moment, a subject can only be sensitive to those frequencies within a certain limited auditory range. This model implies, in essence, that the "normal" subject continually sweeps the auditory field for cues with a beam of limited width.
Using this approach as the model, the present study is an attempt to examine differences in auditory detection performance between various subclasses of schizophrenics and normals. Subjects will be asked to detect a tone masked by white noise in a free running trial task (Rappaport, Silverman, Hopkins & Hall, 1971). In any one block of trials the tone to be detected will be one of two possible frequencies. The distance between these frequencies will vary from one block to another, from being close enough to be enclosed within a single normal critical band to being widely separated. Differences in detection as a function of frequency difference will then be expected to vary with the category of subject. Keeping in mind attentional characteristics of the various schizophrenic subclasses mentioned previously, the following hypotheses were generated:

Hypothesis I: Patients falling into the acute, good premorbid and paranoid classifications will perform better than normal controls, due to their wide beam attentional focussing mechanisms and high scanning rate.

Hypothesis II: The enhanced performance of the acute/good premorbid/paranoid group will be most evident when the two tones in a given ensemble are far apart.

Hypothesis III: Patients falling into the chronic, poor premorbid and nonparanoid classifications will perform at a poorer level than normals due to their minimal scanning pattern and narrow beam attentional focussing mechanisms.

Hypothesis IV: The decreased performance of the chronic/poor premorbid/nonparanoid group will be most evident when the two tones in a given ensemble are far apart.
Gipson, Hause & Janke (1971) pursued pilot work on this research model using good premorbid paranoid and poor premorbid nonparanoid schizophrenics, and normals as subjects. In that study, the data showed mean differences in favor of the patients regardless of their classification. Any differences in the study are tentative as they did not reach statistical significance due to a large error variance. Both the present design and new equipment will reduce this variance considerably. The fact that the patients did perform better in the 1971 study indicates that a probable difference exists in auditory attentional mechanisms between schizophrenics and normals.

Method

Subjects

Ss were 20 patients diagnosed as schizophrenic from Stockton State Mental Hospital located in Stockton, California. Ten were good premorbid and ten poor premorbid as ascertained from their scores on Part I, items A-F of the Phillips' scale (1953). This prognostic instrument covers areas of recent sexual and social maturity. If some items on the Phillips' scale could not be answered adequately because of a lack of sufficient case history material, the remaining items were rated and an average was computed using only the number of items contributing to the total score.

Each of the premorbid groups was selected in such a way that five were paranoid and five nonparanoid. Paranoid or nonparanoid status was determined from the latest psychiatric code diagnosis for each patient,
as well as from descriptive case history material. This information was obtained from the hospital's medical records. It was also noted whether each patient fell into the chronic or acute category; chronics being those with six years or more of continuous hospitalization and acutes being those with two years or less of continuous hospitalization. There were ten acutes and ten chronics. The type and quantity of medication being taken by each patient was also noted.

In an effort to secure maximum co-operation and performance, patients received $1 at the conclusion of each hour session, as well as candy, soda pop, and cigarettes during each session. Because physical presence at the mental hospital in many ways influences behavior, 20 hospital technical staff were used as controls. The staff also received candy, soda pop, and cigarettes as incentive motivation during sessions. Staff and patients were equated for age and education level. All Ss were male.

Apparatus

The experiment was run in a small, soundproof, carpeted room. Illumination was from the ceiling and was muted, giving the room a semi-darkened appearance. Each S wore a pair of TDH-39 headphones and was seated in a comfortable chair with an LVE Human Response Console in front of him. A red light indicated the start of each trial, a green light was illuminated while each trial was in progress (e.g. for the duration of the white noise), and a white light signaled the end of each trial. S also had two cumulative counters in his display which told him how many trials he had completed and how many he had done correctly.

The relay panel which automatically programmed each trial was set up as indicated in Figure 2. Briefly, the random event generators
Fig. 2: Block diagram of control equipment.
were both set at 50%, the first determining if there was to be a tone and the second determining which tone was to be heard. Thus, on any one trial S had an equal probability of hearing only white noise or white noise and one of two tones. Tones of varying frequencies were equated for perceived loudness given a constant background of white Gaussian noise.

**Procedure**

Price (1968) noted three necessary factors that must be considered in work comparing schizophrenics with normal Ss to insure that performance differences are due to differences in the attentional process and not to extraneous factors. These are: 1) the patient must understand task instructions, 2) the patient must be able to discriminate the dimensional properties of the stimuli used, and 3) the patient must be able to retain information relevant to the stimuli used. It is hoped that in giving each patient detailed instructions and extensive training with the auditory apparatus, that 1, 2, and 3 were controlled for. Such pretraining also served to make the patients more comfortable with both the apparatus and experimenter, raising their level of self-confidence and contributing to their interest in doing well at the task.

In an attempt to maximize performance, all trials were conducted in a free-running manner, as opposed to a forced choice procedure (Rappaport et al., 1971). Each trial consisted of one second of warning light (red), two seconds of white noise (during which a green light was on) with .2 seconds of tone centered within the white noise interval, and two seconds of trial end light (white).
Six ensembles of two tones each served as stimuli. These ensembles consisted of the following pairs of pure tones:

| Ensemble I: | 900-950 cps | Ensemble IV: | 1100-1500 cps |
| Ensemble II: | 900-1000 cps | Ensemble V: | 600-1700 cps |
| Ensemble III: | 1100-1300 cps | Ensemble VI: | 500-1900 cps |

As can be seen, the range between tones varied from 50 to 1400 cycles per second.

In order to equalize the sound pressure level of the high and low tones and white noise, all attenuators were set to 0 db. and the A scale of a General Radio sound pressure level meter was used to adjust the tone and white noise generators until a level of 72 db. was achieved for both the tones and the white noise.

To equalize perceived loudness of the different tones, the white noise attenuator was set at 3 db. down from 72 db. and the low tone attenuator was set at 15 db. down from 72 db., as it had been determined during pretraining sessions with the Ss that this sound to noise ratio yielded tone detections at a rate greater than 60%, but less than 75%. Another person listened through the earphones while E adjusted the high tone attenuator so that the two tones in each ensemble sounded equally loud. Settings for the high tone attenuator obtained by this procedure were 17 db. down from 72 db. for Ensembles I-III, 18 db. down from 72 db. for Ensembles IV and V, and 19 db. down from 72 db. for Ensemble VI.

Initially each patient was read the following material:

"Good morning/afternoon ______ (fill in patient's name). How are you today? My name is Ann and I'd like it very much if you could help me with a project I'm working on. Dr. Gipson who is a doctor here at the hospital is helping me, and we are trying to find out more about basic causes of the problems most patients have."
"The reason we are working on this project is to try to support the idea that one of the causes of patients' problems is the way in which they hear things. Now, I don't think your hearing is any better or any worse than mine, only that the way in which you hear a radio or a television or a person talking may be somewhat different from the way I hear that same radio or television or person. If this idea works out, we will know a little more about patients' problems and how to treat them.

"You are one of a small group of people we would like to have help us with this project. We'll pay you $1 for each time you come here. You're very important because if a new and better treatment program is started on account of this project and others like it, you will have helped to make that new and better program happen. I really hope you'll be able to help us make this project a success.

