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The Role of Neuropsychological Deficits in the Comparative Inability of Schizophrenics to Decode Facial Expressions of Emotion

William George McCown
Loyola University Chicago

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The Role of Neuropsychological Deficits In The

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William George McCown

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Finally, the author's wife Judy Johnson was as necessary a component in this dissertation as she is in other areas of the author's life. She is indeed the love of my life.

VITA

William George McCown is the son of George W. McCown, Jr. and Eugenia Garcia McCown. He was born March 8, 1956 in Florence, South Carolina.

His elementary education was obtained in the public schools of Washington D. C. His secondary education was completed in 1974 at Fort Hunt High School, Alexandria, Virginia.

In September, 1974 Mr McCown entered Kenyon College. In September 1976 he attended the University of Aberdeen, Scotland as a visiting student for three terms, reading for Senior Honours in departments of Sociology and Psychology. In 1978, he was elected to Phi Beta Kappa at Kenyon College, receiving departmental awards in sociology and psychology. He received a Bachelor of Arts from Kenyon in May of 1978, Magna Cum Laude, Highest Honors and distinction, with majors in anthropology/sociology and psychology.

Between 1978 and 1984 Mr McCown held a number of clinical and research positions all related to the science and practice of psychology.

In 1984 he was awarded a University Fellowship at Loyola University of Chicago, and enrolled in the doctoral program in Clinical Psychology. In November of that year he received

a research award from the Illinois Psychological Association. In 1986 he completed his Masters thesis entitled "An Empirical Investigation of the Behaviors of Chronic Academic Procrastinators". In September, 1988 he completed a predoctoral child and adult internship at Tulane University Medical Center, with specializations in neuropsychology and behavioral medicine.

Mr McCown has authored or coauthored the following:

Johnson, J., & McCown, W. (In press). An overview of substance abuse. In H. Adams & P. Sutker (Eds.), Handbook of psychopathology and behavioral change.

McCown, W., & Sutker, P. (1988). Alcohol and Stress: Interactions and Relations.(Book Review). Contemporary Psychology, pp.1039-1040.

McCown, W. (In press). Difficulties in securing conference reprints. Social Behavior and Personality.

McCown, W. & Johnson, J., and Petzel, T. (In press). Principal components of procrastination. Personality and Individual Differences.

McCown, W., Johnson, J., & Austin, S. (In press). Patterns of errors in delinquents' comparative inability to decode facial affects. Social Behavior and Personality.

McCown, W., Johnson, J., Austin, S., & Shefsky, M. (In press). Decoding of facial affects by parents of chronic schizophrenics. Psychotherapy and Private Practice.

McCown, W., Johnson, J., Silverman, N., & Austin, S. (1988). Social interest and affect recognition. <u>Individual Psychology</u>, <u>44</u>, 41-48.

McCown, W. (1988). Multi-impulsive personality disorder and multiple substance abuse: evidence from members of self-help groups. <u>British Journal of Addictions</u>, <u>83</u>, 431-432.

McCown, W. Petzel, T. & Rupert, P. (1987). Personality correlates and behaviors of chronic procrastinators. <u>Personality and Individual Differences,</u> <u>11</u>, 71-79.

McCown, W., & Johnson, J. (1987). Conference Reprints (letter). <u>APA Monitor</u>, November, 3.

McCown, W. Johnson, J., & Austin, S. (1986). Inability of delinquents to decode facial expressions of emotion. <u>Social Behavior and Personality</u>, <u>1</u>, 91-97.

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CHAPTER I

INTRODUCTION

Facial expressions are one of the most important nonverbal method of human communications (Ekman, 1982). This is true for two reasons. First, facial expressions are the most frequently utilized method of nonverbal communication. Secondly, facial expressions serve as spontaneous and frequent nonverbal reinforcers of behavior, potently shaping social actions without much notice.

Recent research (Mandal, 1986; Walker, Marwit & Emory, 1980) has suggested that there is a link between the ability to recognize facial expressions of emotion and a wide variety of clinical conditions. Most prominent is the link between this ability and schizophrenia (Muraki & Bates, 1977; Rosenthal & Benowitz, 1986; Rosenthal, Hall, Dimatteo, Rogers, & Archer 1979). The reason for these deficits observed in schizophrenics are unclear. Research to date has simply been correlational, without causal hypotheses (Rosenthal & Benowitz, 1986). Presently, it is impossible to tell whether deficits in nonverbal processing contribute to the

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clinical condition of schizophrenia, are the end result of the disease, or are simply a concomitant.

Any one of these relationships are possible and actually are not mutually exclusive. An inability to understand nonverbal communications might put individuals at risk for schizophrenic behavioral syndromes. By not understanding the nuances of the social world, schizophrenic individuals might become progressively more isolative from social corroborative experiences. It is also possible that the clinical symptoms of schizophrenia, characterized by terrorizing perceptions, delusional thinking, and anxiety-provoking social encounters (Taylor, 1981) could cause the schizophrenic individual to lose many types of nonverbal skills, perhaps because social contacts are simply too terrifying.

A third possibility exists in the causal relationship between affect recognition and schizophrenia. An organic condition could contribute to both schizophrenia and the inability to decode facial expressions. In this case, neuropsychological impairments related to schizophrenia would be the direct cause of facial affect recognition deficits that the literature suggests are found in schizophrenics. Considering the increasing evidence linking some types of schizophrenia (Golden, 1981; Zec & Weinberg, 1988) and many cases of affect agnosia (Cicone, Wapner & Gardner, 1980;

Ley, & Bryden, 1979;) to organic causes of known etiologies, this hypothesis deserves serious attention.

To date, no one has examined whether deficits in schizophrenics' inability to decode facial affects are due to cognitive or social problems related to their disease, or whether they instead can be explained by the numerous neuropsychological and neurological impairments that accompany schizophrenia. This dissertation examines schizophrenics with and without neuropsychological and neurological deficits to establish whether the comparative inability to decode facial affects found in this diagnostic group is directly related to neuropsychological deficits. To do this, six quasi-experimental groups were studied. These included three diagnostic categories: medical patients, depressed psychiatric patients (labeled as "affective patients" in this study) and schizophrenics. Furthermore, these groups were divided equally between individuals who showed clear evidence of neuropsychological or neurological deficits, and those who were neuropsychologically without such deficits.

Affect recognition is clearly a complex phenomena. It involves additional processes other than those measured by simply being able to verbally label specific emotions. Some of these include categorizations of affect intensity, affect pleasantness, and subjective certainty associated with affect recognition (McCown, Johnson & Austin, 1988). To date

only a handful of quasi-experimental studies have addressed these variables. A second goal of this dissertation is to serve as a preliminary study regarding the impact of schizo-phrenia and neuropsychological functioning on perceptions of affect intensity and pleasantness, and on the subjective sense of certainty associated with facial affect recognition.

Prior to a discussion regarding methods and findings, it is first necessary to review the rather lengthy litera-ture on facial expressions of emotion. The literature on schizophrenia and affect recognition will be briefly reviewed. Finally, neuropsychological localization theories of schizophrenia and how they relate to impairment in facial affect recognition will be discussed.

CHAPTER II

REVIEW OF THE LITERATURE

Facial Affect Recognition Research

Research Prior to The Twentieth Century

Jordan (1969) has noted that during the pre-scientific age the study of facial expressions was a popular topic of many philosophers. These included Plato, Aristotle, Epicurus, St. Augustine, and Kant. However, Jordan points out the <u>scientific</u> study of facial affects is relatively new. Previous students of the face were primarily concerned with physiognomy and characterological traits that supposedly were discernable from facial appearances.

The contemporary study of facial affect is often credited to Darwin (1872). Actually, its roots are more than a half century earlier. Charles Bell (1806/1928), an English physician, artist, and actor observed that certain facial muscles seem to have no function except to differentiate emotional expression. Unlike a canine, whose grimace prepares the creature for a rapid assault, humankind, according to Bell, was "endowed by the Creator with facial expressions serving no other purpose than to communicate

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emotional intention" (p. 4). The purpose of human facial affect, Bell concluded, is primarily to express emotions and nothing else. Piderit (1859), a German expatriot physician who lived most of his life in South America, also preceded Darwin in his study of facial expression. Piderit was the first to recognize that certain emotional expressions were common across cultures. Previous physiognomists believed that certain facial affects were peculiar to French, the Europeans, the Africans. Piderit, through meticulous observation, established that both the degree, and the direction of muscular movement of facial affects was the same for individuals in every culture. Certain facial affects, he believed, were universal, an observation more extensively verified by Darwin (1872) several years later. Similarly, there seems to be relatively high concordance about their intensity across similar emotional situations in diverse cultures.

Piderit disagreed with Bell's contention that affects were primarily "designed" for communicative purposes. Instead, he stated that each muscular function had an overriding utility in facilitating or inhibiting sensory perception. He labelled this principle the "maxim of emotional serviceability of facial affects". Each emotional expression, he argued, had a function closely tied to a corresponding sense organ. Disgust, for example, maximizes nostril closure, a useful adjunct for inhibiting noxious

odors. Smiles allow maximal pressure of the front portions of the tongue against the roof of the mouth and teeth, thereby allowing sweet or pleasant tastes to be savored.

Implicit in Piderit's belief was the assumption that facial expressions serve no major communicative function. Indeed, Piderit believed that most people barely attend to facial expressions. Those that do frequently do not properly identify them. Hence, facial affect recognition to Piderit was not an important human communicative capability.

Darwin (1872) expanded Piderit's concept of "emotional serviceability" with his "principle of serviceable associated habits". Darwin believed that at one time in humankind's past facial affect expression had a practical survival function. Natural selection instilled them because of their usefulness. The importance of affects for present day survival was now reduced. However, facial expressions in humans have generalized to analogous situations. Although frequently present in humans, they are less important than in lesser mammals. Human facial affects lack the intensity and survival value seen in lower primates.

Perhaps Darwin's greatest contribution (Ekman, Friesen, & Ellsworth, 1972) was to note that certain facial affects are indeed inborn and developmentally related. Even blind children grimace and smile, Darwin observed. Furthermore, children in every culture exhibit a similar repertoire of

facial expressions at similar ages. This suggests that facial expressions were an important survival mechanism, at least until recent years, when modern civilization seems to have mitigated the immediate need for such skill.

Darwin was unclear about the inheritability of the understanding of facial expressions. Following Piderit, he also vacillated on the importance of affects for present day communicative purposes. In a classic experiment, Darwin (1872) took "obvious photographs of facial affect", most of which were reproductions of larger popular photographic pictures. Although there was high agreement among his 20 subjects about certain facial affects such as smiling and sadness, there was less agreement among his subjects for other expressions. From this small experiment, Darwin concluded three things: first, some expressions are easier to recognize than others. Secondly, individuals seem to differ in their capacity to correctly identify facial expressions. Third, certain situations tend to make facial affect recognition more ambiguous.

Twentieth Century Facial Affect Research Prior to the 1970s.

With the development of experimental psychology, interest in the study of facial affect recognition accelerated. Progress-if it is measured by scientific consensus-however, was exceedingly slow (Ekman, Friesen, & Ellsworth, 1972). Experimental designs and questions asked regarding facial

expressions varied greatly. Results from one laboratory would frequently be unreplicable in another. There was very little agreement on the degree to which humans could correctly identify facial expressions. Simultaneous researchers were inadvertently asking dissimilar questions.

An exhaustive review of conflicting early twentieth century research on facial affect recognition has been provided by Ekman, Friesen, and Ellsworth (1972). Some outstanding examples of conflicting findings are apparent from the early literature. Langfeld (1918) was one of the first experimenters to systematically examine subjects' abilities to decode facial expression. He tested 11 subjects on the ability to correctly identify posed actors' emotional states. Over 111 emotional labels were presented for each affect. Accuracy ranged from 17% for one subject to 58% for another.

Furthermore, Langfeld found that subjects could readily be persuaded to endorse an emotional label incorrectly, even if they had previously correctly identified the same emotion. Langfeld interpreted these findings as indication of the general instability of the capacity for individuals to correctly identify facial affects. His conclusion was that other environmental cues are responsible for the process of attributing motives to facial expressions.

Langfeld's experiment can be severely criticized.

Langfeld presented different faces, each one portraying a differing "emotion", or set of instructions to the actor who portrayed them. There was no evidence that actors <u>correctly portrayed</u> these emotions. Furthermore Langfeld had no <u>a priori</u> basis to claim that these subtle differences he used as stimulus labels ("playful interest" vs. pretended astonishment", or "fury" vs. "sullen anger") corresponded to emotions commonly presented in real life situations. Langfeld's study showed only that his subjects had difficulty with the stimuli that he presented them, and that there were tremendous individual differences in these abilities despite what was certainly a large error variance. Indeed, with 111 choices for each facial affect displayed, even the subject who identified only 17% of the stimuli correctly did so considerably above the chance level that Langfeld claims.

The work of Feleky (1928) further reflects the confusion that prevailed in the early research on facial affect recognition. Feleky asked a different question, used a different method, and came up with an altogether different view of human capacities to recognize facial expressions of emotion. Feleky suggested that while subjects might err in identical agreement of emotional expression, (that is, they might not use the same labels for particular presented affects) the direction of choice of affect labelling is similar for most people. Feleky had subjects rate 86 facial affects with any adjective they chose. These categories

were then collapsed into those that were logically similar. He found that if seven categories were allowed and equal intervals were assumed for each category, interrater reliability ranged from 80% to 92%, depending upon the emotion tested. These results are the opposite from those of Langfeld, who denied that humans could accurately recognize facial expressions.

A similar set of results to those of Feleky's was obtained by Kanner (1931). Subjects were asked to subjectively select the "best word" to fit a description of facial displays. Categories were collapsed by expert judges, who maintained a high degree of interrater reliability for this task. Kanner concluded that there are essentially six emotional clusters which can be differentiated from each other. These include happiness, sadness, fear, surprise, anger, and disgust.

Schlosberg (1954) found essentially the same emotional clusters by applying the technique of factor analysis to facial affect research. Schlosberg suggested that the over two hundred popular labels of facial expression could be reduced to six groups: happy, sad, fear, anger, surprise, and disgust. Remaining affects could be seen as a combination of these more basic six, just as chemical compounds are combinations of more basic elements. Dismay, for example is a combination of two more primary emotions, in this case anger and sadness.

On the other hand, a number of studies indicated that people cannot reliably recognize facial expressions (Fernberger, 1928). This led Bruner and Tagiuri (1954) to question whether the ability to consistently identify facial affects had ever been established within the laboratory, much less in the real world. Bruner and Tagiuri's review article occurred in the same year that Schlosberg's factor analytic study was published. The former was published in widely read text. As Ekman et al. (1972) note, the results of this unfortunate coincidence was to discourage interest in facial affect recognition. While Schlosberg had shown that individuals can delineate the six basic affects consistently his research was mostly ignored for the next 15 years.[1]

The Work of Ekman and Friesen

This trend was reversed almost single-handedly by the work of Paul Ekman and his associates. Ekman, Friesen and Ellsworth (1972) suggested that Schlosberg's six components

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[1] A few exceptions were present. Frijda (1958) remained convinced that facial affect recognition was not reducible to situational context. VandenBerg and Mattson's (1961) work on affect recognition deficits in schizophrenics (discussed below) was published during this period. Abelson and Sermat (1962) devised a multi-dimensional scaling procedure for ratings of faces that continues to be popular to this day. Davitz (1964) edited a volume on communication of emotional meaning. Haggard and Isaacs (1966) even suggested that facial affects could be used as a variable in psychotherapy research. However, most of the important studies on facial expression were confined to Europe (Noummenna, 1964; Osgood, 1966; VandenBerg & Mattson, 1961), rather than to the more behaviorally oriented American laboratories.

could be used to establish reliably interpretable test stimuli for measuring the ability to decode facial expression. In a reanalysis of all of the major studies conducted on the ability to decode facial expression, Ekman et al. concluded that subjects could indeed make context independent and reliable judgments of emotional affect. This was true if the following conditions were met: Test stimuli were photographic, rather than artistically drawn (as previous studies of Fernberger (1928) and other had been), and, test stimuli were restricted to the six major affects described by Schlosberg (1954).

As Ekman et al. note, reliability of test instruments is a serious problem with facial affect research. This difficulty has been responsible for a great deal of the inconsistent findings in facial affect recognition literature. Candid photographs of unposed emotions-an early popular stimulus utilized by Darwin and others-are difficult to obtain and portend legal and ethical boondoggles. Actors instructed to pose emotions have had no criteria to judge their own success of facial accuracy. Artists' renditions of affect are equally handicapped without an objective criteria. Ekman and his associates attempted to solve this problem by constructing an objective measure of facial expression that could be used to validate subsequent emotional test stimuli. Their solution, the Facial Affect Scoring Technique (FAST) is based on the objective measure-

ment of patterns of muscle covariation. Numerical deter-
minants from the system are used to classify the degree of
accuracy with which a particular actor or model has por-
trayed a particular primary emotion.

Utilizing this scoring system, Ekman and Friesen (1976)
examined several thousand photographs of models portraying
facial expressions. Those with the highest validity, as
established by the FAST were included in a large study of
reliability. Expressions from models that had the highest
test/retest and interrater reliability were included as the
111 slides in Ekman and Friesen's (1976) Basic Affect
Recognition Test. In general, the interrater reliability of
individual facial stimuli is quite high, ranging from .97
for female models portraying smiling to .73 for several
models portraying the affect of disgust. To date, the Basic
Affect Recognition Test is the only empirically reliable
measure of facial affect recognition dedicated solely to
facial decoding.

Individual Differences in Affect Recognition

Accuracy

Most of the research in the twentieth century has
attempted to establish the universality of facial affect
recognition across cultures. Having done this researchers
have attempted to examine individual differences in these
abilities in greater detail. Actually, the foundations for

this effort began during the 1920s. F. Allport (1924) tested the hypothesis that the ability to recognize facial affects was improvable through practice. He found that a group of college women showed a small but meaningful increase in the ability to recognize facial expressions after a lecture on facial anatomy. More importantly, he found substantial, and rather consistent individual differences in the ability to recognize facial affects both before and after the treatment intervention of the lecture.

Guilford (1929) repeated a portion of this experiment with more systematic training provided to subjects. He found considerable individual difference in the ability to recognize 96 poses of facial expression. With training, the least successful subjects could be brought up to slightly below the mean level of the most successful subjects. Further training did not help the successful subjects.

Guilford believed that the results of this study had two implications. First, training teaches the less accurate subjects which areas of facial expression to attend to in decoding affect. Secondly, training seems to increase motivation to do well on this task. In retrospect, the most important finding of this study was ignored; even normal subjects show broad differences in their pre-intervention abilities to decode facial expressions.

Qualitative Approaches to Affect Recognition

All but about a dozen of the studies of affect recognition have used a dependent variable of a frequency count of the numbers of facial affects correctly decoded (Johnson & McCown, submitted). In the typical study, such as that done by McCown, Johnson, Austin, and Shefsky (in press) groups are shown facial expressions of emotion, usually in randomized order. Mean differences between groups of clinical or theoretical interest are then analyzed, generally for aggregate numbers of total errors of facial affect recognition.