"Today your hearing will be tested to be sure you are able to do your best for us, and you will have your first practice session. Within the next week, you will have two more practice sessions with the equipment so that you will be familiar with it. Over the next month you will have five more sessions that last about an hour apiece. All you will have to do for both the practice and the regular sessions is to listen through these earphones (indicate) for a tone like this one (present a tone for $ to listen to). You will also hear static through the earphones just like the static on a radio, and your job will be to let me know whenever you hear both the tone and the static. I'm sure you'll be able to do very well at this task."
"Now I'm going to test your hearing. It'll only take a couple of minutes. Do you have any questions about what we are doing before your hearing test?"

Initially all control Ss were individually briefed on the project. Just prior to their first pretraining session, all control Ss were read the following material:

"Your co-operation in participating in this experiment is greatly appreciated. I am attempting to support the theory that schizophrenics evidence concentration problems in their use of hearing. I realize you have had to take time off your ward to help me, and I thank you very much for doing this. Today your hearing will be tested, and you will have your first pre-training session with the apparatus."

Each Ss hearing was tested with an audiometer to insure that he was capable of perceiving all stimuli used. More than a 20 db. deficit at 500, 1000, or 2000 hz was taken as evidence that the S's hearing was not adequate for the purposes of this experiment.

Then all Ss were exposed to three hour-long pretraining sessions, the first one occurring on the same day as the hearing test. Two of the six stimulus combinations were used for the first pretraining session, three stimulus combinations for the second, and four stimulus combinations for the third. By the end of the third session, each S had been exposed to all six stimulus combinations once, and to three of the stimulus combinations twice. The order of presentation of the initial six stimulus combinations was randomized separately for each S. It was then randomly determined for each S which three of the six ensembles he would be exposed to twice. Prior to the first pretraining session each patient was instructed:

"This will be your first training session with this equipment. Now I want you to listen very carefully to what I tell you and be sure to ask me questions if there's something you don't understand."
"See this button here on your right (point to it). Touch that button for me. OK. Now, at the beginning of each trial, that button (point to it again) will turn red. That's the signal for you to get ready. When that same button turns green, you will hear static through the headphones (point to them) for about two seconds. Now put on the headphones and listen so you'll know what the static sounds like (sound static for 2). OK, take off the earphones. Did you hear it? Good. Now, sometimes when you hear the static you will also hear a tone like this one (have 2 put on earphones again, sound tone) played at the same time as the static. Not every trial will have a tone. When you hear a tone like that your job is to press the button you touched a few minutes ago. Do you remember which button it is? Touch it for me again. Good. You must remember to only press the button once. After two seconds, the static will go away and the green light will turn into a white light. This will mean that the trial is over. You can press the button when either the green light or the white light is on.

"Each trial will last about five seconds. That might not seem like very long, but don't worry, you'll have plenty of time. You will get to rest after 50 trials. These two counters (indicate) are to help you keep count of the trials so you can see where you are. The upper one (point) tells you how many trials you've completed and the lower one (point) tells you how many trials you've completed correctly. Now which counter tells you how many trials you've done? Good. And which one tells you how many you have right? Good.

"See the button on your left (point to it)? That button is not to touch. But whenever you make a correct choice that button will flash green. That way you won't have to watch the counters all the time. Remember, since there isn't a tone for every trial there are two ways you can make a correct choice, by pressing the button on a trial that has a tone with the static, and by not pressing the button on a trial that has just static. When 50 trials are over that button on your left (point to it again) will turn white, and that means you get a three minute rest. Before each set of 50 trials starts, the tones you are to listen for will be played for you.

"I think we're ready for a trial run. Are you ready? Do you have any questions? OK, put on the headphones and begin. (Go through ten trials, tell 2 when he makes a correct choice.) Well now, that was really good for the first time. We'll run through the procedure one more time and then you can start on your practice session. Now, point to the button that you press. Good. When do you press that button? Very good. Remember to only press it once when either the green or the white light is on. And how can you keep track of your trials? Good, I think you'll do really well at this. Now we'll start for real. Remember not to talk except during rest periods and try as hard as you can to hear the tone. We'll rest after you've done 50. OK, put on the headphones (hand them to him if he doesn't reach for them) and then we'll begin.
"You did very well. Here's that dollar I promised you. Would you like candy or a cigarette and a coke while we're on our way back to your cottage?"

Prior to the first pretraining session, all staff were instructed:

"You did very well on the hearing test. Trials for your first pretraining session will begin when you are ready. When the button on your right (point) lights up red, it will signal the start of each trial. When it lights up green, you will hear static from the headphones. Sometimes you will also hear a tone, but not every trial will have a tone. When you hear a tone, press the button (point to it again). Be sure to only press the button once. The button may be pressed during either the green or the white light. The trial is over when this same button (point again) turns white. The other button, the one on your left (point) will flash green whenever you have made a correct choice. Trials will continue automatically at the rate of one every five seconds whether or not you press the button. Remember, there are two ways you can be right, by pressing the button on trials with a tone and by not pressing the button on trials that have only static. After every 50 trials, the left hand button will turn white and you will have three minutes to rest. These numbers (indicate) will tell you how many trials you have completed and how many were correct. Before you start each set of trials, the tones you are to listen for will be sounded for you. Do you have any questions?"

Each S was then given five test sessions. Each session consisted of six blocks containing 50 trials each, with a three minute rest between blocks. Each block utilized a different stimulus ensemble, and every block began with its component tones sounded for S with a reduced noise background. Thus, in the context of the five test sessions, each S was exposed to all six ensembles five times. The order of the ensembles was randomized independently for each S.

Prior to the second and third pretraining sessions and all test sessions, patients were instructed:

"Hello, I'm glad to see you again ______ (patient's name). You did so well last time, I hope you do just as well today. Now remember what we do? Which button do you press? Good. When do you press the button? How many times per trial? Good. What is this button on the left for? Very good, you have a really good memory. And what do these counters tell you? Very good. I guess we're ready
to start. Are you sure you don't have any questions? OK, we can begin. Remember not to talk to me except during rest periods. You can rest after 50, OK? (If S cannot answer any of the above questions, he will be reminded of the functioning of the various parts of the apparatus.)

"You did very well, would you like a cigarette and a coke? Here's your dollar for helping me. Let's go back to your cottage now."

For the second and third pretraining sessions and all test sessions, staff were reread their initial instructions as to when the lights come on and which button to press.

For all pretraining and test sessions a preliminary block of trials was run using either Ensemble I or Ensemble II. After the initial tones were sounded with reduced noise background, the block began with a tone sounded every trial. This continued until S got eight in a row correct. Then the apparatus was switched to 50% probability. This initial procedure insured that S was attending and responding as desired (Rappaport et al., 1971). This first block of trials was not included in the analysis, as it deviated from the standardized form of the other blocks.

Stress was placed on personalizing all verbal instructions to all Ss by speaking directly to them rather than reading from a printed sheet. It is hoped that the more personal approach added incentive to the Ss' desire to perform well.

Sessions lasted for an hour. At the completion of each session, each S was assured he was performing well and thanked for his co-operation. In the case of patients, E walked (or drove) them back to their cottage.
Results

The principal dependent measure used in the analyses of variance was $d'$. $d'$ is a measure which is used to indicate signal detection efficiency. Via $d'$, it is possible to "make inferences regarding the sensitivity of peripheral and central sensory mechanisms for detecting and responding to stimuli, independently of such factors as the set, motivation and attitude of $S$" (Rappaport et al., 1971).

$D'$ values for all five trials over each ensemble were obtained from published tables by using both the conditional probability of responding when signal and noise were present (number of hits/total number of trials with tones), and the conditional probability of responding when noise alone was present (number of false positive responses/total number of trials without tones). Other dependent measures were hits (correct detections of a tone), false negatives (indicating that there was no tone when, in fact, there was one), correct negatives (correct detection of noise alone), and false positives (indicating that there was a tone when, in fact, there was none).

Analyses of Variance With One Between-Subject Variable

The original set of 15 split plot analyses of variance were arranged in such a way that there were two within-subject variables, ensembles and the five test sessions nested within each ensemble; and one between-subject variable, subject psychiatric categorization.

With $d'$ as the dependent measure, the subject variable of psychiatric categorization was significant only for the paranoid/nonparanoid/normal group comparison ($F(2, 37) = 3.55, p < .05$). Multiple comparisons via Tukey's HSD test (Kirk, 1968, pp. 88-90, 306), corrected for unequal n, indicated
that paranoids differed from both nonparanoids ($p < .01$) and normals ($p < .05$), but that there were no significant differences between nonparanoid performance and normal performance.

The ensemble variable was significant at the .01 level for all three subject dimensions ($F(5, 185) = 20.41$ for the paranoid/nonparanoid/normal group comparison, 20.98 for the good premorbid/poor premorbid/normal group comparison, and 20.14 for the chronic/acute/normal group comparison). (See Figure 3.) Tukey's HSD test over each subject dimension indicated that significant pairwise differences existed between all six ensembles at the .05 level of significance, with the exception that no differences were found between Ensemble V and Ensemble VI over the good premorbid/poor premorbid/normal subject dimension.

The test session variable was also found to be significant at the .01 level across all three subject dimensions ($F(4, 148) = 4.98$ for the paranoid/nonparanoid/normal group comparison, 5.01 for the good premorbid/poor premorbid/normal group comparison, and 5.05 for the chronic/acute/normal group comparison). (See Figure 4.) Tukey's HSD test indicated differences existed at the .05 level between test sessions 1 and 2, 2 and 3, 2 and 4, 3 and 4, and 4 and 5 for the paranoid/nonparanoid/normal group comparison and for the good premorbid/poor premorbid/normal group comparison. These two subject dimensions also yielded differences at the .01 level between test sessions 1 and 4, 1 and 5, 2 and 5, and 3 and 5. The chronic/acute/normal group comparison differed only slightly, showing no significant difference between test sessions 2 and 3, and a difference at the .05 level between test sessions 3 and 5. Otherwise results from Tukey's test were identical to the results obtained for the other two
Fig. 3: Mean signal detection efficiency scores ($d'$) across ensembles for each subject categorization.
Fig. 4: Mean signal detection efficiency scores across test sessions for each subject categorization.
subject dimensions.

The only significant interaction with d' as the dependent measure was the Ensemble X Test Session interaction (F(20, 740)=1.93, p < .01 for the paranoid/nonparanoid/normal group comparison; F=1.90, p < .05 for both the good premorbid/poor premorbid/normal group comparison and the chronic/acute/normal group comparison). The error rate was determined per hypothesis. (See Figure 5. The graph is identical for all three subject dimension comparisons.) Simple main effects tests for the paranoid/nonparanoid/normal group comparison indicated that the test session variable was significant at the .05 level for Ensemble I (F(4, 888)=2.49), Ensemble II (F=2.68), Ensemble III (F=3.28), Ensemble IV (F=2.72), and Ensemble V (F=3.33). The ensemble variable was significant for test session 3 (F(5, 925)=2.98, p < .05), test session 4 (F=3.36, p < .01), and test session 5 (F=2.77, p < .05). For the good premorbid/poor premorbid/normal group comparison, the test session variable was significant at the .05 level for Ensemble I (F(4, 888)=2.37), Ensemble II (F=2.89), Ensemble III (F=2.69), and Ensemble IV (F=2.71); while the ensemble variable was significant at the .05 level for test session 2 (F(5, 925)=2.54), test session 3 (F=2.44), and test session 4 (F=2.26). Significance was obtained at the .01 level for test session 5 (F=3.41). For the chronic/acute/normal group comparison, the test session variable was significant at the .05 level for Ensemble I (F(4, 888)=2.39), Ensemble II (F=2.66), Ensemble III (F=2.47), and Ensemble V (F=2.70); while the ensemble variable was significant at the .05 level for test session 3 (F(5, 925)=2.98), and test session 4 (F=2.63), and at the .01 level for test session 5 (F=3.04).
Fig. 5: Mean signal detection efficiency scores for each ensemble across test sessions.
With hits as the dependent measure, the subject variable was significant only for the paranoid/nonparanoid/normal group comparison ($F(2, 37) = 3.73, p < .05$). Multiple comparisons via Tukey's HSD test corrected for unequal n indicated that differences existed between paranoids and both nonparanoids and normals at the .05 level of significance. No other significant differences were obtained. The ensemble variable was significant at the .01 level over all three subject dimensions ($F(5, 185) = 65.60$ for the paranoid/nonparanoid/normal group comparison, $66.83$ for the good premorbid/poor premorbid/normal group comparison, and $60.89$ for the chronic/acute/normal group comparison). (See Figure 6.) Tukey's HSD test indicated that significant differences existed at the .05 level between all ensemble pairs over all three subject dimensions with the exception of no significant differences between Ensemble I and Ensemble II for the chronic/acute/normal group comparison, and between Ensemble V and Ensemble VI for both the chronic/acute/normal group comparison and the good premorbid/poor premorbid/normal group comparison. The test session variable was significant only for the paranoid/nonparanoid/normal group comparison ($F(4, 148) = 2.57, p < .05$), and for the good premorbid/poor premorbid/normal group comparison ($F = 2.53, p < .05$). (See Figure 7.) For the former group, Tukey's HSD test indicated (at the .05 level of significance) that differences existed between test sessions 1 and 2, 1 and 5, 3 and 5, and 4 and 5 only. For the latter group, differences at the .05 level were found between test sessions 1 and 2, 1 and 5, 2 and 4, 3 and 4, and 4 and 5.

The Subject X Test Session interaction (See Figure 7) was significant for the paranoid/nonparanoid/normal subject dimension ($F(8, 148) = 2.06,$
Fig. 6: Mean number of correct tone detections across ensembles for each subject categorization.
Fig. 7: Mean number of correct tone detections across test sessions for each subject categorization.
p < .05) and for the good premorbid/poor premorbid/normal subject dimension (F=2.28, p < .05). Simple main effects tests for the former group indicated that the paranoid dimension was significant at the .05 level for test session 2 (F(1, 185)=3.98), test session 4 (F=4.61), and test session 5 (F=4.01); the nonparanoid dimension was significant at the .05 level for test session 1 (F=3.72), and test session 2 (F=4.77). The normal dimension was not significant for any test session. The test session variable was significant at the .05 level for the paranoid group (F(4, 148)=3.33) and the nonparanoid group (F=2.48). Simple main effects tests for the good premorbid/poor premorbid/normal group comparison indicated that the good premorbid dimension was significant at the .05 level for test session 2 (F(1, 185)=4.01), test session 3 (F=4.21), test session 4 (F=4.50), and test session 5 (F=4.37); the poor premorbid dimension was significant at the .05 level for test session 4 (F(1, 185)=3.92), and test session 5 (F=5.96). The normal dimension was not significant for any test session. The test session variable was significant at the .01 level for the good premorbid group (F(4, 148)=3.51), and at the .05 level for the poor premorbid group (F=3.41).