This type of experiment has the advantage that it is relatively easy to implement and replicate. Furthermore, data analysis is straightforward, and unless there is a theoretical reason to suggest that a prior differences in affect recognition exist for certain types of facial expressions, the increase in power that such a broad test seems to offer suggests a strong utility for the above "shotgun" approach. However, this sort of "fishing net" affords no information about particular emotions that might be problematic for particular groups. A more sophisticated approach is sometimes indicated if such questions arise.

The idea of categorical errors extends at least from Feleky (1928), who suggested that normal individuals err in the same general "direction". The importance of categories of errors has frequently been ignored in the literature. The

previous type of facial affect recognition task examined the dependent measure of accuracy facial expressions as a function of <u>correct naming</u>. If the subject identifies an angry face as a happy one he is "no more wrong" than if he identifies an angry face as a disgusted one, emotions that seem more intuitively similar than happiness and anger.

The type of research with accuracy of affect naming as the sole dependent variable has been criticized in the literature for failing to reflect the subtlety in affect judgment (Mandal, 1986). A most thorough discussion of the necessity for an alternative to "mere accuracy" as the sole dependent variable has been made by McCown, Johnson and Austin (1988). These authors note that all mistakes in identifying specific facial expressions are not necessarily equally deleterious. Errors made in emotional affect recognitions involving substitutions with similar emotions are probably not too serious.

Drawing on Feleky's (1928) notion of patterns of errors they note that little is lost in normal social interchange if a person receiving the emotional expression-the decoder-- makes errors that are congruent with the <u>direction</u> of the emotion presented. An example is helpful in illustrating this point. Consider if the decoder confuses happiness with feelings of pleasant surprise. In this case the decoder has been able to decipher the encoder's mood trend of experienc- ing a pleasant feeling, while not being able to actually

label the specific emotion. Similarly, a person who consistently confuses anger and sadness would experience only slight social impairedness. He or she would sense that the encoder-the person presenting the emotion-was dysphoric. While the name of the particular emotion, and indeed its nuances, might escape the decoder, the general mood state of the encoder would not be missed.

It seems important, therefore that research go beyond a simple tally of numbers of affects perceived incorrectly, and instead examine patterns of errors made in facial affect recognition. Simply, some patterns of errors would logically seem to be more serious than others. Discussion of whether errors of emotional recognition cluster around similar emotions presupposes reliable underlying dimensions of similarity.

Pleasantness/Unpleasantness

Several such dimensions have been suggested by Schlosburg (1954), including that of pleasantness/unpleasantness. Past research has found this dimension to be very stable (Osgood, 1966), even across different cultures (Noummena, 1964). At least three types of methods are possible in studying this dimension.

McCown, Johnson, & Austin (1988) utilized types of errors in their study of patterns of errors made by delinquents. If neutral expressions are included in the analysis of errors that are logically possible, as they

would be in daily face-to-face interactions, there are eight possible types of errors based on the pleasantness/un-pleasantness dimension. McCown, et al. found that delinquents were more likely that other youths to make more errors of interpreting unpleasant emotions from pleasant facial affect stimuli. The hypothesis that this represents some type of psychodyanamic "anti-people" projection, however, has to be questioned since delinquents are more likely to rate neutral affects as both pleasant and un-pleasant. The authors admit they are perplexed what these findings mean, suggesting that perhaps delinquents have difficulty in emotionally ambiguous situations, and try to impart affect or intention in others when none is there.

An alternative approach to rating affect pleasantness that does not involve the clumsiness of the dimensional analysis of McCown et al. is advocated by Mandal (1986). Mandal has each subject rate individual expressions for their degree of resemblance to a pleasant face. Post hoc determinations of dimensions are found by factor analysis, providing numerous comparisons and ratings are made. This latter requirement is a handicap making it less appropriate for extended clinical research with impaired populations.

A third approach is more straightforward, though has appeared rarely in the literature. Subjects simply rate the affects on a Likert type scale for their assessment of affect pleasantness. This is a convenient and easily

understood procedure that promises to be more conducive to investigation with difficult populations, such as the medically ill or the chronically schizophrenic.

Intensity

Pideret (1858) was the first to consider cultural and individual differences in intensities of emotional displays. Additional interest in this area awaited Schlosburg (1954), who found the dimension of affect intensity to be orthogonal to perceived pleasantness /unpleasantness. Schlosburg suggests facial affects differ in their degree of intensity, and that perceived intensities cause the degree of response to the particular emotion that is displayed. This suggests the corollary that individual's perceptions of identical stimuli might also differ regarding how intensely they subjectively believe facial affects are being portrayed.

To date, no one has examined whether individuals differ in subjectively perceived intensity of experiencing facial expression as a function of either aspects of personality or of membership in a diagnostic subgroups. This is a surprising gap in the literature. Clinically, a frequent observation is that an expression that appears to one person as slight bother appears to yet another as gross outrage. Or, what appears to be a pleasant smile to one person appears to be ebullience to another. It not difficult to imagine a host

of personality factors and learning histories that might influence an individual's perceptions of affect intensity.

One example is familial history. Halberstadt (1983) found that people from expressive and emotional families were less accurate in decoding facial affects, presumably because of not learning to attend to subtle cues. It also seems that individual variations in perceived intensity might relate to dominant psychological themes present in the individual at time of stimulus presentation. Just as on the Rorschach, where the absence of color indicates a degree of emotional constriction, (Exner, 1978) the absence of the evaluation of particular emotional stimuli as intense when other individuals rated it as so could serve as an indicator of personality processes. This would seem to be fertile ground for future researchers which has been ignored by the present generation of facial affect studies.

Subjective Certainty

On the basis of his research with delinquent youths, the author and his colleagues (McCown, Johnson, & Austin, 1988) have suggested that a major variable of interest for affect recognition researchers might be individual differences in subjective sense of certainty of judgment associated with emotional perception. Relying on the concept of corrective feedback loops, McCown et al. argue that delinquents might lack the ability to seek confirmational evidence of their

subjective impressions of emotional situations. Simply, they might be unjustifiably <u>too certain</u> of the particular meaning of a perceived emotion. They then might react inappropriately on the basis of this too hasty attribution, and be unable to subsequently modify this misattribution.

To date, few researchers have examined subjective certainty of judgment of emotional expressions. Excessive certainty of affect judgment could easily be as pathognomic as consistent misattribution. In many situations affect information is not complete. Individuals insisting upon making judgments in these conditions would demonstrate a <u>de facto</u> error rate in real life situations that would probably impair their overall social functioning.

In summary, then, a number of tasks seem of use in examining individual differences between groups regarding the ability to decode facial expressions. Research might wish to begin with an aggregate tally of total errors, and then examine either patterns of errors or ratings of pleasantness and intensity associated with each emotion. Researchers might wish to examine individual differences in subjects' perceptions of affect intensity. Finally, experimenters might wish to examine differences in subjects' subjective perceptions of their own degrees of certainty regarding these emotions presented as stimuli. Individual differences with clinical significance might be observable in any of these areas.

Facial Affect Recognition Deficits and Schizophrenia.

Accuracy of Emotional Recognition

A persistent finding throughout the literature has been that schizophrenics show impairment in ability to decode facial expressions of emotion (Mandal, 1986; Rosenthal & Bekowitz, 1986). These studies have been reviewed for meta-analysis (Johnson & McCown, submitted). Eighteen of the studies found in the literature used a dependent variable of affect recognition alone. No study to date has examined subjective assessments of intensity or surety of judgment in schizophrenics and normal subjects. The study utilizing a multidimensional scaling task did so by showing pictures of affects and asking schizophrenics and normals to rate test stimuli on a degree of similarity with the affects being presented previously. In this manner, verbal labels were avoided (Mandal, 1986), although to date there is no clear indication that such a procedure presented any experimental advantage over a procedure which simply asked subjects to rate emotions with verbal labels without the anchoring stimuli being presented.

In the majority of studies schizophrenics showed a deficit. This comparative deficit has important ramifications for those working with schizophrenics: to the extent that schizophrenics are relatively immune to recognition of nonverbal communication, therapy with these patients would

need to rely on purely verbal and behavioral interventions, rather than on the typical emotional nuances that categorize the successful bond in a therapeutic experience. Furthermore, therapists attempting a psychosocial rehabilitation of schizophrenics would maximally ensure effective interventions only if they addressed these comparative deficits in nonverbal communication perception.

Pleasantness

Mandal (1986) has examined the dimension of pleasantness/unpleasantness. In this study, it was found that schizophrenics rated pleasant emotions as less pleasant, suggesting that schizophrenia involves deficits in hedonic attribution, as well as simply a thought disorder. Interestingly, unpleasant faces were not rated as less unpleasant, as would be expected if a mere reduction in variance were due simply to schizophrenia. Schizophrenics just seemed to dislike pleasant faces more than normals.

This is a finding explainable by the general personality paradigm of Eysenck and Eysenck (1985). They argue that a common personality core in schizophrenics and criminals is the original diathesis for schizophrenia. While not necessarily the precipitant cause of the full-blown syndrome, this factor of personality, labelled Psychoticism, or "P" appears to be strongly linked with a genetically based predisposition towards schizophrenia. "P", a factor analytic

construct loads heavily on personality traits that show a comparative antipathy for human tenderness and kindness, including a dislike and distrust of more pleasant motives of others. Additionally, P loads highly on items tapping hostile and oppositional thinking. Individuals with a "high P" have more negative attitudes towards those around them. Since schizophrenics are score at the high end of the distribution on the personality factor of P, it would be expected that they would rate pleasant faces of emotion as less pleasant.

Subjective Level of Intensity

No studies to date have examined the subjective level of ascribed intensity to facial expressions by schizophrenics as compared to ratings made by normal subjects. [2] There is literature consensus that schizophrenia is related to a "loosening of boundaries" and a deficit in perceiving the environment in a manner that others do (Andreasen, 1985; Exner, 1978). With this onset of a schizophrenic syndrome

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[2] What comparisons could have existed would be invalid under today's psychiatric nomenclature. The current Diagnostic and Statistical Manual, III, Revised (American Psychiatric Association, 1987) and earlier diagnostic manual DSM-III have relabeled acute and reactive schizophrenia (popular diagnoses until the mid 1970s) as "schizophreniform disorder". This entity is specifically excluded as being a diagnosis of schizophrenia. Consequently, there has been a narrowing of the definition of schizophrenia to include only what has been typically thought of as chronic or process schizophrenia. Because of this, great care needs to be made in citing literature published before the utilization of DSM-III.

comes the tendency for thought and judgment to become more idiosyncratic and less stimulus based. It might be expected that no particular direction in ratings of affect intensity by schizophrenics would emerge. Instead, it would be expected that schizophrenics would manifest more variance compared with non schizophrenics, since their responses are based less on stimulus characteristics and more on idiographic and illogical factors. An analogy with projective tests is useful here. Patients with schizophrenia have more atypical responses than normal subjects, simply because they are not bound by adequate reality testing (Exner, 1978).

Affect Certainty

Affect certainty is another area that has not been explored with schizophrenics. According to Eysenck and Eysenck (1985) schizophrenia is characterized by a psychotic personality core composed of dogmatic inflexibility and a difficulty in toleration for other points of view. If this is true, then we might see more subjective certainty of facial affect stimuli by schizophrenics. Schizophrenics would be less likely to hold such judgments in abeyance by rating them as less sure. Normal subjects should approach their task with a greater degree of possible ambivalence, and because they remain flexible about being corrected should show less subjective certainty of ratings for facial expressions of emotion.

Laboratory data relevant to this hypothesized finding is furnished by Claridge (1981). Claridge has found that schizophrenics tend to attenuate large portions of experimentally presented stimuli. Once they "lock in" on a portion of the stimuli they have difficulty processing information from other sources. Furthermore, DSM-III schizophrenics demonstrate mental inflexibility and difficulty in "set shifting" (Seeman, 1985), that seems to have persisted despite the changing criteria regarding which patients are presently classified as schizophrenics.

In an unpublished study Duncan and McCown (submitted) examined the Eysencks' personality variable of Psychoticism and its impact on the construct of affect certainty. In an outpatient psychiatric setting, 21 patients of mixed diagnoses attending group therapy sessions were administered the Psychoticism scale of the Personality Inventory, Revised (Eysenck, Eysenck, & Barrett, 1985). Subjects were then shown 12 different facial expressions of emotion, one for each of the primary affects (happy, sad, anger, fear, surprise and disgust) portrayed by a male and female. Subjects were then asked to rate each of their judgments on a 10 point scale for certainty, with one being simply a guess and 10 being absolute certitude. A mean rating of affect certainty was obtained by averaging each person's ratings. These averages were found to correlate .34 with the Psychoticism scale. Since the Psychoticism scale is elevated

in schizophrenics (Eysenck et al., 1985) such a finding would probably be more pronounced with individuals with a schizophrenic diagnosis. No one has tested such a hypothesis directly.

On the other hand, one study with DSM-II schizophrenics have suggested that schizophrenics are less consistent and certain of their overall interpersonal judgment of others (Livesay, 1981). Whether this applies to DSM-III schizophrenia, and whether it extends to the molecular social judgment of facial affect recognition is an empirical question needing research.

Schizophrenia, Affect Recognition, and
Neuropsychological Deficits

Hemispheric Dysfunction

It is widely believed that the ability to decode affect is localized to the right parietal lobes in left dominant hemispheric individuals (Cicone, Wapner, & Gardner, 1980; Dekosky, Heilman, Bowers, & Valenstein, 1980; Ley & Bryden, 1979); Brain damaged individuals with impairment in this area show two deficits. First, they are less accurate on tests of facial affect recognition. Secondly, they rate facial affects as less pleasant than non brain damaged people (Etcoff, 1983).

Schizophrenics' behavior on tasks of nonverbal information processing is relatively indistinguishable from

patients with right hemispheric damage [3]. A meta-analysis of studies of affect recognition deficits (Johnson & McCown, submitted) shows a similar effect size of the effects of right hemispheric damage on facial affect recognition as is found with schizophrenia. What is anomalous about this behavioral concordance is that schizophrenia is thought to be related to general left hemispheric, and particularly left frontal lobe deficits (Seemen, 1985), rather than to right parietal dysfunction as the neuropsychological evidence from affect recognition studies would seem to indicate.

To date, the preponderance of evidence suggests strongly that many schizophrenics show dominant hemispheric brain dysfunction (Andreasen, 1985). This evidence exists on several different theoretical and empirical levels. Since schizophrenia is thought to be a diseases of language and reasoning ability it is theoretically logical that it should localize to areas of the brain responsible for receptive speech and logical functioning, the left hemisphere. And if this is true, schizophrenia should not normally include other symptoms that would indicate right brain dysfunctioning.

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[3] Hemispheric lateralizations are used throughout this dissertation in reference to the typical, right handed, left dominant individual, who composes approximately 85% of the population (Taylor, 1981).

In general this is the case. Rarely, will schizophrenics hallucinate spatial distortions that would indict the right hemispheric functions (Taylor, 1981). On the other hand, as Taylor notes, dementias and acute brain syndromes ("course brain disease" or "organic brain syndromes") will display visual and spatial hallucinations not usually found in schizophrenia. Consequently, schizophrenia is believed to be a disease primarily of the left hemisphere.

A second argument concerns data from neuropsychological tests. Until recently, the literature on lateralized deficits in schizophrenics observed from neuropsychological or performances tests was not large. In general, neuropsychological evidence weakly supported the belief that schizophrenics have left brain impairment, particularly left frontal impairment (Golden, 1981; Taylor, 1981). The results were not unequivocal; for example, schizophrenics have poorer performance I.Q.s on the WAIS than do non-schizophrenics (Wechsler, 1958). However, the comprehension subtest of the WAIS, a particularly localized left frontal function (Golden, 1981) is especially depressed in schizophrenics.

A rather extensive literature has developed in the last 10 years indicating that at least one large subgroup of schizophrenics do remarkably poorly on tests that tap left frontal lobe functioning (Goldberg, Weinberger, Berman, Pliskin & Podd, 1988; Silverstein, 1988; Zec & Weinberger,

1988). On tests such as the Wisconsin Card Sort-a test extremely sensitive to left frontal lobe functioning, and involving hypothesis testing and set shifting-schizophrenics patients make significantly more errors than any type of control group (medical, affective, etc.) Furthermore, patients often continue to perseverate despite instructions, and a demonstrated capacity to grasp the contingencies of reinforcement and rule changes. These types of studies suggest that at least one type of schizophrenia is categorized by profound left frontal lobe changes involving a genuine dementia including a dissociation between knowledge and the ability to make use of this knowledge for gainful action (Goldberg et al., 1988).

The literature is clear regarding the findings of organic deficits in schizophrenics that localize to the left frontal lobes, in particular, and to the left side in general. However, this literature is not causal. A number of factors could cause neurological and organic deficits in schizophrenics including bad diet, bad living conditions, substance abuse, medication, and even stress from the disease itself. Regardless, a number of different, and not necessarily mutually exclusive explanations and behavioral correlates of this hypothesized deficit have been advanced or observed. These shall be discussed on the basis of their relative strengths in present day literature.

The first is that schizophrenics show less dense gray matter in the dominant hemisphere, as measured from CT scans. These findings have been reported by one laboratory (Golden, 1981), and replicated by another (Andreasen, 1985). This would mean that schizophrenia is related to a relative absence of left cortical gray matter. Schizophrenics simply have less necessary brain tissue in these regions.

Schizophrenic deficits in left frontal tasks are also related to decreased regional cerebral blood flow, as measured by radioactive scintillation techniques. Results have rather consistently shown small but significant differences in dominant cerebral blood flow levels between schizophrenics and control patients (Franzen & Ingvar, 1975; Ingvar & Franzen, 1974). Brain impairment, even necrosis of gray matter would be a possible outcome of this lack of adequate blood supply, compared with non schizo-phrenics. It is notable that these deficits do not appear to have been found elsewhere in the brains of schizophrenics outside of the left frontal lobes.

Enlarged frontal ventricles of left frontal lobes of schizophrenics have also been reported in the literature (Golden, Moses, Zelazowski, Graber, Zatz, Horvarth, & Berger, 1980; Weinberger, Torrey, Neophytides & Wyatt, 1979). Since such enlargement is almost always evidence of lobe atrophy (with the ventricles filling in the space that the shrinking lobes once occupied) this is strong evidence

of permanent cerebral changes secondary to or the cause of schizophrenia. Golden's group further found that the amount of ventricle enlargement, and hence atrophy, was predictable from the Luria-Nebraska Neuropsychological Battery (described below). This rather remarkable finding links behavioral performance to physiological processes associated in schizophrenia, and suggests that an organic etiology may have been found for at least one subtype of schizophrenia.

Another relevant organic deficit postulated to separate schizophrenics from others is differences in ability of the corpus callosum to transmit information between hemispheres of the brain. While not a left hemispheric problem directly such a deficit implicates severe hemispheric dysfunctioning that might render the dominant hemisphere incapable of performing its functions adequately. The corpus callosum is a bundle of neural fibers that seem to act as the gateway between hemispheres. Either a poorly developed or too well developed fiber track could provide difficulty associated with transmission of cortical information. Bigelow, Nasrallah, and Rauscher (1983) have presented autopsy evidenced suggesting that the corpus callosa of schizophrenics is often enlarged. As Andreasen (1985) notes aberrant corpus callosa would result in a case where the two hemispheres were not able to communicate well with one another, resulting in a decay of information processing en route to or from the dominant hemisphere. These authors
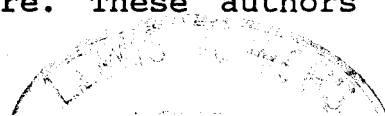
believe that this hypothesized process could easily fit the clinical picture of schizophrenia.