The Ensemble X Test Session interaction was significant over all three subject dimensions (F(20, 740)=2.12, p < .01 for the good premorbid/poor premorbid/normal group comparison; F=1.90, p < .05 for the chronic/acute/normal group comparison, and F=2.10 p < .01 for the paranoid/nonparanoid/normal group comparison). (See Figure 8. The graph is identical for all three subject dimension comparisons.) For the paranoid/nonparanoid/normal group comparison, simple main effects tests indicated that ensembles were significant at the .05 level for test session 2.
Fig. 8: Mean number of correct tone detections for each ensemble across test sessions.
(F(5, 925)=2.96), test session 3 (F=2.43), test session 4 (F=2.73), and test session 5 (F=2.60); while test sessions were significant for Ensemble I (F(4, 888)=2.41, p <.05), Ensemble II (F=3.34, p <.01), Ensemble IV (F=3.14, p <.05), and Ensemble V (F=3.03, p <.05). For the good premorbid/poor premorbid/normal group comparison, simple main effects tests indicated that ensembles were significant at the .05 level for test session 3 (F(5, 925)=3.01), test session 4 (F=2.83), and test session 5 (F=2.68); while test sessions were significant for Ensemble I (F(4, 888)=2.49, p <.05), Ensemble II (F=2.97, p <.05), Ensemble IV (F=3.48, p <.01), and Ensemble V (F=2.81, p <.05). For the chronic/acute/normal group comparison, simple main effects tests indicated that ensembles were significant at the .05 level for test session 3 (F(5, 925)=2.29), test session 4 (F=2.52), and test session 5 (F=2.31); while test sessions were significant at the .05 level for Ensemble I (F(4, 888)=2.50), Ensemble II (F=2.39), and Ensemble IV (F=2.60).

With false negatives as the dependent measure, the subject variable was not significant. The ensemble variable was significant at the .01 level over all three subject dimensions (F(5, 185)=33.54 for the paranoid/nonparanoid/normal group comparison, 36.40 for the good premorbid/poor premorbid/normal group comparison, and 34.91 for the chronic/acute/normal group comparison). (See Figure 9.) Tukey's HSD test indicated that pairwise differences existed at the .05 level between all ensembles except Ensemble I and Ensemble II, Ensemble IV and Ensemble V, and Ensemble V and Ensemble VI for the chronic/acute/normal group comparison. The test session variable was significant only for the paranoid/nonparanoid/normal group comparison (F(4, 148)=2.43, p <.05). (See Figure 10.)
Fig. 9: Mean number of false negative responses across ensembles for each subject categorization.
Tukey's HSD test indicated that differences existed at the .05 level between test sessions 1 and 5, and 3 and 5.

The Subject X Test Session interaction was significant for the paranoid/nonparanoid/normal group comparison ($F(8, 148)=2.18$, $p < .05$), and for the good premorbid/poor premorbid/normal group comparison ($F=2.29$, $p < .05$). (See Figure 10.) For the former group, simple main effects tests indicated that the subject dimension was significant at the .05 level for test session 3 ($F(2, 185)=4.11$), test session 4 ($F=4.63$), and test session 5 ($F=4.87$). The test session variable was significant at the .05 level for the paranoid group ($F(4, 148)=3.39$) and the nonparanoid group ($F=2.94$).

Results for the simple main effects tests over the good premorbid/poor premorbid/normal group comparison yielded a subject dimension significant at the .05 level for test session 1 ($F(2, 185)=4.03$), test session 2 ($F=4.31$), and test session 3 ($F=3.98$). The test session variable was significant at the .05 level for the good premorbid group ($F(4, 148)=3.14$) and the poor premorbid group ($F=2.56$).

The Ensemble X Test Session interaction was significant for the paranoid/nonparanoid/normal group comparison ($F(20, 740)=2.12$, $p < .01$), for the good premorbid/poor premorbid/normal group comparison ($F=2.02$, $p < .05$), and for the chronic/acute/normal group comparison ($F=2.03$, $p < .05$). (See Figure 11. The graph is identical for all three subject dimension comparisons.) Simple main effects for the paranoid/nonparanoid/normal group comparison yielded test sessions significant for Ensemble I ($F(4, 888)=2.96$, $p < .05$), Ensemble II ($F=3.48$, $p < .01$), Ensemble III ($F=2.70$, $p < .05$),
Fig. 10: Mean number of false negative responses across test sessions for each subject categorization.
and Ensemble V (F=2.41, p < .05). Ensembles were found to be significant at the .05 level for test session 3 (F(5, 925)=2.98), test session 4 (F=2.64), and test session 5 (F=2.96). Simple main effects tests for the good premorbid/poor premorbid/normal group comparison yielded test sessions significant at the .01 level for Ensemble I (F(4, 888)=3.51) and Ensemble II (F=3.77), and at the .05 level for Ensemble III (F=2.62) and Ensemble V (F=2.43). Ensembles were found to be significant at the .05 level for test session 3 (F(5, 925)=2.30), test session 4 (F=2.71), and test session 5 (F=2.85). Simple main effects tests for the chronic/acute/normal group comparison yielded similar results with test sessions significant at the .05 level for Ensemble I (F(4, 888)=3.30) and Ensemble II (F=3.16). Ensembles were found to be significant at the .05 level for test session 4 (F(5, 925)=2.97) and test session 5 (F=2.81).

Neither correct negatives nor false positives yielded any significant differences for main effects or interactions.

In an effort to explore further possibilities, the 15 analyses were reanalyzed reducing the subject variable to two levels — pathology and non-pathology. No new main effects or interactions of any interest eventuated from these analyses.

Analyses of Variance With Two Between-Subject Variables

An optimal arrangement to utilize for data analysis in this experiment would have been tripartite, with three between-subject variables: paranoia, premorbidity, and acuteness. However, due to a small patient population at Stockton State Hospital, even a bipartite arrangement, with
Fig. 11: Mean number of false negative responses for each ensemble across test sessions.
subjects categorized over two dimensions, was difficult to obtain, ne-
cessitating a loss of some subjects.

A bipartite analysis was achieved in the following way: Premorbi-
dity was divided into paranoid and nonparanoid levels, paranoia was divi-
ded into acute and chronic levels, and premorbidity was also divided into
acute and chronic levels. These three new groupings were examined over
the ensemble variable only. Unfortunately, the test session variable had
to be collapsed within the ensemble variable for lack of an appropriate
computer program. All Ss were retained in the premorbidity/paranoia analyses.
However, due to non-proportional, unequal n's for the paranoia/acuteness
and for the premorbidity/acuteness analyses, four subjects were randomly
dropped from each to achieve a situation where all levels of n were equal
to four. Two significant Subject X Ensemble interactions (one for $d'$
and one for false negative responses) were found via this procedure.

The significant Subject X Ensemble interaction for $d'$ was found
within the context of a premorbidity/acuteness analysis ($F(5, 60)=2.60,$
p < .05). (See Figure 12.) Simple main effects tests indicated that the
good premorbid dimension was significant at the .05 level for En-
semble III ($F(1, 96)=4.01$), Ensemble IV ($F=4.33$), Ensemble V ($F=3.97$),
and Ensemble VI ($F=4.52$); and that the poor premorbid dimension was sig-
nificant at the .05 level for Ensemble I ($F=6.11$), Ensemble II ($F=4.19$),
Ensemble III ($F=4.06$), and Ensemble VI ($F=5.39$). Ensembles were signi-
ficant at the .05 level for the good premorbid group ($F(5, 80)=3.01$), and
at the .01 level for the poor premorbid group ($F=4.86$).