In summary there are a number of hypothesized observations and explanations concerning the comparative deficit of the dominant hemisphere in schizophrenia. Regardless of the hypothesized cause of the deficit there seems reasonable evidence that at least a portion of schizophrenics demonstrate dominant hemispheric dysfunction of some type, with some type of organic brain involvement (Andreasen, 1985; Seeman, 1985). This would result in a wide variety of neuropsychological deficits, with schizophrenics generally demonstrating superiority of right hemispheric functioning over left. What is surprising then, is that facial affect deficits of schizophrenics resemble those encountered in patients with right brain dysfunction. This contradiction would seem to beg for an explanation, of which one of several are possible.

Language Deficit and Motivation Hypothesis.

This explanation is rather straightforward. It states that although schizophrenics might be able to be as accurate as normals in facial affect recognition, they either are uncooperative in experimental tasks, or lack language skills to express what they are perceiving. The first part of this hypothesis, lack of cooperation, is always a possibility. Rapport with schizophrenics is difficult to obtain, and such a hypothesis is probably impossible to completely refute.

Language deficits and instructional difficulties are not a likely candidate for the cause of comparative inabilities of schizophrenics to decode facial expressions. Mandal (1986) gave schizophrenics a multidimensional scaling procedure, asking them to discriminate degrees of similarities among emotional faces. Even without language labels of primary emotions, schizophrenics still showed comparative deficits. Similar target emotions were judged less similar than those judged by depressed patients or non-patients. Opposite emotions were also judged less distant. Mandal concludes that schizophrenic emotional processing deficits are not a function of language difficulties but appear to be due to genuine errors in discrimination ability.

Feinberg, Rifkin, Schaffer, and Walker (1986) utilized four different affect recognition tests with schizophrenics. Two of these tests did not involve verbal labelling, but instead consisted of various matching tasks of stimuli with each other. In all four tests schizophrenics showed comparative inabilities with other psychiatric patients. This suggests against the hypothesis that schizophrenics show a comparative inability to decode facial expression primarily because of verbal deficits.

A Coincidence Hypothesis

The second possible explanation is that the results are coincidental. A different and poorly understood process

causes schizophrenics to fail to decode facial affects. This explanation seems to have merit. After all, a variety of clinical groups have shown an impairment in the ability to recognize facial affect, including juvenile delinquents (McCown et al., 1986), parents of schizophrenics (McCown, Johnson, Austin, & Shefsky, in press), and psychotic children (Cutting, 1980).

McCown, Johnson and Austin (1986) have suggested that a common genetic core might be present in these diverse populations causing such deficits. One such cause could be the Eysencks' notion of a similar biological personality factor common in both delinquents and schizophrenic (Eysenck & Eysenck, 1976), namely Psychoticism (P). Since Psychoticism has a high hereditability coefficient (approximately .80; Eysenck & Eysenck, 1985) it would be expected that parents of criminals or schizophrenics might demonstrate an impairment in affect recognition that corresponds with their genetic covariance with the population showing the affect recognition deficits.

While this interesting hypothesis has yet to be tested, Psychoticism as the lone explanatory construct in the comparative inabilities of diverse clinical groups to decode affect is not supported by two studies. Rosenthal, Hall Dimatteo, Rogers, and Archer (1979) administered the Eysencks' Psychoticism Scale to a group of inpatients. Rosenthal et al. also administered their test of nonverbal

sensitivity, the PONS, a multichannel measure of affect recognition accuracy to these same patients. The correlation between the ability to decode facial affect (a subscale of PONS) and the Eysencks' P scale was approximately .30 in a mixed schizophrenic/severely neurotic inpatient group. Even correcting for range restriction and for reliability of the measure which boosts the correlation to the upper .40's, only 20 to 25% of the variance of schizophrenics' deficits in the ability to decode facial expressions of emotion is attributable to P.

In a normal, nonclinical population, this correlation becomes more questionable. McCown (1988) found a correlation of .24 between psychoticism and the ability to decode facial affects in college students. This accounts for only 6% of the total variance. However, this correlation was found only when subjects viewed facial expressions for extremely brief periods of time through a tachistoscope. When subjects were free to inspect slides for five seconds, either as projected on screen or when handed pictures, the correlation between P and affect recognition became insignificant.

Since the version of the Psychoticism inventory used was a revised form from the one Rosenthal et al. administered, and since the affect recognition tasks were different, rigid comparisons between results are unfeasible. However, the difference in correlations could suggest that additional

processes in schizophrenics are responsible for the comparative inability to decode facial expression, other than simply their amount of psychoticism. While the personality variable of Psychoticism might account for some of the deficits in schizophrenics' inability to decode facial expressions, the great percentage of the variance appears due to other processes, for example, subsequent neuropsychological injury that might covary with psychoticism in a clinical population. The results could also suggest that the a negative relationship between psychotic symptoms and facial affect recognition is not linear but increases at a higher function for individuals whose Psychoticism scores are in the highest portions of the distribution.

Latent Neuropsychological Deficits

A third explanation is that schizophrenia either masks or is associated with more diffuse neuropsychological deficits, some of which might involve areas of the brain that process facial expressions, including, but not necessarily limited to the right hemisphere. This brain damage, causal, concomitant with, or subsequent to the disease, is the reason for the relative inability of schizophrenics to decode facial expressions of emotion. At least one study has suggested that schizophrenics resemble right hemispheric head injured patients in their comparative inability to decode facial expressions (Rosenthal & Benowitz, 1986). To

date, no one has directly attempted to compare schizophren-
ics with and without such damage in regards to their
ability to decode facial expressions.

A review article by Morrison and Bellack (1987) has
suggested that deteriorative brain damage might be respon-
sible for facial affect recognition deficits in some schizo-
phrenics. The implication is that by targeting subgroups
likely to be organically affected more differentially
efficacious psychosocial rehabilitation can be provided.
These authors argue that some of the initial promise of the
psychosocial rehabilitation movement may have been stymied
by a lack of knowledge regarding neurological possibilities.
Social skills training with neuropsychologically impaired
patients-especially schizophrenics-is much less likely to
achieve the magnitude of effect size that is possible with
neuropsychologically intact patients.

Affect Certainty, Intensity and Perceived Pleasantness and
Neuropsychological Deficits

The above discussion has reflected the literature
interest in affect recognition accuracy at the expense of
other theoretically fruitful variables. As noted previously,
individuals with organic conditions, especially right
parietal dysfunction have been found to rate facial expres-
sions as less pleasant. Regarding affect certainty and
perceived intensity of expression in neuropsychologically

deficit individuals, the literature is silent. Predictions must be made on what is known about the behavior of brain injured and deficit individuals. In a sense, this portion of the dissertation is a pilot study examining relatively newly conceptualized variables.

Affect certainty as well as other judgments of certainty regarding social stimuli would seem to be determined by at least two cortical function (Luria, 1973). One function would be the ability to self-monitor, highly dependent upon the frontal lobes (Lezak, 1983; Stuss & Benson, 1986). According to Luria (1973) individuals with such frontal lobe deficits have difficulty holding decisions in abeyance. They are likely to make "all or none" judgments, and have little tolerance for ambiguity. They appear to demonstrate an egotistical self assurance that they are correct. Since individuals with neuropsychological deficits would presumably be at risk for lacking the cortical equipment for self-monitoring, we might expect to see more subjective feelings of affect certainty in individuals with neuropsychological deficits.

Affect certainty is also likely to be related to right parietal deficits (in left hemisphere dominant individuals). Since Critchley's classic study (1953) it has been known that non dominant parietal dysfunction is accompanied by a randomness of response to the environment regarding visual and spatial identification, as well as an intense subjective

conviction that the response made is essentially reasonable and correct. These types of deficits have been discussed at length by Luria (1973). The most striking manifestation of this deficit is seen in unilateral neglect, a nondominant parietal dysfunction where the individual with an impairment in this area literally neglects and actually ignores input to the appropriate contralateral visual and spatial field, yet is absolutely certain he or she is not doing so.

Regardless of whether affect certainty is a function of the frontal lobes or the nondominant parietal lobes, individuals with organic neuropsychological impairment would likely demonstrate changes in affect certainty. We can hypothesize that they might rate themselves as more sure of facial expression judgments than nonneuropsychologically deficit controls. We might also suspect a compounding of the problem when the subjects are schizophrenics. Therefore we would predict an interaction between schizophrenia and the neuropsychological deficits on ratings regarding of subjective affect certainty.

Regarding affect intensity, observed effects for any particular constellation of neuropsychological variables have not been systematically investigated, thus requiring speculation based on clinical observation. Neuropsychological deficits are associated with less accurate neurological information processing (Luria, 1973). Consequently, critical information would likely be lost by those with such

deficits which might result in a general downward drift of subjective affect intensity. Since all of the information in the stimulus is not arriving correctly at its neurological target, the stimulus in question is likely to be rated as less intense. Unlike schizophrenia, which is hypothesized to be associated with affect intensity ratings that show a wide variance, neuropsychological damage will likely be associated with decreased intensity of affect perception.

CHAPTER III

STATEMENT OF THE PROBLEM

If the coincidence hypothesis were true, then the accurate diagnosis of schizophrenia _per se_ should contribute substantially to the comparative inability to decode facial expressions of emotion. Schizophrenics who tested negatively for neurological and neuropsychological dysfunction should still demonstrate significant deficits in facial affect recognition.

On the other hand if the hypothesis of "latent brain damage" is true, then schizophrenics screened for such deficits should demonstrate no more impairment in facial affect recognition than a sample of either affective disordered patients, or of medical controls. In this case we would argue that it is not the schizophrenia that causes these deficits, but accompanying neurological and/or neuropsychological damage.

It is also possible that schizophrenia and neuropsychological deficits combine interactively, so that affect recognition deficits are worse among brain damaged schizophrenics. They would also be expected to be impaired,

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though not as seriously, with both neuropsychologically deficit nonschizophrenics and schizophrenics without neuropsychological deficits.

These rival explanations have important theoretical and rehabilitative implications. If affect recognition deficits are due to neuropsychological impairments-which can be presumed to be permanent in the advanced stages of schizophrenia (Golden, 1981)-then it is an unreasonable therapeutic goal to suggest that such patients develop increased interpersonal empathy and social skills dependent upon facial affect recognition. Rehabilitation strategies involved would focus on coding information through other modalities, similar to the manner in which patients with brain injury are treated. However, if these deficits are due more directly to schizophrenia and its biopsychosocial syndrome-and not directly to brain damage-then a different strategy for rehabilitation might be in place. Affect recognition deficits might be found to be caused by any number of emotional or social situations associated with schizophrenia, such as paranoia, anxiety, depression and inability to concentrate. In this case, restoration of affect recognition would be an appropriate goal of therapeutic treatment, and might actually correlate with improvement from neuroleptics and gross symptom reduction.

Any test should not repeat the mistakes of previous researchers by undertaking a one-dimensional analysis of

affect recognition. Affect pleasantness, affect intensity and subjective certainty of affect identification can be fruitfully studied with predictions made from the literature regarding schizophrenia. Predictions can also be made regarding neuropsychological deficits and these variables. Pleasantness is hypothesized to be related to both schizophrenia and neuropsychological deficits, with both groups rating affects less pleasant. Affect certainty is hypothesized to be positively related to both schizophrenia and brain damage, with an interaction between the two conditions. Finally, affect intensity is hypothesized to be related to more variance in the schizophrenic group and less subjective intensity in the neuropsychologically deficit group.

CHAPTER · IV

HYPOTHESES

On the basis of the previous discussion, the following hypotheses are advanced:

Hypothesis One predicts that psychiatric diagnoses will have a significant effect on the ability to decode facial expression. The null hypothesis is that diagnoses will have no effect.

Hypothesis Two predicts that individuals with schizophrenia will be significantly impaired in their ability to accurately identify facial expression of emotion, compared with medical patients. The null hypothesis is that there will be no difference between these groups.

Hypothesis Three predicts that individuals who are schizophrenic will be significantly impaired in their ability to accurately identify facial expressions of emotion, compared with patients who are diagnosed as having an affective disorder. The null hypothesis is that there will be no difference between these groups on this variable.

Hypothesis Four predicts that a main effect will be found on the variable of neuropsychological impairment on the dependent measure of the ability to accurately decode facial expressions. The null hypothesis is that this variable will have no effect.

Hypothesis Five predicts that there will be a significant interaction between psychiatric diagnosis and neuropsychological impairment on the ability to decode facial expressions of emotion. The null hypothesis states that no interaction will be found.

Hypothesis Six predicts that patients who are schizophrenic and who are neuropsychologically impaired will make significantly more errors in facial affect recognition than individuals who are schizophrenic, but who are not neuropsychologically impaired. The null hypothesis is that there will be no difference between groups on this variable.

Hypothesis Seven predicts that psychiatric diagnosis will have an effect on patient ratings of certainty regarding responses to facial expressions. The null hypothesis is that there will be no significant effect for this variable.

Hypothesis Eight predicts that schizophrenics will be more certain of their judgment of facial affect recognition than medical patients. The null hypothesis is that there will be no difference between these groups.

Hypothesis Nine predicts that schizophrenics will be more certain of their judgment of facial affect recognition than patients with affective disorders. The null hypothesis is that there will be no difference between these groups.

Hypothesis Ten predicts a main effect of neuropsychological deficits on the dependent measure of subjective certainty of facial affect recognition. The null hypothesis is that there will be no effect.

Hypothesis Eleven predicts a significant interaction between neuropsychological impairments and schizophrenia on subjective certainty of facial expressions. The null hypothesis is that there will be no interaction.

Hypothesis Twelve predicts that neuropsychologically impaired schizophrenics will be more certain of their perception of affects than non impaired schizophrenics. The null hypothesis is that there will be no difference between these groups.

Hypothesis Thirteen predicts that the status of psychiatric diagnosis will have an effect on the variable of subjective ratings of pleasantness of emotions. The null hypothesis is that this variable will have no effect.

Hypothesis Fourteen predicts that schizophrenic patients will rate slides of facial expression as less pleasant than medical patients rate them. The null hypothesis is that no difference will be found between these groups.

Hypothesis Fifteen predicts that schizophrenics will rate slides of facial expression as less pleasant than patients with affective disorders. The null hypothesis is that there will be no difference between these groups.

Hypothesis Sixteen predicts that neuropsychologically impaired subjects will rate slides of facial expressions as less pleasant than non impaired subjects. The null hypothesis is that there will be no difference between these groups.

Hypothesis Seventeen predicts that there will be a significant interaction between neuropsychological impairment and schizophrenia on ratings of pleasantness of facial

expression. The null hypothesis is that there will be no interaction.

Hypothesis Eighteen predicts that neuropsychologically impaired schizophrenics will rate slides of facial expression as less pleasant than non impaired schizophrenics. The null hypothesis is that there will be no differences between these groups.

Hypothesis Nineteen predicts that unequal within group variances will be found between the schizophrenic group and the pooled variance of the other groups The null hypothesis is that variances will be the same between these groups.

Hypothesis Twenty predicts that neuropsychologically impaired patients will state less subjectively perceived intensity than non impaired patients. The null hypothesis is that there will be no differences between these two groups.

As it will be seen in the next chapter, six quasi-experimental groups will be used to test different hypotheses regarding the role of the schizophrenia and neuropsychological deficits on affect recognition. Since the literature suggests (Mandal, 1986) that affective disorders have a small but significant effect on affect recognition, hypothe-

ses regarding the comparison between the three groups of medical patients, affective disordered patients, and schizophrenics should be constructed to first find a main effect for the independent variable of diagnosis, and then to test more specific hypotheses regarding the order of mean differences between groups. Consequently, Hypotheses I, VII, XIII and XIX predict nonspecific differences between three groups. Even though they represent <u>a priori</u> theorization, related hypotheses following each of these (II, VIII, XIV and XX, respectively) should be tested only if the overall omnibus F test for the accompanying previous hypotheses are significant (Hays, 1981).

CHAPTER V

METHOD

Design

The design of this study is a 2 (neuropsychologically impaired vs not impaired) X 3 (status of diagnosis) quasi-experimental design. Assignment to quasi-experimental groups was dependent upon psychiatric diagnosis and categorization of patients as being neuropsychologically impaired.

Subjects

Inclusion Criteria

The psychiatric patient subjects were consecutive admissions to four psychiatric units of a large inner city general hospital. The medical patient subjects were admissions to the same hospital. All psychiatric patient subjects carried the DSM-III-R diagnosis of schizophrenia or depressive affective disorder. This study is a quasi-experimental design; therefore, no attempt was made to balance quasi-experimental cells by gender, race, or socioeconomic status, despite obvious interests in such

variables. Because of the demographic nature of "typical" patients-minority, female, and poor-such an attempt would have sacrificed external validity.

Patients were admissions to one of four separate 20 bed psychiatric units. Initially, only one 20 bed unit was to be utilized. However, preliminary statistics suggested a high proportion of patient admissions to this initially selected unit were schizophrenic. There was some concern that despite the safeguards (described below) a teamwide diagnostic bias was being felt, similar to the manner described by Janis (1972). Further analysis revealed that approximately the same number of admissions to each unit carried the diagnosis of schizophrenia, thus allaying concern. As an additional check on diagnostic integrity, each diagnosis was subject to frequent review from senior faculty members at one of two medical teaching universities utilizing the hospital for a training site.

Selection of patients from each unit was made on the basis of an interdisciplinary treatment team diagnostic decision. Treatment team members were not informed that the study was in the progress. This was done in an attempt to eliminate any potential bias that such knowledge might make upon diagnostic reliability.

Each team had essentially the same diagnostic procedures, with slight variations in personnel. Patient diagnosis on all units was made by an interdisciplinary team

headed by a board-certified psychiatrist, in consultation with a clinical psychologist. Additional personnel present at each staffing included one to two social workers, clinical psychology interns and two or three psychiatric residents, as well as nursing personnel from each unit. A unanimous diagnostic decision was necessary for inclusion of subjects in this experiment. The researcher was not involved in this classification whatsoever.

Psychiatric subjects were asked to complete a brief neuropsychological testing battery at the time psychological testing is routinely done (usually within five days of admission, providing the patient is cooperative). All subjects had received an Electro Encephalogram (ECT) and/or a Computerized Tomography Scan (CT) to rule out neurological damage, with one of these two procedures being decided upon by the attending physicians. These laboratory tests were screened blindly by a neurologist. Additionally, all subjects were screened with the Mini Mental State Examination (Folstein, Folstein, & McHugh, 1975) by either a psychiatrist, or a resident in neurology/psychiatry. The Mini Mental State Examination is a brief orally administered screening tool that is presently quite popular with physicians, who utilize these scores as a screening device to determine if further neurological work ups are necessary.