The significant Subject X Ensemble interaction for false negatives
was found within the context of a premorbidity/paranoia analysis ($F(5, 80)=$
Fig. 12: Mean signal detection efficiency scores across ensembles for good and poor premorbid.
2.48, p < .05). (See Figure 13.) Simple main effects tests indicated that the good premorbid dimension was significant at the .05 level for Ensemble II (F(1, 96) = 4.04) and Ensemble III (F = 4.17); and that the poor premorbid dimension was significant at the .05 level for Ensemble II (F = 5.14), Ensemble III (F = 6.11), and Ensemble V (F = 5.30), and Ensemble VI (F = 4.41). Ensembles were significant at the .05 level for the good premorbid group (F(5, 80) = 3.81) and at the .01 level for the poor premorbid group (F = 2.46).

**Multiple Regression Analyses**

For the last set of analyses performed on the data, d' values for each subject were used in the following way: As the Subject X Ensemble interaction was of principal theoretical interest, a slope value indicating the relative change in d' from Ensemble I to Ensemble VI was calculated for each subject. This slope value was then the dependent measure used in a multiple regression analysis which was run on the data.

A multiple regression analysis was chosen in order to ascertain the magnitude of a possible relationship of the derived slope values with the independent variables used in this experiment. In addition to premorbidity, paranoia, and acuteness; age, education level, and most particularly, amount of phenothiazines, stimulants, and depressants served as the independent variables for this analysis (Rappaport et al., 1971).

These independent variables were coded in the following way for the analysis: Premorbidity was measured via the Phillips' scale with scores from 0 - 3 indicating good premorbid adjustment and scores from 4 - 6 indicating poor premorbid adjustment. The paranoid dimension received
Fig. 13: Mean number of false negative responses across ensembles for good and poor premorbids.
a numerical value of 2, while the nonparanoid dimension received a value of 1. Acuteness was measured in terms of months spent in the hospital. Patients with scores from 0 - 24 (months) were considered acute, while those with scores from 25 on up were considered chronic. The age variable simply represented each subject's age in years, just as the education level represented the number of years of school completed. The three medication variables represented the amount of that particular type of medication, measured in mg, taken by each subject each day of the experiment.

This analysis was performed using the Burroughs Assist computer program package. In addition to a product moment multiple correlation (multiple $R$) of $d'$ with the independent variables, the program also yielded a "corrected" $R$ based on expected shrinkage in $R$ should the analysis be run again with the same sample size, an $F$ test for significance of regression, a partial correlation between the dependent variable and each of the independent variables, and a breakdown of explained versus unexplained variance within the dependent variable.

The Assist analysis was run twice, once using all 40 subjects and once using patients only.

Where data from all 40 subjects was utilized, the multiple $R$ was .52, with the corrected $R$ equal to .23. The $F$ test for significance of regression failed to reach the $p < .05$ level ($F(8, 31)=1.41, p < .25$). Out of a total variance of 41.31 in the dependent variable, 11.04 represents explained variation. Thus roughly 1/4 of the variance in the dependent variable was accounted for by the various independent measures used in this analysis.
The partial correlations between slope and the independent variables were strongest for premorbid adjustment (-.3319), acuteness (.2915), amount of phenothiazine medication (.3433), and amount of depressant medication (-.2183).

As the majority of slope scores in this experiment were negative, this instance shall be considered first. The closer to zero a slope value is, the less tilt there is to the line. Hence, a low slope value indicates fairly even performance across ensembles with little deterioration as the tones in the ensembles draw farther apart. Thus, a higher slope value in a negative direction is indicative of performance that drops off more rapidly as the tones in the ensembles draw farther apart. A positive correlation between negative slope values and any one of the independent variables would indicate that the higher the score for the independent measure, the higher the slope value and the more deterioration is evidenced in performance across ensembles. A negative correlation between negative slope scores and any one of the independent variables would indicate that the higher the score for the independent measure, the lower the slope value and the more even the performance across ensembles.

All but seven subjects (five patients and two staff) evidenced negative slope values ranging from -.06 to -.38, with 60% of these values greater than -1.5. For the patients with positive slope values, the range was from .03 to .32. This latter group arranged itself in such a way that three were good premorbid, two were poor premorbid, two were paranoid, three were nonparanoid, two were acute, and three were chronic. The values for the two staff members with positive slope scores were
.18 and .35.

For the seven subjects whose slope values were positive: The higher the slope value, the greater the increase in performance over ensembles. As can be seen, none of the positive slope scores approach the magnitude of the negative slope scores. A positive correlation between positive slope values and any one of the independent variables would indicate that the higher the score for the independent measure, the higher the slope value and the more increase evidenced in performance across ensembles. A negative correlation between positive slope values and any one of the independent variables would indicate that the higher the score for the independent measure, the lower the slope value and the more even the performance across ensembles.

The Assist program retained information regarding the sign of the slope value for each subject. Hence, the partial correlations are overall, taking into consideration the relationship between each independent variable and the positive or negative slope value paired with it.

The negative partial correlation between slope and premorbid adjustment indicates that the higher the score for premorbid adjustment, the higher the slope value was in a negative direction (and the poorer the performance across ensembles). As premorbid adjustment was derived from the Phillips' scale (previously mentioned), it appears that decreasing performance across ensembles is correlated with a poor premorbid history. Likewise, the negative partial correlation between amount of depressant medication and slope indicates that the higher the amount of depressant medication taken, the more a subject's performance deteriorated across ensembles.
The positive correlation between acuteness and slope indicates that as time spent in the hospital increases, the subject's slope value also increases in a positive direction, indicating more even performance across ensembles with increasing chronicity. The positive correlation between amount of phenothiazines and slope indicates that as phenothiazine dosage increases, slope also increases in a positive direction, indicating more even task performance across ensembles with increased phenothiazine dosage.

For the Assist analysis using only the data for the patients, the multiple R value was .71, with the corrected R equal to .37. Although these correlation coefficients were considerably higher than when all Ss were included in the analysis, the F test for significance of regression still failed (F(8, 11)=1.37, p > .25) to reach significance at the .05 level of probability. Out of a total variance of 25.95 in the dependent variable, 12.93 or roughly $\frac{1}{2}$ was explained, or accounted for, by the various independent measures used in the analysis. This is roughly 25% more variance than was accounted for in the first Assist analysis.

The partial correlations were highest again for premorbidity (-.4684), acuteness (.3051), amount of phenothiazines (.2517), and amount of depressants other than phenothiazines (-.2359). As can be seen, these partial correlations are all somewhat stronger than those found when all subjects were included in the analysis. Once again negative correlations were found between slope and premorbidity and between slope and amount of depressants. Positive correlations were also found again between slope and acuteness, and between slope and amount of phenothiazines. Thus the interpretations of these correlations previously mentioned remained
stable for both sets of analyses.

Discussion

Although the four hypotheses forwarded in the introduction were not totally substantiated by the data, reasons for their failure appear to lie more with procedural and design difficulties than with deficiencies in the attentional theories underlying the study. Hypothesis I (i.e. that paranoid, good premorbid, and acute patients should perform better than their normal controls due to wide beam attentional focussing mechanisms and a high scanning rate) and Hypothesis III (i.e. that nonparanoid, poor premorbid, and chronic patients should perform at a poorer level than their normal controls due to a minimal scanning pattern and a narrow beam attentional focussing mechanism) receive some support, as can be seen from the text and references to graphs which follow; however, Hypothesis II (i.e. that the enhanced performance of the paranoid, good premorbid, and acute patients would be most evident when the tones in an auditory ensemble were far apart) and Hypothesis IV (i.e. that the decreased performance of the nonparanoid, poor premorbid and chronic patients would be most evident when the tones in an auditory ensemble were far apart) remain unsupported with the analytic techniques employed.