Subjects who showed no indication of EEG or CT abnormality, showed no neuropsychological deficits in neuro-

psychological testing, and had neurological impairment ruled out as a diagnostic consideration on the basis of physician examination were operationally defined as neuropsychologically normal. Patient who showed neuropsychological deficits on testing and who showed laboratory data associated with neuropsychological abnormalities (EEG or CT) were operationally defined as neuropsychologically deficient. Subjects who met only one or two of these criteria were not included in this study.

Two categories of comparison groups were utilized. One group was composed of patients who were diagnosed as unipolar affective disorder by the above diagnostic decisionmaking process. These included patients diagnosed as DSM III-R Dysthymic Disorder, Major Depression, or Adjustment Disorder with Features of Depression, but not bipolar manic depressive illness.

As with the schizophrenic group, this quasi-experimental group was subdivided into two subgroups, those with and without neuropsychological impairments. The criteria utilized within the schizophrenic group was also that of the affective group, including neuropsychological testing, a neurological screening, and the presence or absence of laboratory data.

A second comparison group was composed of medical patients from the same hospital. Physicians were asked if they would be willing to suggest eligible patients for this

study. Potential patients were selected at random from the availability lists of several medicine and surgical units. They were then verbally screened regarding their history of psychiatric hospitalizations. Prior screening was also routinely administered by medical students as part of a medical history and physical examination upon admission.

Eligibility was limited to non immediately terminally ill or nonterminally ill individuals with no history of psychiatric disorders. Although medical patients included in this study had in some cases terminal disorders, none of the patients were in critical condition.

As with the schizophrenic and affective disorder groups, two subgroups with and without neuropsychological impairment were obtained. All patients received the Mini Mental State administered by a medical resident of the relevant discipline. For medical patients who were included as neuropsychologically impaired the following criteria were required: Examination and positive findings by at least one neurologist or a resident (under a neurologist's supervision) of a neurological diagnosis; secondly, impairment on neuropsychological tests; third, laboratory data of neurological impairment, as indicated above.

The second subgroup of medical patients was composed of patients without neurological or neuropsychological impairment. These patients were also screened by a resident with the Mini Mental State. However, hospital practice was that a

neurology consultation and subsequent laboratory testing was not invoked if the patient did not have diagnostic signs prompting such a referral. By definition, these signs would exclude patients from this non impaired group. Consequently, these patients did not have had a neurological consult or lab work up for neurological problems. All patients, however, had been reviewed by internists, or other appropriate specialists screening for neurological disorders. Additionally, patients were included in this group only if their medical charts were noncontributory for neurological impairments and neuropsychological testing did not find indication of neuropsychological deficits.

Informed consent was be obtained from each patient in the medical and the psychiatric groups. This consent form was approved by the participating Internal Review Boards for Human Subject Research at the location of the study. Subject demographic variables for the entire study were as follows: 37% (44) of the subjects were male, while 63% (76) were female. Approximately 37% (44) of subjects were caucasian, while 59% (71) were black and the remaining 5 subjects were either Hispanic or Asian. Only 28% (34) of the subjects were presently married. The remaining subjects were either widowed, unmarried, never married, or divorced. Eighty-three percent of the subjects (100) were on some form of public assistance or supported by relatives. Fifteen

percent stated being employed or retired on pension. The remaining 2% did not furnish this information.

Exclusion Criteria

Patients were excluded from the psychiatric groups in this study if their was uncertainty about their Axis I diagnosis or if they could not give informed consent for legal or for personal reasons. Under state law minors (under 18 unless married) and legally detained "court-ordered" patients cannot give legal consent and therefore were excluded.

Subjects were routinely screened for visual impairment by a third year medical student (as part of the workup admission physical examination) to ascertain whether their vision was sufficient to see the test stimuli, described below. Vision better than 20/60 (corrected) was required. Only three patients were excluded from this study for this reason.

Instruments

Facial Stimuli

As Ekman, Friesen, and Ellsworth (1972) have shown, it is difficult to obtain facial affect stimuli that are reliable portrayals of the affects they are attempting to represent. An additional problem, even with a reliable measure of facial affect, is the method of stimulus ad-

ministration. Ekman et al. (1972) believe that the most natural procedure is to administer the stimuli via a tachistoscope, since their evidence suggests that most facial affects are presented for extremely brief periods of time. Another popular approach is to present emotional stimuli in the form of brief film vignettes (Rosenthal et al., 1979). On the other hand, many researchers opt for the more time efficient method of presenting slide stimuli on a projector for group testing. There is no published data concerning the superiority of one method of affect presentation compared with another, or whether one particular method produces results that are either more reliable or valid.

In a pilot study for this dissertation, the author (McCown, 1988) tested the reliability and intercorrelations of three different methods of presentation of facial affect test stimuli. These included screen projection of facial affect slides via a group format, individual tachistoscopic projection, and reproduction of slides onto 3" by 5" photographs that were hand inspected personally by each subject involved.

Both the "slide method" and the individual presentation of 3" by 5" photographs had essentially the same mean and standard deviations of recognition accuracy. The correlation between these two methods was reasonably good, .695 at a one hour interval. Tachistoscopic presentation correlated .581 with slide presentation, and .373 with individual

picture presentation. These results suggest that the slide and picture formats are essentially comparable and are tapping the same perceptual processes.

The slide format has the advantage that it can be administered in a group presentation. Unfortunately, the slide method presentation requires subjects to sit upright for prolonged periods of time in a fixed position at a specific distance form a lenticular screen. A second pilot study was conducted on 17 medical outpatients patients participating in a cardiac rehabilitation program that the author led. Fourteen of these <u>outpatients</u> reported that this method was highly uncomfortable. It was feared that even greater difficulties might be encountered in more seriously ill inpatients. Consequently stimulus presentation to each subject was by the individual picture method, a more comfortable procedure.

The test for facial affect recognition was selected from Ekman and Friesen's facial affect recognition test (1976), a series of 111 slides of the six primary facial expressions of emotion. The author's previous research suggests that equal numbers of slides of each emotion be utilized, with equal numbers of slides being portrayed by male and female models, as much as is possible with the distribution of gender in the slides. For obvious psychometric reasons, reliability is increased by additional test items. Consequently, when working with normal adults preference is

for a large number of slides, usually approximately 100 or more.

However, such a procedure might prove too long for medical patients and more severely ill schizophrenics and brain damaged subjects. Prior to collection of data it was important to pretest the ability of a random sample of schizophrenics and medical patients from the population included in this study to ascertain if the procedure would prove too exhaustive for accurate stimulus response.

In a third pilot study 14 schizophrenics and 11 medical controls were shown 98 slides of facial expression. Subjects were asked to circle an emotion that corresponded to the affects seen in the test stimuli. Several problems were evident. Two schizophrenics and two medical patients had difficulty consistently reading the stimulus words. Individual words were read with no difficulty. However, there was evidence of insertion and omission of key words when the pretest form was presented in toto. It was uncertain whether this represented a difficulty with literacy tasks due to age, lack of formal education, or primary dyslexia. However, it was clear that while the individual words (i.e. happy, sad, fear, surprise, anger and disgust) could be read once or twice, they became confusing on an answer sheet.

The second problem was found, surprisingly, with the medical patients, but not with the schizophrenics. Two subjects appeared to be answering essentially randomly after

about the halfway point of testing. Subsequent interviews with these patients indicated that the procedure was too tiring in its length.

In fact, on subsequent interviews four more medical patients stated that the procedure was too long to complete comfortably. Since Feinberg, Rifkin, Schaffer, and Walker (1986) report adequate variance for a similar task with schizophrenics utilizing only 22 slides from the Ekman and Friesen series it was determined that a reduction in facial stimuli was an adequate trade-off to prevent subject fatigue. Pilot study four found 42 presentations (three each for happiness, sadness, anger, surprise, fear, and disgust, as well as for neutral affects, each being displayed by both genders) to be a sufficiently short task that encountered no complaints, even for two patients that were acutely ill with exhaustive HIV complex disorders.

Neutral slides were not included in this study, because of issues regarding their reliability. No norms are available regarding what percent of the population correctly decodes neutral slides as neutral. Consequently, the number of affects presented was reduced to 36.

Another difficulty was anticipated by Feinberg et al. If schizophrenia is related to left hemispheric dysfunctioning, it would be expected that schizophrenics might demonstrate difficulty in emotional labeling that is independent of their actual capacity to recognize emotions. They might

recognize emotional similarity but fail to come up with the adequate words necessary. Feinberg et al. found that schizophrenics often knew which emotions were similar, but could not attach verbal labels to the stimuli in a free, unstructured recall test where such words needed to be spontaneously produced without prompts. Their solution was a task of matching, in which schizophrenics were shown affects of different people two-at-a-time, and asked if the faces displayed similar emotions.

This type of matching procedure has an advantage in that it eliminates the need for subjects to be able to read the very basic words involved in this test. While pilot data ($\underline{N}$=4) by this author demonstrates that the matching method of the above authors was workable, this procedure was rejected for two reasons. First, chance approximations (.5) reduce the variance involved with such a method, making it much less sensitive a measure of deficits.

Of more concern is the dual processing nature of such a matching task. The patient must make two apparently diverse judgments. One relates to emotional categorization, while the other regards canceling interference based on an absence of facial identities, a frontal lobe function that schizophrenics are at risk to perform poorly (Golden, 1981). In a nonverbal matching test subjects do not have to respond to words. However, they do have to make a second and more complex response involving ignoring the interference genera-

ted by the fact that the stimulus affects and the categorization affects are of different people. This involves a set shifting process that might further impair schizophrenics.

The author's test of four schizophrenics with a matching paradigm found that one subject was so impaired that he could not comprehend that he was to ignore that the two faces he was to match were of different people. He was, however, able to circle appropriate words corresponding to emotions he perceived a series of affects to be displaying. This finding is entirely predictable from neuropsychological theory. Golden states (1978) that verbal information processing of words is the least likely skill to be disrupted by either schizophrenia or brain damage. The rather extensive literature on the Stroop Color Word Naming Test supports this (Jensen & Rohwer, 1966). Simple printed words are overlearned and responded to very quickly, even by severely cognitively impaired individuals. Schizophrenics and brain damaged subjects seem to have no difficulty understanding the words "happy, sad, anger, fear, surprise, and disgust" as long as they were presented in a limited fashion.

In the author's pilot study a subject who had less than a third grade education was able to perform the reading requirement and the task at hand, despite the presence of a global inability to respond to sentences with more than three words. However, there seemed to be some subject

confusion when an entire <u>page</u> of words-even repetitive ones-was presented at once. Because of this it was decided to administer the affect recognition test orally, a more cumbersome procedure, certainly, though one that better guarantees proper subject response.

Four inch by six inch cards with the emotional labels were prepared with two inch Roman stencil. These were placed on a flat surface, approximately three feet from the sub-jects' faces if they were medically able to sit. For bedridden subjects, similar cards were attached to a small poster board held up approximately two to three feet from the subjects' heads by a portable chart stand. Exact measures were occasionally compromised by medical equipment and demanded flexibility. As a safeguard patients who could not read these words from the distance of two feet-either due to total illiteracy, or to additional eye problems not screened out in the preliminary medical student examination were immediately eliminated from the study. Nine subjects were eliminated from the testing procedure on the basis of the reading criteria described above.

Aphasia Screening

Despite the ability of schizophrenics to read simple prompt words, one caveat must be applied that seemed to have eluded previous researchers. Individuals who manifest one of the many aphasias might not be able to perform the required

task. Aphasias-or difficulty in processing language-can interfere with the ability to understand verbal directions, or the ability to respond correctly to written directions. Indeed, in a sixth pretest one individual with aphasia could read the stimulus words adequately, but was not able to comprehend the instructions. Another could read the stimulus words adequately, yet was not able to respond consistently when stimuli were immediately repeated. A third neurological patient demonstrated echoic perseverance, and answered each question exactly the same.

To guard for these aphasia related problems encountered during the pretest the author utilized the verbal portions of the Halstead Aphasia screening test (Reitan, 1957) a brief and very accurate examination to rule out aphasias that might interfere with testing procedures and results. Subjects were excluded from the study if aphasia scores were beyond the cut off suggested by Reitan, and those published in the norms of Russell, Neuringer, and Goldstein (1970). Six subjects were excluded on this basis.

Neuropsychological Testing

This study utilizes neuropsychological testing as one of the procedures to rule out or diagnose brain impairment. Three method are typically used in such psychological screening when it occurs in either a research or clinical setting. The first method involves the utilization of a

single test that literature has indicated is highly sensitive to neuropsychological deficiencies. According to Golden (1981) and Lezak (1983) such tests include the Digit Span and Digit Symbols, from the Wechsler Adult Intelligence Scale. They also include the Stroop Color Word Naming Test, the Benton Visual Retention Test, the Trailmaking Test, Finger Tapping and Strength Dynonometer Tests, and the Wisconisn Card Sort. A review of the literature indicates that the majority of neuropsychological studies published to date utilize only one test as a measure of organicity.

A second and probably more accurate method for assessing organic impairment and subsequent neuropsychological damage is the utilization of a comprehensive neuropsychological battery. Two batteries are commonly employed in typical tasks, the Halstead-Reitan, and the Luria-Nebraska (Golden, 1981). The Halstead has a number of strong adherents (Reitan & Davison, 1974), and seems to be the test of "tradition". The Luria-Nebraska is a rather new test, constructed in the late 1970s. Based on the theory of brain functioning by A. S. Luria (1963; 1973), the Luria-Nebraska is a clinician-administered examination that takes about one-and-one half to three hours to complete (Golden, 1981).

For general neuropsychological screening, there is no reason to prefer one of these two tests over the other, except for the factor of convenience that the briefer Luria battery affords (Golden, 1981; Golden, 1987). Both the

briefer Luria and the lengthier Halstead-Reitan seem to possess accurate "hit rates" for diagnosing brain localization dysfunction (Golden, Hammeke, Purisch, Berg, Moses, Newlin, Wilkening & Puente, 1982). On the other hand, comprehensive neuropsychological batteries are probably inappropriate for most research questions. The typical neuropsychological battery assesses scores of functions and rules out an equal number of other diagnoses. It is frankly too lengthy for most research that does not require such information (Golden, 1987). While comprehensive batteries may have a slightly higher hit rate than individual tests, they exhaust the subject receiving them.

In this study there was little need for the additional information such batteries provide. Irrelevant data typically derived from total batteries would include overall I.Q., short and long term memory functioning for multiple sensory stimuli, rhythm and tone perception, agraphia, acalculia, dyslexia, numerous anomias, and the many dyspraxias that are routinely and laboriously assessed in a full battery.[4]

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[4] Yet another approach presently popular in the literature is the utilization of a brief version of standardized test, such as Golden's (1981) Pathognomic scale, a subtest of the Luria Nebraska Inventory. In this case, the Pathognomic scale is composed of items thought to be easy for a non brain damaged individual to "pass" and very difficult for a brain impaired person to accomplish. Clinical experience has suggested that brief tests such as the Pathognomic scale have accentuated "hit rates"in lower socioeconomic populations such as the one used in this study. Literature, however, is lacking on this point.

To avoid the inconvenience to the patient of a lengthy battery, yet increasing the "hit rate" associated with the single test method, Golden (1981) suggests the common clinical practice of utilizing multiple sensitive single indicators in what is known as a "screening battery". This procedure involves the utilization of three or four highly sensitive tests for brain impairment used together. Tests can be chosen to differentially tap impairments in diffuse areas. In this manner, two tests that have a hit rate of 80% each and that are differentially sensitive to different areas of deficits can be combined to produce a hit rate much higher than each test individually, or two or more tests that are equally strong in assessing similar deficiencies.

Golden (1981) recommends screening batteries include assessment of frontal lobe functioning, lateralization deficits, and measures highly sensitive to overall impairment. Since affect recognition is thought to lateralize to the nondominant parietal hemisphere, adequate assessment of this areas is essential. Care must be taken to utilize tests that do not routinely confuse schizophrenia with organicity, a difficult task at best.

Routine Wechsler Intelligence Scales contain some of the best diagnostic information regarding brain damage (Golden,

1981). An essential test of overall neuropsychological functioning is the Digit Span test of the WAIS, or WAIS-R (Lezak, 1983). DeWolfe et al. (1971) found that a comparison between the Comprehension Subtest and the Digit Span produced a sensitive indicator of impairment. In the case of brain damaged subjects, Comprehension was higher than Digit Span.

Golden (1981) has suggested that this indicator might be dependent upon population-specific parameters. While Dewolfe et al. suggest a scale score of one point difference between the Comprehension and Digit Span might be sufficient to suggest a diagnosis of brain damage, normative data on fifteen inner city medical patients similar to those being tested in this study (collected by the author in routine clinical work, as compared to a formal pilot testing attempt) found scaled score differences of three points with one normal subject, and three incidences of scale scores with Comprehension two points higher than Digit Span. Because of this, the criteria of a C/DS difference of <u>four</u> points is utilized as a cut off. The arbitrariness of this cut-off point is tempered somewhat by the clinical experience of the researcher and his immediate supervisors in dealing with populations of the type sampled in this study.

Golden (1981) suggests a very powerful indicator of nondominant hemispheric impairment is the WAIS-R Block Design Subtest. This test has good discriminative abilities

in schizophrenics and neurological patients. Another test with similar discriminative ability in schizophrenics as well as nonschizophrenics is the Digit Symbol Subtest WAIS-R. While the Digit Symbol Subtest is highly sensitive to brain damage, it is also sensitive to anxiety and distraction An alternative test has been suggested by Smith (1982), the Michigan Symbol Digit Modalities Test, essentially a more investigator controlled version of Digit Symbols. This test is administered orally, controlling somewhat for distraction and anxiousness, and more so for tremors that are frequently a side effect of medication or age. This test is highly effective in determining organicity in both schizophrenics and in general patients.

Unfortunately, some of the most sensitive tests for brain damage are performed extremely poorly by schizophrenics. These include the Wisconsin Card Sort and the Halstead Categories Test. However, Golden's modification of the Stroop Color Word Naming Test (1978) is a standard and effective tool that is able to differentiate the presence of organicity with or without the presence of schizophrenia and still serve as a rather sensitive test of frontal lobe functioning. It was also hoped to utilize the Trailmaking Test from the Halstead battery. However, the author's clinical experience has suggested that these scores are significantly depressed by involuntary and intentional tremors that frequently affect individuals who have been on

high doses of phenothiazenes for prolonged periods. Since many patients included in this study fit this category, trailmaking was not included. Unfortunately, there is no literature regarding adjusting such scores for patients with such disorders as Tardive Dyskinesia and Parkinsonianism.

Right hemispheric differences (often used generically in the literature to indicate the nondominant hemisphere) are extremely important to note in this study. To do so, two effective and simple tasks can be employed. The Halstead Finger Tapping Test (Lezak, 1983) simply has subjects tap a telegraph key as fast as they can for 10 seconds. Average scores over five trials are computed with each hand and norms are then utilized to suggest presence or absence of hemispheric dysfunction. A similar second test is the Strength Dynometer, a hand-held squeezing device that can be similarly utilized. Since both tests are approximate measures of the same functioning, either is appropriate, although the finger tapping test would appear to confound perseverance and attention with simple lateralization measures as it requires ten total trials overall, five for each hand. Both tests were utilized in this study.