Analyses of Variance With One Between-Subject Variable

For the original set of 15 analyses, when the subject variable was significant, the dimension involved was the paranoid/nonparanoid/normal subject comparison. This indicates that classification along the paranoid/nonparanoid dimension differentiated patients from one another and
from normals more effectively than classification along either of the other two subject dimensions used in this study. The fact that this paranoid/nonparanoid/normal split was significant when d' was the dependent variable is especially important, as d' is a function of all four other dependent variables (i.e. hits, false negatives, correct negatives, and false positives), and has important psychological significance.

The multiple comparison tests indicated that the significance in the subject variable was due principally to differences between paranoids and the other two subject groups, indicating that the paranoid group consistently maintained a higher proportion of correct to incorrect responses than either the nonparanoid or the normal group. As can be seen from Figures 3 and 6, nonparanoid and normal performance remain quite close together across ensembles. This finding would appear to support the hypothesis of a differential mode of attentional functioning for paranoids as opposed to nonparanoids and normals. Paranoids appear to scan the perceptual field more rapidly and/or with a wider attentional beam width than either nonparanoids or normals, resulting in consistently better performance without regard to the ensemble variable.

The other two subject dimension comparisons (e.g. the chronic/acute/normal and the good premorbid/poor premorbid/normal) failed to reach significance regardless of the dependent measure or analytic procedure used. As there could be no question of accuracy of categorization in the acute/chronic/normal group comparison — patients hospitalized less than two years continuously were acute, while those hospitalized more than six years continuously were chronic — the apparent conclusion
is that splitting patients along this subject dimension does not yield differences in either attentional beam width or scanning rate.

Categorization for the good premorbid/poor premorbid/normal split is not so clear-cut. As data for premorbid adjustment classification was taken from hospital charts which offered sketchy information at best, it is conceivable that patients were incorrectly classified as to premorbidity. The Phillips' scale, used to ascertain premorbid adjustment (See Appendix A), required fairly detailed information regarding friends, associates and sexual patterns prior to hospitalization, and often the necessary information was unavailable from sources at the author's disposal. Assuming, however, that the patients were classified correctly as to premorbid adjustment, the data indicates no existing differences in scanning rate or attentional beam width between good premorbi ds, poor premorbi ds and normals.

The ensemble variable was consistently significant at the .01 level over all three subject dimensions. As was predicted, there is a general decline in performance across ensembles when d' and hits are dependent measures (Figures 3 and 6). As can be seen from Figure 9, there is also a rise in the number of false negative responses (or errors) as detection difficulty increased across ensembles. This was also predicted.

The multiple comparison tests indicated significant differences between all ensemble pairs on all dependent measures, except between
Ensemble I and Ensemble II for the chronic/acute/normal group comparison with hits and false negatives as dependent measures; and between Ensemble V and Ensemble VI for the good premorbid/poor premorbid/normal group comparison (with $d'$ and hits as dependent measures), and for the chronic/ acute/normal group comparison (with hits and false negatives as dependent measures).

The lack of a significant difference between Ensemble I and Ensemble II indicates that apparently the jump from a 50 cps difference in tones to a 100 cps difference had no effect upon performance. As a "normal" critical band width is thought to be in the neighborhood of 80 cps, this lack of a difference in some instances between Ensemble I and Ensemble II might be expected (Greenberg & Larkin, 1968).

The lack of a difference between Ensemble V and Ensemble VI might also be expected as the tonal separation for both ensembles (e.g. 1100 cps for Ensemble V and 1400 cps for Ensemble VI) was so great that — despite differences in critical band width and scanning rate — performance deterioration may have reached asymptote for the subject groups involved. Although this theory cannot be strongly supported without more data points, if performance is at asymptote, this may indicate that the scanning strategy has been dropped in favor of a single tone listening strategy. Were subjects to use this single tone strategy, their attentive mechanism would be fixated on only one of the two tones possible for each trial, rather than continually scanning the perceptual field.

With $d'$ as the dependent measure, the test session variable was significant at the .01 level across all three subject dimensions. When hits were the dependent measure, the test session variable was significant
for the paranoid/nonparanoid/normal group comparison and for the good
premorbid/poor premorbid/normal group comparison. With false negatives
as the dependent measure, test sessions were significant only for the
paranoid/nonparanoid/normal group comparison. The significance of the
test session variable presents evidence of a change in performance over
test sessions. In other words, repeated exposure to each auditory en-
semble led to differential performance from one test session to the next.
The direction of this differential performance was dependent upon the
auditory ensemble involved.

As was expected, the Ensemble X Test Session interaction was signifi-
cant over all three subject dimensions. The fact that this interaction
is significant indicates that performance underwent change at different-
tial rates across test sessions for each ensemble. In other words, some
ensembles showed more of a performance increase (or decrease) from one
test session to the next than others.

With $d'$ as the dependent measure, the overall increase from test
session 1 to test session 5 (indicated by the simple main effects tests)
for Ensembles I-IV is explained by the "practice effect" phenomenon.
The lack of an increase over test sessions for Ensemble V and Ensemble VI
could be explained by the fact that tones in these ensembles were espe-
cially difficult for all subjects to detect due to their wide separation.
This detection difficulty may have led to the adoption of a single tone
listening strategy which would not yield improvement over test sessions
comparable to the improvement given a scanning strategy. The reason for
this lack of improvement is that a single tone listening strategy is
essentially self-limiting in that there is always only a 50% probability
of listening for the correct tone.

With hits as the dependent variable, the Ensemble X Test Session interaction is illustrated in Figure 8. Simple main effects tests showed overall differences tended to be in a slightly upward direction from one test session to the next, with the exception of the significant drop in Ensemble V from test session 1 to test session 2. It is significant that differences between test session 4 and test session 5 are all in an upward direction, and that ensembles were again significant over test sessions 3, 4 and 5. This can be taken as evidence that the practice effect evidenced in the d' analyses also has relevance when hits are the dependent measure.

Simple main effects tests also indicated that test sessions were significant for Ensembles I, II, IV, and V over all three subject dimensions. Performance for Ensemble III and Ensemble VI remained stable across test sessions. The lack of significance for Ensemble III is not overly damaging to the practice effect theory, as the d' analysis showed significance for Ensemble III. Again, were the number of test sessions increased, it is assumed that performance would show an overall improvement.

The Ensemble X Test Session interaction when false negatives are the dependent measure is illustrated by Figure 11. Simple main effects tests showed that trends over test sessions tended to be in a downward direction for Ensemble I - Ensemble IV which is expected, as one would tend to make fewer false negative responses (or errors) with more practice. The lack of a significant downward trend for Ensemble V and Ensemble VI possibly indicates, again, lack of sufficient exposure for a practice effect to show.
It is interesting to note that the multiple comparisons for the test session variable invariably yielded a significant pairwise comparison between test session 1 and test session 5. This difference is always in the direction predicted by the practice effect hypothesis postulated as a result of the significant Ensemble X Test Session interaction.

It must not be forgotten that subjects also had exposure to the ensembles within the context of their practice sessions. The apparent conclusion is that the practice sessions were not continued long enough to yield a stable rate of performance across each ensemble. However, as their purpose was principally to familiarize subjects with the apparatus and its functioning, the obtained significance of the test session variable as well as of the Ensemble X Test Session interaction was expected.