Right parietal functioning is popularly examined directly by visual-spatial performance in the Aphasia Screening Test (Taylor, 1981). Particularly, failure to adequately construct such figures as the Greek Cross- a

subtest of the Halstead Reitan Aphasia Screening Exam-is highly pathognomic of right parietal deficits and could possibly correlate with the inability to decode facial and other nonverbal perception. Similarly, the WAIS-R Subtest of Picture Arrangements is also diagnostic of right hemispheric, and particularly, right parietal functioning. Since neuroleptics and physical and psychiatric symptoms can impair the quality of the "Greek Cross" test in seriously ill patients (due to either motor strength problems or voluntary muscular tremors) the Picture Arrangement Subtest of the WAIS-R, which does not require particularly fine motor movement, was utilized rather than the drawings from the Halstead Reitan Aphasia Screening Test.

On the basis of the above, the screening battery employed in this study consisted consist of the following: 1. Verbal items from the Aphasia Screening Test, 2. A combination of Digit Span and Comprehension Subtest from the WAIS-R, 3. The Symbol Digits Test, 4. The Stroop Color Word Test, 5. The Block Design Subtest from the WAIS-R, 6. Strength Dynometer Test and Fingertapping Tests, 7. The Picture Arrangement Subtest Test from the WAIS-R.

Examiner recording sheets for neuropsychological variables were constructed, recorded and scored. Cut-off scores for evidence of organicity were obtained for age normative data germane to each test, summarized in the test manual, (the Stroop, the Symbol Digits Test, and the Aphasia

Screening Test) or in Lezak (1983), and in Russell, Neuringer, and Goldstein (1970) for the WAIS-R and performance measures. Digit Span/Comprehension comparisons were made on the basis of pretest data as described previously.

Individuals who showed deficiencies in any of the tests were classified as neuropsychologically impaired. Subjects whose Aphasia Screening Test was beyond the cut-off were immediately discontinued from the study as it was possible that they might not be able to follow or execute directions adequately, thus confounding any experimental effects.

The thoroughness of these tests compared with routinely utilized batteries for detecting organicity is noted from clinical testing material collected by the author, while obtaining direct supervision from a neuropsychologist. Eleven complete Luria-Nebraska inventories were also supplemented with the above battery of nine tests, in a manner suggested by Golden (1981) who argues that the Luria-Nebraska should be preceded by a screening battery composed of many of the above tests. In 10 out of 11 cases the results of the Luria were in agreement with those of the screening battery, as defined by one cut-off score in the battery diagnosing brain damage and one critical level in the Luria reaching the same conclusion. In one case the Luria was more lenient, possibly due to the adjustable critical level cut off scores that are generously reduced for lack of education of patients. In this case, four Luria

scale scores approached the critical level, and one brief battery cut off score was superceded.

More impressive was a second set of data collected on middle class psychiatric inpatients with possible organicity who had been given not only a CT scan but also a Metal Resonating Image (MRI) test, a very sophisticated radiological procedure that allows precise color pictures to be made of brain functioning. Eight patients with senile, presenile, traumatic, or HIV related dementias were administered the above battery as part of clinical duties. Administration was prior to scheduling of the MRI. Global impairment was accurately identified all eight times. Furthermore four patients with functional disorders, and two non demented HIV positive patients were correctly identified prior to MRI testing as having no impairments. Only in one case was a diagnosis made on the basis of neuropsychological evidence that was not confirmed by the MRI. In this case the patient was very limited in capacity to speak English. The accuracy of the screening battery utilized in this study should therefore be clear.

Procedures

Psychiatric patients selected for testing were done so by the researcher asking nursing personnel whether newly admitted and "staffed" patients had diagnoses of either schizophrenia or affective disorders. (Over 80% of the

patients fell into these diagnostic groups). The researcher further specified that he did not want to known which of the two diagnostic categories the patient being solicited was classified under, but wanted to include the patient "only if they are one of the two, but please don't tell me which". In this manner the researcher attempted to remain "blind" regarding the patient's diagnosis. Following testing, the researcher would validate the diagnosis from the patients' chart.

Subjects were tested individually. Rapport consistent with instructions in the Wechsler Adult Intelligence Scale, revised (Wechsler, 1977) and the Rorschach Test (Exner, 1978) was maintained. That is, subjects were offered vague encouragements and reinforcement deliberately noncontingent upon response pattern, but instead contingent upon response effort. Only two subjects refused to participate because the task was "too difficult", having decided to terminate participation after the data collection began.

Each subject signed an informed consent agreement. Subjects were then told the purpose of the experiment: "This project is examining the relationship between various abilities and people's perception of emotions".

Following this, subject were given the facial affect recognition test. Responses were solicited orally and then recorded by the researcher on an answer sheet. The procedure was as follows: 3" by 5" photographs from the Ekman

series were randomized and recorded on an answer sheet prior to each presentation. Subjects were presented with the first picture, and given the following instructions:

> Now, we're going to ask you to identify the particular emotion you see here. The emotion can be any one of six different emotions. (At that time the researcher pointed to six filecards with the names of the emotions tested). What I want you to do is to tell me which of these you think it is you are seeing.

The researcher then handed the subject the facial stimuli and allowed visual inspection for five seconds. For the second trial the researcher reminded each of the subjects:

> Now, remember, you can answer any of these: happy sad, anger fear, surprise, or disgust. You said (here the person's response would be repeated and recorded).

At this point, the subject was free to change his or her mind. For the next nine trials the researcher would read each of the affect choices out loud. This was not done after the tenth trial unless the subject requested it or seemed confused.

Subjects were allowed to change their decisions until the next stimulus was presented. If the subject wanted to change a previous opinion he or she was told, "Why don't we just go on to the next one. You are doing real well".

Following selection by the subject of an emotional label for the affect presented the researcher stated:

> Now on a scale of one to seven, with one being very
> little and seven being the most, how I am going to ask
> you how sure are you of this choice you just made.

At this point, the researcher laid out a five inch pictorial

diagram of equally appearing intervals. The following

instructions were read:

> As you can see "1" is marked "not sure at all". 2 is
> marked "not too sure" 3 is marked " a little sure".
> 4 is marked "somewhat sure". 5 is marked "pretty
> sure". 6 is marked "very sure". 7 is marked
> "extremely sure". Your job is to pick the number that
> best describes how sure you are of your choice
> of emotions.

The researcher then repeated this explanation for the

question of "how intense do you think this person is

displaying this emotion" with the above procedure and scale.

Instructions were as follows:

> Now, I'm going to ask you how intense, or forceful
> the emotion you just saw was. We'll use the same seven point
> scale again. (At this point a second five inch scale was
> shown) This time 1 is marked "not intense at all". 2 is
> marked "not too intense". 3 is marked "a little intense". 4
> is marked "somewhat intense". 5 is marked " pretty in-
> tense". 6 is marked "very sure", and 7 is marked "extremely
> intense". Your job is to pick the number that best de-
> scribes how intense each emotion you see is being dis-
> played.

The final task for subjects required them to rate

affects for perceived pleasantness. Subjects were presented

with a seven point equal appearing interval scale as above

for degree of pleasantness with instructions duplicating the

above. The researcher simply repeated the above instruc-

tions, substituting the words appropriate for instructions

regarding ratings of pleasantness.

Instructions were reiterated for trials two through 10 unless the patient appeared to understand the instructions without prompting. They were also repeated each time the researcher believed that the subject had lost site of the task at hand. Subjects were then administered the neuro-psychological battery, according to standardized instructions.

An approximate time for subjects to complete the affect recognition and rating test was 10 to forty minutes. Most subjects "caught on" to the task rather quickly and became adept at rating and categorizing each face without significant prompting of categories or rating scales. This usually occurred before the first 10 slides. Naturally, schizophrenics and more severely brain impaired subjects demonstrated more inability and were slower, although the variance within subjects seemed sufficient to blind the researcher to between group categorizations.[5]

In 11 additional cases (other than the two above) it was necessary to stop the testing. In two cases this was due to medical problems procedures that needed attending during the session. Three patients completed the testing but showed significant evidence of aphasia. Two psychiatric patients withdrew after the beginning of the study for no apparent reason, specifically denying the difficulty of the test was

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[5] It is notable that the group that seemed the fastest on this task (in retrospect, and without formal timing) were the depressed psychiatric patients.

a contributing factor. Four patients had families that interrupted the testing.

Wherever possible an attempt was made to complete the testing during one session. When rapport was threatened, however, or patients appeared to be significantly exhausted or otherwise unwilling, the procedure was postponed to two, or, in four cases, three sessions. Two additional cases were started and then terminated due to interruption, and subsequent patient discharge.

Since rapport was probably a critical factor in this experiment, every attempt was made to ensure its adequacy. Often, the researcher was known to the patients, especially the psychiatric patients, from his general clinical duties within the hospital. This seemed to facilitate patient cooperation for the long, occasionally arduous, testing sessions that were necessary. Care was also taken to explain the nature of the study to the families of patients involved, and to obtain their cooperation and consent.

As there is some literature that suggests that the act of signing consent forms may, in some cases, influence the experimental outcome, copies of consent forms were presented to families only after the data was collected.

Safeguards for Confidentiality and Researcher Expectancy

In experiments where a sole data collector performs as both assessor of quasi-experimental groups and collector of dependent measures the possibility for unconscious investigator bias is worthy of attention. Rosenthal (1976; 1981) has highlighted the problems of unconscious investigator expectancy and has suggested steps that can be taken to minimize the problem.

To some extent this bias was minimized by standardized procedures and the utilization of objective and easy to score set of measures. Diagnostic consensus involved a team approach, and once committed to the medical chart, was objective. Furthermore, the researcher was not present at staffings, and took a deliberately "low profile" regarding staff contact during the period of data collection to minimize inadvertent exchange of information regarding patient status.

Additional safeguards were also employed. These involved performing the assessment that delineated quasi-experimental groups after the collection of data involving the dependent variable. Facial affect data was collected prior to neuropsychological testing, thus minimizing the possibility of subtle gestures or other factors biasing subjects in a particular direction.

Unfortunately, the "agnosia" of neurological functioning and diagnosis in psychiatric patients was occasionally

compromised by comments of medical staff. Furthermore, due to their delusional verbal content or paucity of patient verbalizations it was often very obvious which patients were in the schizophrenic group and which patients were depressed. For example, one patient introduced himself as "Rainbow Doughnut".

The expectancy bias problem with the affective disorder and medical patient controls was a less salient issue. It is hypothesized that this group would do well on the affect recognition test. Expectancy bias usually operates to lower ability performance (Rosenthal, 1963), rather than raise it, at least for short durations. This is especially true where ability results in a natural ceiling effect for each subject.

To protect confidentiality of patients, however, computer safeguards involving generation of coded files were employed. Data was scored and entered into the computer, usually within a matter of hours. All identifying information was then destroyed to protect patient confidentiality. This was an especially necessary provision since some of the patients were suffering from HIV related diagnoses. The relational data base kept a count of how many subjects had been tested in each quasi-experimental category. Finally, when CT or EEG information was available, these values were placed into the computer file.

The data base would then, if necessary, revise the numbers of patients appropriately fitting each of the categories, dependent upon the new information regarding neurological status. Testing for new subjects was discontinued when the data base indicated a sufficient number of patients meeting each group was obtained. Because of this procedure five additional patients were tested that were not needed.

Criteria Checks of Between Group Differences

While neuropsychological diagnosis was determined objectively, DSM-III-R diagnosis, or absence of a DSM-III-R syndrome was dependent upon diagnostic assessment rather than a test instrument. Physician diagnosis is frequently unreliable and invalid (Taylor, 1981). Consequently, measures were administered to different groups to make sure that group differences did indeed exist between the quasi experimental cells. These measures were not designed to corroborate individual diagnoses, but to ensure that sufficient diagnostic accuracy was existing between groups.

Schizophrenia was assessed by the psychoticism scale of the Eysenck Personality Inventory, Revised (Eysenck, Eysenck, & Barrett, 1985), and by 15 items from the Whitaker Index of Schizophrenic Thinking (Whitaker, 1980) an instrument that purports excellent ability to discriminate schizophrenics from other types of patients. Items chosen from the Whitaker were those used by he researcher and his

supervisors on a routine basis and found to have excellent discriminative validity. The Beck Depression Inventory was also administered. to ascertain group differences in depression.

Tests were administered following other collection of other data. Usually this was done during the same time as the previous data collection, however, in some cases as much as three days elapsed following the primary testing session. In most instances, the validity measures were administered orally, with the assistance of a medical student, or, in several cases, nursing personnel. Oral administration was again necessary due to the fact that little was known about subjects' reading abilities, a serious concern with this lower socioeconomic, and frequently uneducated population. This procedure usually took 20-30 additional minutes.

CHAPTER VI

RESULTS

Table one shows the mean values Psychoticism, the Beck Depression Inventory, and the Whitaker Index of Schizophrenic thinking for each of the quasi-experimental groups. These mean values suggest that the quasi-experimental groups did indeed differ according to the indices of depression and schizophrenia.

Hypothesis One predicted that a main effect of diagnosis would be found on the dependent variable of facial affect recognition. A 2 (status of neuropsychological impairment) X 3 (psychiatric diagnosis) analysis of variance was performed. This hypothesis was supported, F (2, 114) = 5.818, p < .004. [6] What this means is that _some_ statistically significant difference exist between the three groups of medical patients, affective disordered patients and schizophrenics. This test is a necessary, but not sufficient step for indicating that schizophrenics differ from affective

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[6] The most exact probability for levels of statistical significance will be utilized throughout this work, rather than the traditional rounding of probability values to increments of .05, .01, .005, etc. This is done to facilitate meta-analysis by future researchers, where more exact probability levels are desired.

Table One

Mean and Standard Deviation of Diagnostic

and Demographic Data

<u>Variable Values</u>

<u>Group</u>	Beck (SD)	EPQP (SD)	Net-work Size (SD)	Age (SD)	Whit-aker (SD)
Medical Non Impaired	15.30 6.85	2.95 1.39	5.50 2.61	45.45 20.72	2.65 1.69
Medical Impaired	17.60 10.07	2.55 2.03	3.35 1.82	53.45 12.67	3.60 1.69
Affective Non Impaired	29.50 7.94	2.60 1.69	5.25 2.12	46.85 16.27	3.75 1.69
Affective Impaired	39.64 21.27	2.80 2.35	3.66 1.37	47.10 15.14	3.42 1.60
Schizophrenic Non Impaired	16.60 9.61	5.75 2.31	6.20. 1.88	38.50 14.87	5.15 2.62
Schizophrenic Impaired	20.00 10.61	6.10 2.73	2.40 1.42	42.90 15.08	5.05 2.37

Beck=Beck Depression Inventory
EPQP=Psychoticism Scale, Eysenck Personality Inventory
Network Size=Number of People Living with Patient at Home
Whitaker=Whitaker Index of Schizophrenic Thinking

disordered patients or from medical patients on the variable of affect recognition accuracy (Winer, 1971). Table Two shows the means and standard deviations for correct facial affect recognitions for each specific emotion for each quasi-experimental groups.

Hypothesis Two predicted that schizophrenics would make significantly more errors in facial affect recognition than medical patients. Hypothesis Three predicted that schizophrenics would make significantly more errors than patients with affective disorders. For the medical patient group the mean number of total correct responses was 31.52, with a standard deviation of 3.29. For the affective disorder group the mean number of correct responses was slightly less, 31.05, with a standard deviation of 5.05. For the schizophrenic group the number of correct responses was 28.67, with a standard deviation of 4.37. These averages "translate" into an average number of correct responses per emotion of 5.25, 5.15, and 4.77 respectively. These latter means, simply division by the number of categories of affects presented, are more intuitively understandable and will be utilized for additional hypotheses.

Table Two

Accuracy of Facial Affect Recognition by

Quasi-Experimental Subgroups

	Emotion Mean and Standard Deviation					
Group	Happy	Sad	Anger	Fear	Sur-prise	Dis-gust
Medical Non Impaired	5.45 .68	5.60 .50	5.40 .68	5.35 1.13	5.25 .71	5.05 1.10
Medical Impaired	5.75 .44	5.55 .89	4.95 1.23	5.15 1.08	4.95 1.23	4.60 1.50
Affective Non Impaired	5.80 .41	5.55 .75	5.70 .47	5.25 1.11	5.45 .99	4.95 .88
Affective Impaired	5.65 .49	5.25 1.01	5.40 .82	4.50 1.57	4.45 1.39	4.15 1.38
Schizophrenic Non Impaired	5.20 1.11	5.00 1.29	4.60 1.35	4.80 1.19	4.95 1.19	4.25 1.06
Schizophrenic Impaired	5.10 1.11	5.05 1.31	4.75 1.06	4.40 1.42	5.00 1.21	4.25 1.20
Mean Total	5.49 .79	5.33 1.01	5.13 1.04	4.90 1.26	5.08 1.16	4.54 1.23
Mean Medical	5.60 .59	5.57 .71	5.17 1.01	5.25 1.10	5.10 1.08	4.82 1.29
Mean Affective	5.75 .45	5.40 .90	5.50 .69	4.87 1.39	4.95 1.30	4.55 1.21
Mean Schizo-phrenic	5.15 1.09	5.33 1.01	4.65 1.27	4.60 1.32	4.97 1.18	4.25 1.12
Mean Non Impaired	5.48 .73	5.38 .94	5.23 1.01	5.13 1.15	5.21 .99	4.70 1.05
Mean Impaired	5.50 .71	5.28 1.09	5.03 1.07	4.68 1.39	4.80 1.28	4.33 1.36

Prior to additional statistical tests, a brief discussion regarding multiple hypothesis testing is necessary. The probability of rejecting the null hypothesis when it should not be rejected increases as a function of the number of statistical procedures performed (Hays, 1981). One popular way of controlling for this when it is necessary to test numerous _a priori_ hypotheses is to repartition the between-group variances with orthogonal contrasts. However, this weighted polynomial approach is much more sensitive to departures from normal variance than its immediate predecessor, the overall analysis of variance (Winer, 1971).

Consequently, orthogonal contrasts are inappropriate if nothing is known in advance about a population distribution. Since little is known about the theoretical distributions underlying the dependent measures in this study a conservative approach suggests the imposition of the requirement of non-orthogonal contrasts. While these latter types of contrasts are more frequently utilized for _post hoc_ procedures, they are an appropriate and conservative measure for _a priori_ hypotheses testing (Kirk, 1982).

For procedures where an _a priori_ hypothesis is tested with a less powerful range statistic, the moderately "protective" Duncan test (Kirk, 1982) is utilized in this study. The Duncan procedure affords more power than more conservative _post hoc_ procedures. It simultaneously affords less protection against Type I error. Since, however, it is

being used on a priori hypotheses that would have "allowed" multiple analyses of variance it is a legitimate means of protection and an excellent compromise between Type I and Type II errors.

In addition, the Duncan procedure is very robust, tolerating violations of the normal distribution (Kirk, 1982). If set to the individual mean comparison significance level of .01, the combined probabilities of rejecting the null hypothesis when it should not be rejected are less than .05 for up to six different means compared with each other.[7]

The Duncan multiple range test conducted on mean differences in affect recognition per emotion (ranges = 3.70 and 3.86; pairwise comparison, p < .01) indicates that the schizophrenic group differs from the medical group. The medical group does not differ significantly from the affective group, nor does the affective group differ significantly from the schizophrenic group. For the schizophrenic group the average number correct affects is 4.77 for each emotional category, with a standard deviation of .842. For patients with affective disorders, the mean number of correct affect recognitions per category is 5.18, with a standard deviation of .690. For the medical patients the

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[7] The exact probability is $1-(1-alpha)^{r-1}$ (Duncan, 1955). For six mean comparisons conducted simultaneously (one for each of the affects tested or one for each of the quasi-experimental groups) and each pairwise comparison set at the .01 level, the probability of a Type one error is .0490095. For three comparisons (diagnostic groups) this overall Type I error is .029701.

average number of affect recognitions for each emotion is 5.25 with a standard deviation of .549. Consequently, Hypothesis Two is supported, while Hypothesis Three is not supported.

Hypothesis Four predicted that patients with neuropsychological impairments would make less correct affect recognitions than non impaired patients. For the neuropsychologically impaired groups the mean number of correct affects identified for each emotion was 4.94, with a standard deviation of .77. For the non impaired groups the mean number correct is 5.20, with a standard deviation of .66. The 2 X 3 analysis of variance conducted above (on total errors, or these mean values multiplied by six) indicates that these differences are significant, F (1,114) = 3.94, p < .0494.

Hypothesis Five stated that an interaction would be found between diagnosis and neuropsychological impairments upon the ability to decode facial affects. This hypothesis was tested by examining the significance of the interaction term in the 2 X 3 F test conducted above. This interaction is not significant, F (2, 114) =3.62, p < .261. Therefore this hypothesis is not supported.

Hypothesis Six stated that the neuropsychologically impaired schizophrenic group would show significantly more affect recognition errors than the non impaired schizophrenic group. Although Hypothesis Five, which predicted an

interaction between schizophrenia and neuropsychological dysfunction was not significant, this hypothesis can still be true if the composite results of both deficits are additive. To test this hypothesis, which makes an a priori prediction, a one-way analysis of variance testing between group differences is appropriate. In addition, such a test will maximize statistical power. (Had there been no prior theory behind this test more conservative post hoc statistics would have been more appropriate).

Despite this power, however, the analysis of variance is not significant, F (1, 38) = .0238, p < .8781. The mean number of correctly named affects by the neuropsychological impaired schizophrenic groups' errors was 28.55, with a standard deviation of 5.0521. The mean of the non impaired schizophrenic group was 28.800, with a standard deviation of 5.1870. Actually, the results were contrary to the hypothesis, although results were not significant. Therefore, this hypothesis is not supported.

Regarding the question of accuracy of affect recognition, a post hoc exploration of patterns of recognition deficits between these six quasi-experimental groups might shed additional information of interest. As discussed above, to so involves some statistical risk of a Type I error, because there are no clear theories in advance predicting if specific emotions are more apt to be missed by specific groups. In the case of this type of "data snooping" an

appropriate procedure-wise adjustment to the alpha level should be made to minimize Type I error.

Univariate one-way analyses of variance were run for each of the six emotions for the dependent variable of recognition accuracy. All of these F tests were adjusted with the Bonferoni procedure "hypothesis-wise" (Hays, 1981; that is divided by the number of tests run in this post hoc analysis. In this case again, one test for each emotional presented).

Utilizing the procedure advanced by Kirk (1982) and others, if and only if the level of significance reaches the adjusted Bonferoni alpha-in this case .0083-additional post hoc statistics may be run and interpreted to attempt to explain sources of between-group differences. For this study post hoc group differences that are significant at this level were then examined by the Scheffe test (Winer, 1971). The Scheffe test is the most conservative post hoc tests commonly utilized (Winer, 1971).

The above procedure was performed on group differences on the dependent variable of affect decoding accuracy. Six separate analyses of variance were conducted, requiring an alpha level .05/6 or .0083 to warrant further between-group comparisons for group differences associated with particular emotions. Table 3 shows the results of these univariate tests, and whether the Bonferoni adjustments to the alpha levels were significant.

Table Three

Analysis of Variance for Post Hoc Univariate Tests of Emotional Accuracy

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Emotion	F	Significance level	Significance with adjusted alpha
Happiness	2.887	.017	no
Sadness	1.435	.218	no
Anger	3.803	.003	yes
Fear	2.001	.083	no
Surprise	1.744	.131	no
Disgust	2.081	.072	no

Univariate Tests with (5, 114) degrees of freedom

The six quasi-experimental groups differed among themselves only for the emotion of anger. Since this is true, a Scheffe range test on the mean number of correct responses for this emotion may now be performed. The results of this test (Ranges = 4.79; p < .05) indicate that the non impaired schizophrenic group differs significantly from the non impaired affective disordered group. Non impaired schizophrenics are more likely to misidentify the emotion of anger than are non impaired affective patients. No other contrasts are significantly different.

The next group of hypotheses require a second 2 X 3 analysis of variance, this time with the dependent measure of subjective ratings of affect certainty. Table Four shows the means and standard deviations for each quasi-experimental group, and by independent variables of diagnosis and status of neuropsychological impairments.

Table Four

Ratings of Certainty by Quasi-Experimental Subgroups

	Emotion Mean and Standard Deviation					
Group	Happy	Sad	Anger	Fear	Surprise	Disgust
Medical	31.95	30.65	29.75	28.90	30.25	28.15
Non Impaired	7.28	6.89	7.04	7.94	6.29	6.76
Medical	30.90	32.05	32.15	30.45	28.65	28.25
Impaired	7.10	5.81	7.70	9.06	10.36	9.10
Affective	30.05	25.65	29.15	31.45	28.50	29.60
Non Impaired	10.32	9.54	8.67	5.90	8.90	7.51
Affective	29.05	22.45	20.60	28.85	27.10	28.85
Impaired	8.76	7.85	8.89	8.98	7.94	7.16
Schizophrenic	26.95	29.90	31.55	16.25	29.75	29.30
Non Impaired	8.70	10.33	6.87	16.01	10.03	9.03
Schizophrenic	29.24	27.00	30.25	13.95	26.95	28.30
Impaired	8.88	5.54	8.73	14.86	10.05	8.83
Mean Total	29.24	27.95	28.90	24.97	28.81	28.74
	8.87	8.35	8.76	12.98	7.89	7.97
Mean Medical	31.42	31.35	30.95	29.67	30.05	28.20
	7.21	6.35	7.38	8.44	6.52	7.91
Mean Affective	39.55	24.05	24.87	30.15	30.27	29.22
	9.45	8.78	9.69	7.61	7.86	7.22
Mean Schizo- phrenic	26.75	28.45	30.85	15.10	26.12	28.89
	9.46	8.32	7.79	15.33	8.36	8.83
Mean Non Impaired	29.51	28.73	30.13	25.54	29.41	29.01
	9.50	9.16	7.51	12.65	7.83	7.71
Mean Impaired	28.97	27.16	27.66	24.41	28.21	28.46
	8.30	7.52	9.75	13.40	7.97	8.27

Hypothesis Seven predicted that psychiatric diagnoses will have an effect on patient ratings of certainty of emotional recognition. The analysis of variance discussed above supports this hypothesis, $\underline{F}$ (2, 114) =11.365, $\underline{p}$ < .001. Again, however, this "omnibus" test does not isolate where differences lie.

Hypothesis Eight predicted that schizophrenics will be more certain of the accuracy of their ratings than medical patients. Hypothesis Nine predicted that schizophrenics will be more certain of the accuracy of these ratings than patients with affective disorders. Medical patients produced an average (across all six emotions) total certainty rating of 181.65, with a standard deviation of 24.54. Patients with affective disorders produced a mean certainty total of 168.12, and a standard deviation of 29.34. Schizophrenic patients produced mean total certainty ratings of 156.10 with a standard deviation of 18.80.

A Duncan's multiple range test (ranges =3.70, 3.86, individual comparison, $\underline{p}$ < .01) indicates that these two extreme groups differ from each other at the .05 level or greater. The medical group does not differ significantly from the affective group, nor does the affective group differ significantly from the schizophrenic group. Although one of these hypotheses produced significant results, the results are in the <u>opposite direction from those predicted</u>

<u>by the hypothesis</u>. Individuals without a psychiatric diagnosis rate themselves as most certain of the accuracy of their judgment regarding facial expressions. Those with affective disorders rate themselves as less certain about their judgment. Finally schizophrenics rate themselves as even less certain still.

Hypothesis Ten predicted that there would be a main effect of neuropsychological impairments on the dependent measure of affect recognition certainty ratings. Non impaired subjects had a total mean rating of certainty of 172.33, and a standard deviation of 23.12. Neuropsychologically impaired subjects had a mean total rating of 164.90, and a standard deviation of 29.35. This hypothesis was not supported, $\underline{F}$ (11, 114) = 2.895, $\underline{p}$ < .092.

Hypothesis Eleven predicted a significant interaction between neuropsychological impairment and psychiatric diagnosis on subjective ratings of affect certainty. The 2 X 3 analysis of variance indicates a significant interaction, $\underline{F}$ (1, 114) =3.268, $\underline{P}$ < .042.

Hypothesis Twelve predicted that neuropsychologically impaired schizophrenics would show more subjective certainty regarding judgments of emotional accuracy than non impaired schizophrenics. However, schizophrenics with neuropsychological impairment are <u>less</u> certain than schizophrenics without impairment. Total certainty ratings for schizophrenics without neuropsychological impairment was 158.95

with a standard deviation of 17.13. Schizophrenics with impairments had a mean total rating of 153.25, and a standard deviation of 20.38. However, a univariate analysis of variance failed to find these groups significantly different F (1, 38) = .9167, p < .344. Therefore neither the direction predicted nor the significance of this hypothesis is supported.

Post hoc data analysis is intensely useful, especially since several hypotheses failed to be supported, or were significant in the wrong direction. Table Five indicates the F values for univariate tests on between group differences in ratings of certainty for each emotion, and the significance of the F statistic both before and after the Bonferoni adjustment.

Utilizing the above univariate tests as necessary but not sufficient for the establishment of between group differences for particular emotions, it is seen that differences in certainty of sadness, anger and fear should be examined with a Scheffe test, again chosen because it is an exceedingly conservative measure. For the emotions of sadness and ratings of affect certainty, the Scheffe test (Ranges= 4.79) found that the depressed, non impaired patients rated sad affects as less intense. No other group differences were significant.

Table Five

Analysis of Variance for Post Hoc Univariate Tests of

Emotions and Ratings of Certainty.

Emotion	F	Significance level	Significance with adjusted alpha
Happiness	1.188	.319	no
Sadness	4.144	.002	yes
Anger	5.508	.0001	yes
Fear	9.722	.0001	yes
Surprise	2.774	.021	no
Disgust	.120	.990	no

Univariate Tests with (5, 114) degrees of freedom

For ratings of certainty of judgment regarding the emotion of anger, a Scheffe test (Range= 4.79) found that the depressed, non impaired group differed significantly from the remainders of the groups, and that other groups did not differ significantly.

For subjective ratings of certainty of fear the two schizophrenic groups differed significantly from the remainder of the groups (Ranges =4.79).

Hypothesis Thirteen predicted that the status of psychiatric diagnosis would have an effect on the ratings of perceived pleasantness of facial expressions. A 2 (status of neuropsychological impairment) X 3 (diagnosis) analysis of variance was conducted on the dependent measure of ratings of affect pleasantness. A main effect was found for psychiatric diagnosis, $\underline{F}$ (2, 114) = 7.893, $\underline{p}$, < .001. These results are highly significant.

Table Six shows the mean values and standard deviations for subjective ratings of accuracy by each quasi experimental group.

Table Six

Ratings of Pleasantness by Quasi-Experimental Subgroups

	Emotion Mean and Standard Deviation					
Group	Happy	Sad	Anger	Fear	Sur-prise	Dis-gust
Medical Non Impaired	36.25 4.10	16.90 5.51	13.25 4.78	13.65 4.52	30.25 6.29	14.15 5.45
Medical Impaired	33.10 6.04	19.85 7.61	16.00 9.74	17.45 8.35	28.65 10.36	22.45 9.86
Affective Non Impaired	32.25 4.72	15.90 5.47	15.15 6.03	13.50 6.61	28.50 8.90	15.90 6.40
Affective Impaired	31.80 5.83	19.00 7.13	17.45 9.57	16.75 9.31	27.10 7.94	19.45 7.56
Schizophrenic Non Impaired	32.35 9.32	24.00 8.93	23.00 9.58	14.95 12.65	29.75 10.03	26.00 11.85
Schizophrenic Impaired	32.80 8.25	21.75 9.59	24.45 9.69	11.65 8.99	26.95 10.05	25.10 9.36
Mean Total	33.09 6.78	19.57 7.87	18.21 9.27	14.65 8.81	28.53 8.94	20.50 9.60
Mean Medical	34.67 5.34	18.37 6.72	14.62 7.70	15.55 6.90	29.45 8.49	18.30 8.91
Mean Affec-tive	32.02 5.24	17.47 6.46	16.30 7.98	15.12 8.14	27.80 8.35	17.67 7.14
Mean Schizo-phrenic	32.57 8.97	22.88 9.21	23.72 9.53	13.30 10.96	28.30 10.04	25.50 10.55
Mean Non Impaired	33.61 6.64	18.95 7.63	17.13 8.16	14.03 8.51	29.50 18.45	18.68 9.78
Mean Impaired	32.56 6.94	20.20 8.12	19.30 10.20	15.28 9.12	27.57 9.38	22.33 9.13

Hypothesis Fourteen predicted that schizophrenics would rate slides of facial expression as less pleasant than medical patients rated them. Hypothesis Fifteen predicted that schizophrenics would rate slides of facial expression as less pleasant than patents with affective disorders rated the slides. Both of these hypotheses were tested simultaneously with a Duncan multiple range test (ranges =3.70, 3.86). The results of this test indicate that both the affective (mean =130.97, SD=25.41, and medical patients (mean = 126.40, SD = 18.46) differ significantly from the schizophrenic group (mean = 134.53, SD = 25.16) but not from each other. These results are significant, though in the opposite direction than predicted.

Hypothesis Sixteen predicted that the neuropsychologically impaired schizophrenics would differ in overall ratings of pleasantness compared with non impaired schizophrenics. This was a specific hypothesis that lumped together both pleasant and unpleasant emotions, and predicted a unidirectional effect across each category. For impaired schizophrenics, the mean total ratings of pleasantness of affect were 142.70, with a standard deviation of 27.455. For non impaired schizophrenics, the total mean ratings of certainty were 150.05, with a standard deviation of 26.280. Since this difference is in the opposite direction than it was hypothesized, this hypothesis is not

supported. Differences between the two groups, however, are not significant, F (1, 38) = .745, p < .393.

Hypothesis Seventeen predicted that there would be a significant interaction between neuropsychological impairment and schizophrenia on ratings of pleasantness of facial expression. The interaction was tested by the 2 X 3 analysis of variance and was not significant, F (1, 114) = .115, p < .115.

Hypothesis Eighteen predicted that neuropsychologically impaired schizophrenics would rate affects as less pleasant than non impaired schizophrenics. For the impaired schizophrenics, the mean rating of affect pleasantness was 23.78, with a standard deviation of 4.57. For non deficit schizophrenics, the mean rating was 25.00, with a standard deviation of 4.39. An analysis of variance indicates that these differences, though in the predicted direction, are not significant, F (1, 38) = .74, p < .393.

As in the two previous dependent variables, post hoc analysis is useful in ferreting out the specific types of emotion that are apt to be rated differentially pleasantly or unpleasantly by different quasi-experimental groups. Table Seven shows the result of Bonferoni univariate tests to determine whether a post hoc Scheffe procedure is warranted.

Table Seven

Analysis of Variance for Post Hoc Univariate Tests of Emotions and Ratings of Pleasantness

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Emotion	F	Significance level	Significance with adjusted alpha
Happiness	1.133	.347	no
Sadness	3.166	.010	no
Anger	5.633	.001	yes
Fear	1.232	.300	no
Surprise	.439	.820	no
Disgust	6.235	.001	yes

Two emotions-anger and disgust-are significant at the required .008 level imposed by the Bonferoni adjustment. Post hoc Scheffe analyses were conducted for the effects of belonging to one of the six quasi-experimental groups on the dependent measures of ratings of pleasantness for the emotions of disgust and anger. For the dependent measure of subjective ratings of pleasantness of angry affects, the two schizophrenic groups differed significantly from the medical non impaired group (ranges = 4.79, $\underline{p}$ < .05). Furthermore the neuropsychologically impaired schizophrenic group differed significantly from the non impaired depressed patients. An identical Scheffe test (ranges = 4.79, $\underline{p}$ < .05) was conducted on differences of ratings of pleasantness for disgusted faces. The non impaired schizophrenic group had the highest rating of pleasantness, and it is this group alone that differs significantly from the depressed non impaired group.

Hypothesis Nineteen predicted that an unequal variance would be found between the psychiatric group and the pooled variances of the medical and affective groups. The hypothesis of unequal variance was tested by Bartlett's test for homogeneity, a very sensitive test for heterogeneity of variance that is suited for unequal cell sizes (Winer, 1971). The computation employed was Box's revision, distributed as an $\underline{F}$ ratio. This statistic is significant

when the variances between groups differ, with a level of .05 or less.

The results of this test indicate the variances are not equal, F (1, 119) = 3.213, p < .04. However variance predictions were in the wrong direction. The medical group had a variance of 581.02. The affective disordered patients had a variance of 531.99. The schizophrenic group had a variance of 265.99. Schizophrenics showed less variance in ratings of intensity, not more as was predicted.

Hypothesis Twenty predicted that neuropsychologically impaired patients would rate facial affects as less subjectively intense than non impaired patients. This was tested with a 2 (status of neuropsychological impaired) X 3 (psychiatric diagnosis) analysis of variance. For patients with no neuropsychological impairments the mean total was 27.35 with a standard deviation of 3.29. For patients with neuropsychological impairments the mean aggregate intensity rating was 28.66, with a standard deviation of 4.62. No main effect was found for the status of neuropsychological impairment, F (1,114) = .776, p < .380. Thus this hypothesis is not supported.

Although no prediction was made regarding the status of psychiatric diagnosis, an analysis of variance indicates that this independent variables is significant F (1, 114) = .009, P < .009. The medical patient group demonstrated the highest ratings of intensity (mean = 168.05 SD =

24.01). Affective disordered patients showed the second highest level of intensity ratings (153.40, SD = 23.06). Schizophrenics showed the least subjective intensity ratings (153.42, SD = 16.03). The interaction between diagnosis and neuropsychological impairment was not significant, F (1, 114) = 2.220, p < .113.

Table Eight indicates the ratings of intensity for each emotion by each quasi experimental subgroup.

The suspected finding that schizophrenia has an effect on subjective ratings of intensity can be seen from Table Eight. An admittedly post hoc multivariate analysis of variance was conducted on six dependent variable emotions. This test indicated that there are significant differences between these groups, Hotellings T = .73420, F (30, 537) = 2.628, p < .001. At this point univariate statistics are appropriate to determine where the differences lie.