A significant Subject X Test Session interaction was obtained when hits and false negatives were dependent measures for the paranoid/non-paranoid/normal group comparison and the good premorbid/poor premorbid/normal group comparison. Caution should be exercised in interpreting this interaction in terms of attentional scanning, beam width and accuracy as the Subject X Test Session interaction did not even come close to significance for any of the three subject dimensions in the d' analyses (F > .25 in all three instances).

The interaction is presented in Figures 7 and 10. Looking first at the case where hits are the dependent variable (Figure 7): Simple main effects tests indicate that paranoid performance remained consistently higher than nonparanoid performance over test sessions. The only salient statistical differences obtained from the simple main effects
tests are found in the fact that paranoid performance increases steadily from test session 1 to test session 5, while nonparanoid and normal performance remain at roughly the same level from test session 1 to test session 5 — with the exception of a drop in nonparanoid performance for test session 2. Interestingly, poor premorbid performance begins at a higher rate than that of the good premorbid group on test session 1, and retains its lead until test session 5. However, it can be seen that poor premorbid performance declines slowly over test sessions, while good premorbid performance rises steadily.

These data imply that the nonparanoid and poor premorbid groups benefit little from repeated exposure to the auditory ensembles, while the paranoid and good premorbid groups are able to use the extra practice to their advantage in improving their performance. This might be attributed to the narrow beam, slow scanning attentional mode postulated for nonparanoids and poor premorbrids. If these two subject classifications scan the perceptual field slowly, with a narrow attentional beam, it would be very difficult for them to develop the accuracy and consistency which lead to performance improvement.

The normal group follows the poor premorbid/nonparanoid trend, showing no improvement over test sessions. This finding (which refutes the original hypotheses) could be due to disinterest on the part of the control subjects. This possibility will be dealt with at length later.

Figure 10 presents evidence for much the same phenomenon when false negatives were the dependent measure. Paranoids and good premorbrids were apparently able to take advantage of the practice given over the five test sessions to reduce their error rate; while nonparanoids, poor
premorbs and normals were not.

In terms of attentional processes, this interaction indicates an ability on the part of paranoids and good premorbs to improve the accuracy with which they could pick relevant cues out of an array. As has been noted, this should be accepted only tentatively as a salient attentional trait of the two subject groups involved. Nonparanoids, poor premorbs and the normal subjects used in this experiment apparently lacked this ability.

This latter finding regarding the normal subjects illustrates one of the major problems encountered throughout this experiment — that of recalcitrance on the part of the control subjects. For the most part, they indicated exceptional boredom with the task and no real interest in performing well. Most were anxious to return to their units, as Stockton State Hospital has been very short of staff due to uncertainty as to whether the hospital will remain open. Incentive measures such as cigarettes, sweets and soda pop which greatly interested the patients had little or no effect on the staff and the author experienced a general lack of co-operation from all but two control subjects (who were personally known prior to the onset of the experiment). Perhaps paying the control subjects for their participation would have alleviated this difficulty to some extent.

Analyses of Variance With Two Between-Subject Variables

Two significant Subject X Ensemble interactions were obtained via the bipartite analyses; one of them for a d' analyses, and one for a false negative analysis. In both instances, it was the premorbidity subject dimension which interacted significantly with the ensemble.
variable. When d' was the dependent measure, it can be seen from Figure 12 that poor premorbid performance declined steadily and rather steeply over ensembles — starting at a much higher level than the good premorbid group for Ensemble I and dropping far below it upon reaching Ensemble VI. Good premorbid performance, despite erratic performance on Ensembles III, IV, and V, can be seen to remain at a fairly constant level over ensembles. These findings support the prediction that poor premorbid performance should decline much more rapidly than that of the good premorbid groups due to the former groups' narrow beam, slower attentional scanning mechanism.

The poor premorbid group as a whole were extremely anxious to please the experimenter and were continuously concerned with their performance — desiring continual reassurance. Conversely, good premorbid groups rarely were curious as to how they were performing — most were anxious for the sessions to end. It is significant, and supportive of the predictions, that despite their efforts and interest, the poor premorbid group could not maintain their performance at a significantly higher level than the good premorbid group when the auditory ensembles became more difficult.

Figure 13 presents evidence for much the same phenomenon when false negatives are the dependent measure. Poor premorbs make fewer false negative responses (or errors) until Ensemble VI when the good premorbid groups' error rate becomes less. Also, the error rate for the poor premorbid group rises steadily across ensembles, while the error rate for the good premorbid group tends to be much more stable. Again the hypothesis of a more rapid performance breakdown for the poor premorbid
group appears to be substantiated.

As can be seen from Figures 3 and 6, the statistical trend indicated by the simple main effects tests for poor premorbid performance to surpass good premorbid performance until Ensemble VI when the relationship is reversed, is also found when hits and d' are the dependent measures. Although it is more pronounced for the d' analysis, the statistical tendency for poor premorbid performance to decline at a fairly constant rate across ensembles, while good premorbid performance remains more stable, is also illustrated by these graphs. Thus, results for the bipartite interactions are supported elsewhere in the analyses of variance.

**Summary of Analyses of Variance Results**

The major problems in the experiment which may have affected the results adversely have already been mentioned. Recapitulating: A more accurate method (or alternatively, more accurate patient history records) should be used to assess premorbid adjustment. The auditory ensembles should be chosen so that tonal separation is more gradual and constant from ensemble to ensemble. And, some method should be arrived at to boost control subject interest and co-operation — possibly payment for participation could achieve this end.

The obtained significance of the Subject X Ensemble interaction for the bipartite analyses leads to the postulation that, could the obtained data be analyzed via a tripartite analysis (e.g. premorbidity, paranoia, and acuteness as between-subject variables) with both ensembles and
test sessions as within-subject variables, a greater number of significant Subject X Ensemble interactions would be obtained. It must also be remembered that significant performance differences were evidenced between paranoids and both nonparanoids and normals. This provides further evidence of differential modes of attentional functioning. Thus, the results from the analyses which were performed lend support to the hypothesis that a mode of attentional functioning exists which enhances the chances that a subject in a certain classification will detect tones more readily at some separations than at others.

**Multivariate Regression Analyses**

The Assist analysis was performed to explore, in greater depth, the relationships of the slope scores with the various independent variables used in this experiment. Slope scores were derived from the d' values for each subject, and represent relative change in performance from Ensemble I to Ensemble VI. Both analyses (using patients and staff and then patients alone) indicated that factors other than the independent measures of premorbidity, paranoia, acuteness, age, education, and drug dosage contributed significantly to the change in performance over ensembles for each subject. In other words, change (or lack of change) in performance must also be attributed to extraneous variables which were not controlled for in the context of the experiment.

The analysis using all 40 subjects had a greater amount of unexplained variation contributing to the change in performance across ensembles than did the analysis using only patients (2/3 as opposed to 1/2). This might be explained by the fact that the control subjects brought far more varied backgrounds — as a group — to the experimental
situation than did the patients. The patients, for the most part, shared a common 24-hour environment, experiencing similar schedules and pressures. Thus they presented a more homogeneous group when analyzed separately than when their data was combined with that of the normal group.

The failure of the F test for significance of regression over both analyses further strengthens the theory that things other than the independent measures contributed significantly to the slope values. The failure of the F test for the patients' data, when it was analyzed alone, could additionally be due to lessened degrees of freedom for this analysis.