Univariate F values; levels of significance, and Bonferoni adjustments for these post hoc analyses regarding intensity and particular emotions were also calculated. Since two emotions produce significant differences between quasi-experimental subgroups, a conservative post hoc test again is justified to attempt to isolate the sources of these differences.

Table Eight

Ratings of Intensity by Quasi-Experimental Subgroups

	Emotion Mean and Standard Deviation					
Group	Happy	Sad	Anger	Fear	Surprise	Disgust
Medical Non Impaired	30.05 4.91	23.45 7.98	25.65 10.39	27.65 8.84	28.95 5.87	28.35 6.41
Medical Impaired	30.70 6.50	28.95 8.02	28.75 8.74	28.30 11.31	28.35 8.85	26.95 8.30
Affective Non Impaired	30.95 10.32	25.25 7.96	27.90 7.56	26.60 9.45	27.70 5.98	25.50 6.75
Affective Impaired	29.20 7.56	20.30 6.51	24.95 6.21	26.20 11.01	29.10 8.00	23.10 8.25
Schizophrenic Non Impaired	31.80 7.65	29.25 6.24	30.45 6.73	14.05 12.75	26.45 8.13	24.95 8.49
Schizophrenic Impaired	29.20 6.57	28.50 7.31	27.50 7.37	11.50 11.27	27.90 8.05	25.30 7.42
Mean Total	30.31 6.84	25.95 7.93	27.53 8.00	22.38 12.66	28.07 7.47	25.70 7.66
Mean Medical	30.37 5.70	26.20 8.38	27.20 9.61	27.97 10.02	28.65 7.42	27.65 7.35
Mean Affective	30.07 7.71	22.78 7.60	26.42 6.99	26.40 10.12	28.40 7.05	24.32 7.54
Mean Schizophrenic	30.50 7.17	28.88 6.73	28.98 7.13	12.77 11.95	27.18 8.02	25.12 7.87
Mean Non Impaired	30.93 6.89	25.98 7.71	30.13 7.51	22.76 12.04	27.70 6.70	26.28 7.35
Mean Neuro Impaired	29.70 6.81	25.91 8.23	27.66 9.75	22.00 13.34	28.45 8.21	25.12 8.02

For the emotion of fear, a Scheffe test (ranges= 4.79) found significant differences between the two schizophrenic groups and the remaining four groups at the .05 level. The schizophrenic groups had a pooled mean intensity ratings for the emotion of fear of 12.77, with a standard deviation of 11.95. The nonschizophrenic groups had a total mean rating of 27.18, with a standard deviation of 10.07.

The individual differences in group ratings regarding the intensity of the emotion of sadness is even more unexpected. An additional Scheffe test (range = 4.79) was conducted on the mean differences between groups for this emotion's ratings of intensities. The depressed, neuropsychologically impaired group differed significantly at the .05 level from the schizophrenic groups and the medical neuropsychologically impaired group.

CHAPTER VII

DISCUSSION

The Role of Neuropsychological Deficits in Schizophrenic Errors in Affect Recognition

The major purpose of this dissertation was to determine whether facial affect recognition deficits found in schizophrenics could be accounted for by neuropsychological impairments. In this study schizophrenia and neuropsychological impairment both were found to have a significant impact on patients' ability to correctly decode facial expressions, as previous literature has also found. However, there was neither an additive nor an interactive effect on the task of facial affect recognition in the schizophrenic group for the variable of neuropsychological impairment. Schizophrenics made the same number of errors whether they were neuropsychologically impaired or not.

If affect recognition deficits were entirely due to latent neuropsychological impairment, then the schizophrenics without such impairment should not have shown affect recognition deficits. Furthermore, if affect recognition deficits were entirely due to latent neuropsychological

impairment, then schizophrenics without neuropsychological damage should have shown less affect deficits than brain damaged individuals either with medical or affective disorders. Neither result was found.

Instead, neuropsychologically impaired schizophrenics performed no worse than non impaired schizophrenic subjects on a task that is known to be highly influenced by neuropsychological deficits. Viewing this finding from a different angle, it can be claimed-tongue-in-cheek, certainly-that schizophrenia provides immunity to the effects of neuropsychological impairment on tasks of facial affect recognition!

Potential Validity Problems

The most common response when predictions go grossly contrary to hypotheses is to suggest measurement error, sample error, or other factors influencing the results in the unexpected direction. Cook and Campbell (1979) suggest such discussions be framed in the language of three types of validity: internal, external, and statistical conclusion validity. Internal validity refers to spurious manipulations that impute causality where none is really present (Type I error), or more rarely obscures causality (TYpe II error). Threats to internal validity are caused by such phenomena as maturation, subject attrition, changing instrumentation and statistical regression.

One exception discussed by Cook and Campbell, possibly causing a Type II error, is the so called "ceiling" or "floor" effect. It is possible that the performance of schizophrenics was so poor that even with the main effect of neuropsychological impairment actually being present, test instrumentation was not sufficiently strong to override the decrement associated with schizophrenia.

The possibility of this threat to internal validity existing can be assessed by utilizing the techniques of meta-analysis (Wolfe, 1986). A coefficient <u>delta</u> (Cohen, 1977) can be calculated for the difference between schizophrenic and non schizophrenic performance on facial affect recognition[8]. For this study the differences between the medical control group and schizophrenic patients was <u>d</u> = .73. This means that on average the medical group performs .73 standard deviations better than the schizophrenic group. (According to Wolfe this is a moderate-to-above-average effect for social science literature).

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[8] The formula for this is: <u>delta</u> = mean group 1 - mean group 2/ pooled standard deviation. This statistic gives us a useful comparison tool to view diverse studies conducted separately, and is interpretable as a measure of differences in standard deviations. Meta-analysis is a relatively new procedure (Wolfe, 1986), and alternative meta-analytic statements of effect size exist. Rosenthal, for example, utilizes the coefficient <u>r</u> rafter than <u>delta</u>. Some meta-analyses do not use pooled standard deviation estimates, and instead use the standard deviation of the control group. In the author's experience this usually results in an inflated effect size where homogeneity of variance is not a given.

However, it does not approximate a floor effect. A meta-analysis by Johnson and McCown (submitted) suggests such an effect size is relatively low for the type of task at hand. Typically, the effect size for schizophrenia on a task of facial affect recognition is above one standard deviation. For nonmedicated schizophrenics the effect size is often over three standard deviations, i. e. $\underline{d} = 3.00$, or four times that found in this study. Results in this study are on the low end of the meta-analytic distribution. Obviously, no floor effect was operating as past studies have found much greater effect sizes associated with schizophrenia.

Perhaps the lack of magnitude of the effect size is indicative of an instrumental insensitivity that failed to allow for an interaction between schizophrenia and neuropsychological deficits. Only 36 affects were presented. Some studies on affect recognition utilize three or four times as many stimuli. It is possible that affect recognition deficits are a function of test length as well as complexity. Ability to decode affect might be a relatively exhausting task for those with brain impairments, and more of a main effect might be encountered on longer tests.

Whether this argues for greater validity of longer affect recognition tests is uncertain. It should, however be noted that facial affect recognition is more impaired in "acute schizophrenics" and those not medicated. Johnson and McCown (submitted) have shown that there is a significant

correlation between chronicity and <u>reduced</u> effect size. The values in this study are not as atypical if it is remembered that all of the patients in the schizophrenic groups were inpatients at a state hospital and heavily medicated. More will be said about this below in the discussion of external validity.

Internal validity is also threatened when an unmeasured variable differentially represented in a group of interest has effects on the dependent measure that obscure those of the hypothesized independent variable. It is also possible that affect accuracy-apart from any brain deficits-is a practiced skill correlating with social network intensity or size. Schizophrenics probably offer very little socially, and would be expected to have few friends. Consequently, they would be expected to have the smallest, most insular networks, and might simply be out of practice for the ability to decode expressions. In this explanation the lack of social contacts would decrease opportunities for social learning. Prior neurological impairments in affect recognition would be completely overridden by the lack of practice due to no social contact. In this manner, neuropsychological deficits might have had a main effect, but the main effect would be overshadowed by the lack of social practice and learning opportunities.

This is an intriguing line of reasoning, because it has potential importance for rehabilitation of schizophrenics.

First, it argues for the greater needs of half-way houses and other socialization programs for chronic schizophrenics who might benefit in their overall social skills functioning from simply being in the presence of other people. Secondly, it argues that neuropsychological deficits might still play an important role in comparative affect recognitions skills, though one that is overridden by the confounding variable of lack of practice.

In this study two measures of social network were utilized, one of status of marriage, and the other of the number of people living with the patient.[9] While the status of being married failed to correlate with affect recognition (r = .113, p < .112) the number of people living in one's household was almost significantly correlated, r = .143, p < .053. The failure to find significance here is not particularly disheartening to those advocating the role of social support in affect recognition. The measures of social support employed in this study are very crude, and absolutely nothing is known about their validity apart from what is obvious at face value.

A fruitful area of research might be to further assess the impact of such variables on affect decoding utilizing more sophisticated measures of social support. However, even

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[9] Since many of the subjects were old and poor, this number is rather large in some cases. Differences in cultural norms between middle class Northern, urban families of majority cultures, and the rural poor should be recalled.

if social support were positively related to affect recognition, causality would be hard to sort out. It might be that those with no social skills find no one willing to live with them. On the other hand, an equally strong case can be made that individuals who do not live with significant others lose the ability to decode affects from lack of experience. Only a longitudinal study addressing changes in affect recognition and social support as a function of disease process can adequately answer this question.

Cook and Campbell also discuss a general category of validity known as <u>statistical conclusion validity</u>. According to Cook and Campbell the first relevant issue regarding statistical conclusion validity involves statistical power. Insufficient power can threaten statistical conclusion validity by causing Type II error. In this study the test utilized to compare differences between groups was a factorial analysis of variance, a powerful test for detecting differences (Winer, 1971). Furthermore, ANOVAs are relatively robust regarding violations from the normal distribution, another cause of statistical conclusion validity problems.

Reliability is another relevant threat to statistical conclusion validity. Reliability of diagnostic categories was maximized by having multiple criteria in the operational definitions, such as by having a diagnostic <u>team</u> agree on DSM-III-R categorization, and by insistence on both

laboratory and psychometric data regarding neuropsychological deficiencies. Reliability of measurements was attempted by utilizing scales with a high degree of formal psychometric reliability.

Another cause of statistical conclusion validity problems is labeled by Cook and Campbell (1979) as "random irrelevancies in the experimental setting". To some extent this problem was controlled by utilizing two diagnostic groups of psychiatric patients (affective disorders and schizophrenics). Testing was done in the same locations for each group. Yet care should be taken in interpreting these results. Schizophrenics are notoriously difficult to test, especially while sufficiently impaired to require an inpatient, acute treatment milieu. Attentional factors operating in the schizophrenic groups could have interacted with unknown confounds in the environment to distort any possible findings.

An additional statistical conclusion validity problem exists regarding hypothesis concerning affect pleasantness, certainty and intensity. In retrospect the procedure utilized allowed for a tremendous amount of error variance. This procedure used aggregate measures of these variables as comparison units, since directionality was hypothesized. The problem with this measure is that it may have failed to find more subtle differences.

An example is appropriate. If particular slides were rated as "2" and "7" by normal patients, and "7" and "2" by schizophrenics, individual differences cancel out with the method employed. A statistical analysis that utilized absolute value might have highlighted differences, although it also would have made directionality harder to study, perhaps requiring a dichotomously coded statistical test which would have been less powerful. With hindsight as experience, power to detect differences might have been more important than power to detect directionality of differences.

A number of threats to external validity also exist in this study. The overall low intelligence level of this sample might have depressed levels of significance for any of the affect recognition variables. Certainly, this sample is atypical. It was drawn from one of the most economically depressed communities in the country. A cycle of poverty and lack of educational opportunity is chronic. Poor nutrition and lack of prenatal and other medical care may operate to reduce potential intellectual acumen of both medical and psychiatric patients utilizing hospital services. As many as 75% of patients at this sample site are "functionally" illiterate, being limited to fourth grade reading skills or less. Many of the patients tested were severely ill physically, which may have also reduced the effect size.

It is also notable that most subjects were minorities and the researcher is a member of the dominant culture. This might have affected the study results, increasing error variance, and hence reducing sensitivity of the instruments employed. Rosenthal et al. (1979) have commented on the confusion in the literature regarding whether minorities can detect nonverbal affects of dominant cultural members as well as they can members of their own cultures. In all likelihood, there are great individual differences in the application of this skill by minorities across situations. These differences would likely be due to specific ideographic historical factors, and atypical lifetime opportunities, such as employment patterns, neighborhoods lived in, successful and pleasant experiences with members of majority cultures, etc.

Such uncontrolled potentiality could easily increase the error variance and decrease any differences between groups likely to be found. A weakness of this study was not replicating it with diverse cultural groups, or splitting the sample into different socioeconomic groups. On the other hand both subject race and socioeconomic status failed to correlate with any of the dependent variables.

Alternative Explanations

A less cultural and more neurological answer regarding the failure to find a main effect for neuropsychological

impairment may also be appropriate. This argument is a bit more speculative, but finds some support in the literature. It suggests that several disparate areas of the brain may be involved in neural processing of affects. The nondominant hemisphere might be <u>necessary</u> for primary reception, but <u>not</u> <u>sufficient</u> for sensory integration and the establishment of meaning.

Luria (1963) has essentially solved the equipotentiality vs. localization controversy by demonstrating that diverse and disproximate neural locations combine together to form functional systems. For example, Luria has shown that while it is true that voluntary motoric movement involved in writing may localize to the primary motor strip, afferent and efferent neurons connect with areas as diverse as the cerebellum, the occipital lobes, Broca's area, and the right parietal areas. Damage to any of these areas can destroy the functional system, even if the integrative and executive areas-the tertiary areas in Luria's system-remain intact.

It is possible that some of the primary or subintegrative areas of affect processing involve neuropsychological functioning other than in the right hemisphere, the typical area suspected of localization of facial affect recognition (Cicone, et. al, 1980). This might be especially true when the experimental task involves a verbal labeling of the affects presented. There is some evidence for this diverse

lateralization and localization argument. Dekosky et al. (1980) found that while not as impaired as right hemispheric brain damaged patients, left hemisphere damaged patients were significantly more impaired on two tasks of facial affect recognition than neurological patients utilized as controls. From this it seems that both hemispheres are important for correct facial affect decoding, and not only the right hemisphere, as some authors has assumed.

If this is the case then <u>accurate transmission between hemispheres</u> would be essential for correct decoding of facial expressions. Damage to either hemisphere <u>or to connecting fibers</u> (the corpus callosum), could cause deficits in affect recognition. This point seems to have been missed by previous researchers.

During the review of the literature the hypothesized relationship between schizophrenia and corpus callosum deficits was discussed. As noted earlier, Bigelow, Nasrallah, and Rauscher (1983) have presented autopsy evidence suggesting that the corpus callosa of schizophrenics is often enlarged. As Andreasen (1985) argues, aberrant corpus callosa would result in a case where the two hemispheres were not able to communicate well with one another, resulting in a decay of information processing en route to or from the dominant hemisphere. Similar arguments about the necessity of correct hemispheric synchronization are advanced by Green, Hallet, and Hunter (1983).

In the case of schizophrenics without noted neuro-psychological impairment <u>but with interhemispheric process-ing difficulties</u> the <u>reception</u> of appropriate facial expressions might be possible, as well as the preliminary decoding and pattern analysis that appears to be a right hemispheric task. However, <u>integration and interpretation</u> of these emotions might be absent. This deficit would tend to overshadow any additional receptive or localized deficits, particularly those involving right hemispheric functioning. The trickle of water caused by brain damage would be ir-relevant because the damage of schizophrenia has destroyed the pipelines.

Since none of the neuropsychological tests administered were particularly sensitive to interhemispheric communica-tion, this theory has additional credence. If schizophre-nia is related to difficulties in interhemispheric process-ing, subjects who show more deficits on cross-modality neuropsychological tests, for example, on dichotic listening procedures, (Green, Hallet & Hunter, 1983) should show more deficits in affect recognition. This is an interesting hypothesis that remains to be tested.

Furthermore, the measure of affect recognition in this study involved verbal matching of affects with names. This is a left hemisphere function that might be more disturbed in schizophrenics if corpus callosum deficits are present. Right hemispheric <u>understanding</u> of a particular stimulus

might well be accomplished, but the right hemisphere might not be able to communicate this information to the left hemisphere in an effective and timely manner. In this way, tasks that did not involve verbal labeling of emotions, but that did involve emotional recognition might be performed better by schizophrenics than those that did involve verbal labeling. Procedures such as those utilized by Mandal (1986) are an example of such affect recognition tasks that do not involve verbal labels.

A very interesting experiment is suggested by the above arguments. If difficulties in interhemispheric message transmission are responsible for comparative deficits in schizophrenics' inability to decode facial affects then schizophrenics without neuropsychological deficits should be more aware, on some level, of which affects they were seeing. They might not be able to communicate this aware-ness. They might not even be aware of their understanding. They would be similar to the "split brain" patients studied by Sperry (1968) who point to an object they have seen before but cannot identify it verbally or admit that they were aware of their knowledge.

However, schizophrenics with brain damage, particularly right hemispheric damage, would show less of this decoding skill on nonverbal tasks. Furthermore, they might be expected to show less Galvanic Skin Responses in the presence of affects for which they have been reinforced

previously. This might be true even though their total recognition scores on tasks involving verbal categorizing was no worse than the previous group of schizophrenics.

There is an extensive clinical lore regarding the supposed nonverbal sensitivity of schizophrenics. Arguments of this type state that schizophrenics are imbued with unusual emotional discernment regarding the feelings of others, the stress of which might, in fact, be responsible for their psychosis. While this argument is unsupported in the literature, it might have an interesting corollary with the present research.

If schizophrenics are "aware" of emotional affects in one hemisphere, yet are unable to act fully upon this awareness, a good deal of social frustration might be encountered. This might lead to social avoidance often characteristic of this disorder, as well as the suspicious-ness of others often seen. It is possible that in social situations the schizophrenic gets a set of signals from his right brain, and he is unable to verify the reality of their content, or even fully verbalize them. He is left with a weird feeling of anomalous dread, with occasional intrusions from apophanous social thoughts that have no way of being verified in reality.

Affect Certainty

Regarding affect certainty, the effects of schizophrenia were in the opposite direction than those predicted. The findings here contradict a pilot study which found that outpatient psychiatric subjects rated expressions as more certain about their affect choices if they had a higher "P", or psychoticism factor. For this entire sample, Psychoticism correlates -.182 ($\underline{p}$ < .02) with ratings of affect certainty. Eysenck and Eysenck (1985) have argued that P is the original diathesis for schizophrenia, although not the precipitant or a direct measure of the disease. Through a pathological process not known at this time, individuals high on P apparently are also at risk for schizophrenia and manic depressive illness. Either another gene, a pathological agent, or a psychic trauma might be necessary to put those at risk due to high P over the edge towards diagnosable schizophrenia. Outpatients who might have been high P but were not schizophrenics might demonstrate affect certainty due to factors associated with psychoticism as a variable of personality. Schizophrenics, who also have high P, might demonstrate affect uncertainty, due to specific neurological problems secondary to the onset of their disease.