The most interesting findings yielded by the Assist analyses were the partial correlations. The same four independent variables (premorbidity, acuteness, depressant medication, and phenothiazines) obtained the highest partial correlations for both analyses, although these correlations were consistently higher for the analysis using data from the patients only.

Premorbid adjustment yielded a consistent inverse correlation with the slope values indicating that poor premorbid patients tended towards a greater breakdown in performance across ensembles than did good premorbid patients. This finding supports the theory that poor premorbid performance should decrease more markedly across ensembles due to this group's narrow attentional beam width and minimal scanning pattern.

Acuteness was positively correlated with slope, indicating that there was less breakdown in performance across ensembles as the patients
became more chronic. This is surprising due to the fact that McGhie (1969) found chronic patients to be less responsive to all stimuli than acutes.

The positive correlation between the amount of phenothiazine medication taken and slope indicates that the greater the phenothiazine dosage, the less deterioration evidenced by each patient across ensembles. As the phenothiazines exert a calming, anti-psychotic effect, it is to be expected that their overall effect would be to enhance performance by changing attentional beam width and scanning rate in such a way that general attentional efficiency and cue utilization are increased.

On the other hand, the Assist program yielded an inverse correlation between depressant medication and slope, indicating that the higher the amount of depressant medication, the greater the deterioration in a subject's performance across ensembles. This indicates that depressants apparently do have a deleterious effect on scanning rate and attentional beam width.
Conclusions

The original hypotheses forwarded in the introduction were constructed upon the theory that normal subjects would perform at a level between the performance levels of each of the three patient categorizations used in this study (e.g., that normals would perform at a level between paranoid and nonparanoid performance etc.). Due to several factors previously mentioned, normal performance remained at very low levels, thus refuting the original presentation of the hypotheses.

However, several factors in the analyses point to the conclusion that, within at least two of the three patient classification systems used (e.g., paranoid/nonparanoid and good premorbid/poor premorbid) striking differences do exist in modes of attentional functioning.

For the paranoid/nonparanoid subject dimension, evidence points to the fact that paranoid performance both across ensembles and across trial blocks is substantially different from nonparanoid performance. As indication of this is the fact that paranoids do perform at a higher level, in terms of signal detection, than nonparanoids, and that they are able to profit from the practice afforded by the five test sessions to improve their accuracy at tone detection, whereas nonparanoids are not. Given this information, it appears that paranoid patients do indeed possess a wider-beam, more rapid attentional scanning mechanism than do their nonparanoid counterparts.

Likewise, good premorbid s also indicate an ability to improve their performance across test sessions as opposed to the poor premorbid group. This provides some evidence that good premorbid s may possess a more flexible, variable attentional mechanism (e.g., a higher limit
to scanning rate) than poor premorbid s. As good premorbid performance in terms of signal detection efficiency was not at a higher level than that of the poor premorbid group, the hypothesized difference in beam widths in open to question.

Thus, a mode of auditory attentional functioning has been shown to exist which enhances the chances that a subject in a certain classification will detect tones more efficiently at some separations than at others. Given the structure of the experiment and the lack of cognitive components in the experimental task, obtained differences appear to be due to the attentional mechanisms of either scanning or beam width. Hence, schizophrenic visual attentional deficit is, at least tentatively, paralleled in the auditory field. This indicates that schizophrenics could perhaps benefit from a program oriented around restructuring their attentional processes.

Future definitive research in this area should be carried out in an area with access to a large patient population in order to achieve a tripartite subject classification system. Could this end be achieved, along with the implementation of a more interested, co-operative control group, further support would probably be found for the trends appearing in this study.
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I. PRE-MORBID HISTORY

A. Recent Sexual Adjustment

1. Stable heterosexual relation and marriage .......................... 0
2. Continued heterosexual relation and marriage but unable to establish home .......... 1
3. Continued heterosexual relation and marriage broken by permanent separation .... 2
4. (a) Continued heterosexual relation and marriage but with low sexual drive ........ 3
(b) Continued heterosexual relation with deep emotional meaning but emotionally unable to develop it into marriage .......................... 3
5. (a) Casual but continued heterosexual relations, i.e., "affairs," but nothing more .... 4
(b) Homosexual contacts with lack of or chronic failure in heterosexual experiences 4
6. (a) Occasional casual heterosexual or homosexual experience with no deep emotional bond ........ 5
(b) Solitary masturbation with no active attempt at homosexual or heterosexual experiences ........ 5
7. No sexual interest in either men or women .................................. 6

B. Social Aspects of Sexual Life During Adolescence and Immediately Beyond

1. Always showed a healthy interest in girls with a steady girl friend during adolescence .... 0
2. Started taking girls out regularly in adolescence .................................. 1
3. Always mixed closely with boys and girls .................................. 2
4. Consistent deep interest in male attachments with restricted or no interest in girls ........ 3
5. (a) Casual male attachments with inadequate attempts at adjustment to going out with girls .... 4
(b) Casual contacts with boys and girls .................................. 4
6. (a) Casual contacts with boys and with lack of interest in girls .................. 5
(b) Occasional contacts with girls .................................. 5
7. No desire to be with boys and girls; never went out with girls .................. 6

C. Social Aspects of Recent Sexual Life: 30 years of Age and Above

1. Married and has children, living as a family unit .................................. 0
2. Married and has children but unable to establish or maintain a family home .......... 1
3. Has been married and had children but permanently separated .................. 2
4. (a) Single, but has had engagement or deep heterosexual relationship but emotionally unable to carry it through to marriage .................. 3
(b) Single, with short engagements or relationships with women which do not appear to have had much emotional depth for both partners, i.e., "affairs" .................. 4
6. (a) Single, has gone out with a few girls but without other indications of a continuous interest in women .... 5
(b) Single, consistent deep interest in male attachments, no interest in women ........ 5
7. (a) Single, occasional male contacts, no interest in women .................. 6
(b) Single, interested in neither men nor women .................. 6

D. Social Aspects of Recent Sexual Life: Below 30 years of Age

1. Married living as family unit, with or without children .................. 0
2. (a) Married, with or without children, but unable to establish or maintain a family home .......... 1
(b) Single but engaged or in a deep heterosexual relationship (presumably leading toward marriage) .... 1
3. Single, has had engagement or deep heterosexual relationship but has emotionally been unable to carry it through to marriage .................. 2
4. Single, consistent deep interest in male attachments, with restricted or lack of interest in women .... 3
5. Single, casual male relationships with restricted or lack of interest in women .... 4
6. Single, has gone out with a few girls casually but without other indications of a continuous interest in women .... 5
7. (a) Single, never interested in or never associated with either men or women .......... 6
(b) Antisocial .................................. 6

E. Personal Relations: History

1. Always has had a number of close friends but did not habitually play a leading role .......... 1
2. From adolescence on had a few close friends .................................. 3
3. From adolescence on had a few casual friends .................................. 3
4. From adolescence on stopped having friends .................................. 4
5. (a) No intimate friends after childhood .................................. 5
(b) Casual but never any deep intimate mutual friendships .... 5
6. Never worried about boys or girls; no desire to be with boys and girls .......... 6

F. Recent Premorbid Adjustment in Personal Relations

1. Habitually mixed with others, but not a leader .................................. 1
2. Mixed only with a close friend or group of friends .... 3
3. No close friends; very few friends; had friends but never quite accepted by them .... 4
4. Quiet; aloof; seclusive; preferred to be by self .................................. 5
5. Antisocial .................................. 6