One solution for this contradiction is that P might not be as relevant to schizophrenia as other variables, a

criticism leveled by Block (1977) and others who believe it is simply a genetic covariant associated with downward drift encountered in mate availability by schizophrenics. This argument states essentially that criminality and interpersonal hostility associated with P places people in an economic disadvantage, where they frequently intermarry with individuals who are genetically loaded for schizophrenia. On the other hand, the second psychoticism measure derived from the Whitaker Index also correlated negatively with affect certainty, (r =-.156, p < .044). This is a direct index of schizophrenic thinking and its correlation with P (r =.421, p < .001) lends support to the Eysenck's theory of a normally distributed trait being responsible for schizophrenic diathesis. It also suggests that schizophrenic symptoms, as measured by the Whitaker Index, is associated with less affect certainty. These findings are in agreement with Livesay's (1981), who found that schizophrenics were much less certain of social judgment than non schizophrenics.

The obvious suggestion is that in addition to a high P factor, to become schizophrenic an individual also needs to have a deficit in interhemispheric communications. Interhemispheric difficulties could also cause a situation where individuals were less certain of their judgments of emotions. High P in individuals who are not schizophrenic may correlate with affect certainty, while inter hemis-

pheric discoupling (which is often associated with high P) might be related to less affect recognition certainty. Another explanation might be that P is curvilinearly related to certainty of affect recognition.

Perhaps under the loci of a specific gene responsible for turning on the full diathesis of P (Eysenck & Eysenck, 1985) the toughmindedness of this variable translated into an opponent process following repeated instances of stimulus presentation. Evidence regarding this possibility has been furnished by Claridge (1981). Claridge has iden- tified the "phenomena of reverse covariation" in in- dividuals, including schizophrenics who score high on the Eysencks' P scale. Claridge provides evidence that the psychophysical properties of Psychoticism are related to the tendency of the high P individual to form a stimulus discoupling characterized by essentially a reverse physiolo- gical response to that evoked in the individual upon initial stimulus presentation. Low P individuals habituate to loud stimuli following repeated presentations, whereas high P individuals show indices of relaxation below prestimulus baseline. It is possible that given the repetitive nature of the task at hand, subjects with extremely high P began to form opponent responses to those initially voiced, another manifestation of the "reverse covariation" phenomena. More research is needed to uncover the relationship between P and the tendency to form opponent responses.

Still another possibility is that the medications involved or the social stigma of being in a hospital weakened the feelings of certainty regarding affect recognition in individuals who would otherwise be more certain and toughminded regarding their judgments. This will be discussed below.

It is surprising that neuropsychological impairment had no effects on certainty. The interaction found was in the wrong direction. The most obvious explanation here is that certainty is related more to premorbid personality factors and situational settings than any neuropsychological function that was assessed with this battery.

An interaction was found between brain damage and schizophrenia on the variable of affect certainty. The interaction was in the wrong direction from that predicted. No immediate explanation is indicated in the literature. Perhaps the causal agent associated with this interaction, and even main effect is not neuropsychological or schizophrenic damage but the loss of confidence that comes from being hospitalized and stigmatized. Neuropsychologically impaired schizophrenics and those who had been in the hospital for longer periods of time may have absorbed feelings that they are incompetent or otherwise incapable of adequate reality testing. Consequently they would be reacting in response to the demand characteristics of their community.

One variable in this study supports this line of reasoning. The number of times an individual had been psychiatrically hospitalized was a significant covariant in a post hoc ANOVA measuring the effects of quasi-experimental group membership on affect certainty, F (1, 110 = 6.067, p < .015. However, two problems arise with direct interpretation of this covariance. First is its obvious ex post facto discovery, and as such needs the appropriate suspiciousness afforded to such findings until it is replicated in a study where it is predicted in advance. The second is that it is reasonable to assume that the number of times persons have been hospitalized is linearly related to the severity of their schizophrenia. However, the role of social support and "institutionalization" remain topics worthy of further research.

Unfortunately, this study did not include sufficient numbers of "acute" schizophrenics to compare the length of hospitalization with affect recognition or certainty variables. Current psychiatric nomenclature (DSM-III-R, American Psychiatric Association, 1987) has eliminated the diagnosis of acute schizophrenia. By definition such patients (who are now labeled as brief psychotic reaction or schizophreniform disorder) are not included in this study. They simply do not fit the definitional criteria.

The hypothesis that affect certainty is related to social support variables is not, however, supported by the post hoc

analysis of the data available in this study. Marital status failed to correlate significantly with affect certainty (r= .008, P < .455). The number of people in a patient's household also failed to correlate significantly with affect certainty (r = -.205, p < .392). These are extremely crude measures of social support, and clearly more exacting, and perhaps multidimensional assessment might be of use.

Affect Pleasantness

The next variable studied was affect pleasantness. A main effect was observed for psychiatric diagnosis, although it was in the opposite direction from that predicted. Implicit in the hypothesis was a theorized component similar to psychodynamic projection. The assumption was that the schizophrenic subject feels hostile and suspicious towards the world, and consequently rates social stimuli more negatively and less pleasantly across all situations.

Perhaps what is occurring is a reverse situation where the individual compares outward stimuli to his own state. Rather than project, schizophrenics might compare. In this process, an internal standard of self-reference is implicitly utilized in the judgement of the external world. According to this theory, by comparison with more normal individuals schizophrenics would tend to rate affects more pleasantly, since their own point of comparison-their own internal experiences-are elevated in the direction of

unpleasantness. It is not denied that projections occur, but that under some circumstances, judgement by self-reference provides for social assessments antithetical to a person's states or traits. This is an interesting area of research that could easily be expanded by the utilization of induction of mood and ratings of affect pleasantness.

An additional look at the data in the study suggests another explanation for why the hypothesis was not supported. Schizophrenics fail to "adjust" their perceptions of pleasantness for the emotions of anger and disgust. As the post hoc tests indicated, these emotions are rated differently by the quasi-experimental groups. In the schizophrenic groups there is a lack of discrimination of these unpleasant emotions. These unpleasant emotions are rated as pleasant as other emotions are by schizophrenics, whereas non schizophrenics seem to make more of a differential response.

Whether this is due to response set, attention, or dynamic factors is unknown. However, the idea that schizophrenia is associated with a total rejection of the dimension of unpleasantness is not supported, since they seem to rate fear as reasonably unpleasant. Ratings of pleasantness of the emotion of fear do not differ significantly from those made by the other diagnostic groups. In fact, schizophrenics rated fear as the least pleasant of all of the groups, although differences were not significant.

Perhaps because of their personal experiences with fear they related it as less pleasant than those whose experience is more vicarious.

To some extent these findings contradict those of Mandal (1986). Mandal had schizophrenics and normal patients rate affects on a multidimensional scaling task that avoided direct verbal labels, Dimensions were collapsed through factor analysis. The pleasantness dimension was absent for schizophrenics. The nonschizophrenic groups failed to demonstrate the collapsing of this dimension. Mandal concludes that schizophrenics are markedly less aware of the pleasantness/unpleasantness distinction, at least in a multidimensional scaling procedure. The present findings suggest that schizophrenics are rejecting, or nondiscriminatory of <u>unpleasant</u> emotions only. This implies perceptual awareness, and probably unconscious rejection, an altogether different finding than Mandal's claim that schizophrenics attenuate this dimension.

The key to solving this apparent contradiction would seem to be in the method employed. In this study, as well as most studies on affect recognition, clear anchoring phrases and faces are presented. In Mandal's study the multidimensional scaling did not utilize these verbal cues. It is quite possible that in the absence of external cues, schizophrenics rate facial expressions as less pleasant, or

even ignore the emotional dimension associated with hedonic responses altogether.

However, in the presence of external cues-and perhaps the fact of hospitalization-schizophrenics overcompensate. It is even possible that in the absence of clear cues projection is utilized, while in the presence, subject self reference is evoked. In social psychological literature, a consistent trend has emphasized the important of verbally mediated responses to environmental cues in determining attitude change (Petty & Cacioppo, 1981). "Cognitive response theory" realizes that different processes of attitude change exist. Making people think about a topic-even by drawing attention to it discretely-produces a different degree of attitude formation than if internal verbalizations are not present. This is probably true for affect recognition and ratings of affect qualities as well. Introducing verbal categories and choices forces a more conscious process onto the task that might not have been present otherwise.

Neuropsychological impairment had no effect on affect pleasantness in this study. This is surprising. The relationship between affect pleasantness and impairment was suspected of being maximized by individuals with right hemispheric damage who generally display "a behavioral mosaic of mood lability, dysthymic neurosis, and depression " (Fromm-Auch, 1983, p. 83). This is in contrast to patients

with left hemispheric damage who are more likely to show pathological ebullience.

It is possible that since the number of focal lesion patients with a clear right or left lesion were approximately, the same, differences cancelled out. To test this hypothesis a Kruskal-Wallis analysis of variance was conducted on the 22 patients with focal lesions. Of these nine were right hemispheric, seven were left hemispheric, and the remainder were frontal lobe disorders. Differences were not significant, F (2, 21) = .546, p < .56. This of course simply could be due to the small sample size employed.

Internal validity of the present findings may be compromised by what Cook and Campbell (1979) have labeled "method bias". Etcoff (1983) found that left hemispheric brain damage produced impairment in perceptions of affect pleasantness. Her method employed was a multidimensional scaling task without verbal referents. Again, it is possible that without such anchor points brain damaged subjects-at least those with left hemispheric damage-minimize the pleasantness of affects presented operating on projection rather than social context or comparison. Perhaps the left hemisphere or the frontal lobes acts as a check, integrating social desirability factors and obvious stimuli context into a more pleasant oriented perception.

If this is the case for either schizophrenics or neuro-psychologically impaired individuals we might find a social-

evaluative component "censoring" initial perceptions. This hypothetical process could best be examined by a twofold experiment which had subjects make multidimensional judgments of pleasantness, without verbal referents and then had them perform more left hemispheric, integrative task of rating pleasantness directly and verbally. It would be expected that schizophrenics and subjects with brain damage would show more differences between these two measures (less correlation) than other subjects. Again, this is an interesting area for additional research.

Affect Intensity

The final variable examined in this study was affect intensity. It was expected that schizophrenics would behave more idiosyncratically. This was not supported by the findings. Schizophrenics rated affects as less intense than medical patients or affective (depressed) patients. Furthermore, they showed less variance in ratings.

These findings are in line with those of Claridge (1981), who found that schizophrenics attenuate portions of stimuli. Initially they appear to contradict other general findings in the literature on schizophrenics. An example is the extensive work of Mednick (1974), who presents convincing arguments that schizophrenics are characterized by a

condition of overarousal.[10] On a multidimensional scaling task, Mandal found that schizophrenics were overly sensitive to the dimension of arousal or intensity. Again, this would seem to be a contradiction between multi-dimensional scaling methods and category presentation methods, such as the one employed in this study.

The major source of the difference between schizophrenic and nonschizophrenic groups in this study, according to post hoc tests, was with the emotion of fear, which schizophrenics did not see as intense at all. A similar phenomena is found with the neuropsychologically impaired depressed, who did not see sadness as an intense emotion. An obvious hypothesis for further testing is that schizophrenics, and perhaps to a lesser extent, depressive patients, block out or repress the intense emotions of others in their environment that would tend to magnify their own negative states. Because schizophrenics are overaroused, they attenuate. For schizophrenics, fear is overwhelming, all other emotions pale by comparison. Consequently, they reject the emotion.

This hypothesis can be synthesized with those of Mandal, who believes schizophrenics are overly dependent upon the arousal dimension for affect categorization. Perhaps because they are internally hypersensitized to this dimension, they tend to reject obvious stimuli that are intense. Perceptual

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[10] However, Mednick's sample was obtained prior to DSM-III, when the definition of schizophrenia has changed.

acknowledgement and cognitive processing of these stimuli would place them at a much greater arousal level. Evidence regarding this was furnished by Doughtery, Bartlett, Izard (1974). These authors found that schizophrenics were particularly upset after viewing angry or fear facial expressions. Apparently, normal subjects can look at an angry face, and say, "Yes, it is aroused and intense". If schizophrenics-who are very sensitive to this emotional dimension-did so their naturally aroused conditions would become overstimulated.

Another explanation is also possible. What appears to be happening in these situations is that judgments of intensity are clearly made on the basis of comparison of internal states, rather than as projections. The groups that were the most fearful-the schizophrenics-minimized the intensity of the affect of fear. The group that was probably the saddest-the patients with depression and strokes, or depression and early onset dementia, also "knew" what intense sadness was like. And they grasped the fact that the pictures they were seeing-intended to invoke an intense affect with which they were intimately familiar-failed markedly at this task compared to their own depths of feeling.

Further testing of the hypothesis that verbal mediation invokes internal comparison, while nonmediation involves projection could be accomplished with patients on these extremes. Depressed neuropsychological impaired patients and

schizophrenics could be given a multidimensional scaling test to determine whether they accentuate or minimize the dimension of intensity for either sadness or fear respectively. Standardized scores could then be compared with performance on a task where verbal labeling or an anchor point is involved. It would be expected that for tasks not involving verbal labeling, the dramatic rejection of intensity for these two affects would not likely be found.

Conclusion

One of the major goals of this study was to demonstrate that some of the social deficits schizophrenics display were directly explainable by neuropsychological knowledge presently within our arsenal. However, this was not the case. Like most studies, this one invoked more questions than it answered.

Affect recognition is a complex, multivariate phenomena with results clearly dependent not only upon what type of questions are asked, but what tools are used to answer these questions. Probably, the most important conclusion of this study is that the study of how well different diagnostic groups decode facial expressions can generate many fascinating testable hypotheses regarding brain functioning, perceptual processes and the nature of mental illnesses. It is hoped that the complexity of this area will not serve to discourage future interest.

REFERENCES

Abelson, R., & Sermat, V. (1962). Multidimensional scaling of facial expressions. _Journal of Experimental Psychology, 63_, 546- 554.

Allport, F. (1924). _Social Psychology_. New York: Holt.

American Psychiatric Association. (1987). _Diagnostic and statistical manual, revised_ (3rd. ed). Washington: American Psychiatric Association Press.

Andreasen, N. (1985). Structural brain abnormalities in schizophrenia. In M. Menuck & M. Seeman, (Eds.), _New perspectives in schizophrenia_ (pp. 43-60). New York: Macmillan.

Bell, C. (1806/1928). _Anatomy and physiology of expression_. Oxford: Oxford Press.

Bigelow, L., Nasrallah, H., & Rauscher, F. (1983). Corpus callosum thickness in chronic schizophrenia. _British Journal of Psychiatry, 142_, 284-287.

Block, J. (1977). The Eysencks and psychoticism. _Journal of Abnormal Psychology, 86_, 653-654.

Bruner, J. & Tagiuri, R. (1954). The perception of people. In G. Lindzey, (Ed.), _Handbook of social psychology, Vol. 2_. (pp. 323-355). New York: Colliers.

Cicone, M., Wapner, W., & Gardner, H. (1980). Sensitivity to emotional expressions and situations in organic patients. _Cortex, 19_, 145-158.

Claridge, G. (1981). Psychoticism. In R. Lynn (Ed.), Dimensions of personality (pp. 79-109). London: Pergammon Press.

Cook, T., & Campbell, D. (1979). Quasi-experimentation: design and analysis issues for field settings. Boston: Houghton Mifflin.

Critchley, M. (1953). The parietal lobes. London: Arnold.

Cutting, J. (1981). Judgment of emotional expressions in schizophrenics. British Journal of Psychiatry, 139, 1-6.

Darwin, C. (1872). Expressions of the emotions in man and animals. London: John Stouton.

Davitz, J. (1964). Communication of emotional meaning. New York: McGraw-Hill.

Dekosky, S., Heilman, K., Bowers, D., & Valenstein, E. (1980). Recognition and discrimination of emotional faces and pictures. Brain and Language, 9, 206-214.

Dewolfe, A., Barrell, R., Becker, B., & Spaner, F. (1971). Intellectual deficit in chronic schizophrenia and brain damage. Journal of Consulting and Clinical Psychology, 36, 197-203.

Doughtery, F., Bartlett, E., & Izard, C. (1974). Response of schizophrenics to expressions of the fundamental emotions. Journal of Clinical Psychology, 30, 243-246.

Duncan, N., & McCown, W. (submitted). Psychoticism and certainty of emotional judgment. Tulane University, Department of Psychiatry and Neurology.

Ekman, P. (1980). <u>The face of man</u>. New York: Garland STPM Press.

Ekman, P. (1982).<u>Emotion in the human face</u>. New York: Cambridge University Press.

Ekman, P., & Friesen, W. (1976). <u>Basic Affect Recognition Test</u>. San Diego: Consulting Press.

Ekman, P., Friesen, W., & Ellsworth, P. (1972). <u>Unmasking the face: a guide to the research of facial expression</u>. New York: Academic Press.

Etcoff, N. (1983). Hemispheric differences in perception of emotion in faces. Doctoral dissertation, Boston University.

Exner, J. (1978). <u>The Rorschach: a comprehensive system</u>, New York: Wiley.

Eysenck, H. & Eysenck, S. (1976). <u>Psychoticism as a dimension of Personality</u>. London: Hodder & Stoughton.

Eysenck, S., Eysenck, H., & Barrett, P. (1985). A revised version of the psychoticism scale. <u>Personality and Individual Differences</u>, 6, 21-29.

Eysenck, H., & Eysenck, M., (1985). <u>Personality and individual differences: A natural science approach.</u> New York: Plenum.

Feinberg, T., Rifkin, A., Schaffer, C. & Walker, E. (1986). Facial discrimination and emotional recognition in schizophrenia and affective disorders. <u>Archives of General Psychiatry</u>, <u>43</u>, 276-279.

Feleky, A. (1928). Feelings and emotions. New York: Pioneer Press.

Fernberger, S. (1928). False suggestions and the Piderit model. American Journal of Psychology, 40, 562-568.

Folstein, M., Folstein, S., & McHugh, P. (1975). Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. Journal of Geriatric Research, 12, 189-199.

Franzen, G., & Ingvar, D. (1975). Abnormal distribution of cerebral activity in chronic schizophrenia. Journal of Psychiatric Research, 12, 199-218.

Frijda, N. (1958). Facial expression and situational cues. Journal of Abnormal and Social Psychology, 57, 149-153.

Fromm-Auch, D. (1983). Neuropsychological assessment of de-pressed patients before and after therapy: clinical profile interpretation. In P. Flor-Henry & J. Gruzelier (Eds.), Laterality and Psychopathology (pp. 83-102). Amsterdam: Elsevier.

Goldberg, T., Weinberger, D., Berman, K., Pliskin, N., & Podd, M. (1988, January). Prefrontal-type cognitive dysfunction in schizophrenia. Paper presented at the 16th annual International Neuropsychological Science Association, New Orleans, Louisiana.

Golden, C. (1978). Stroop color word test: a manual for clinical and experimental uses. Chicago: Stoelting Company.

Golden, C. (1981). <u>Diagnosis and rehabilitation in clinical neuropsychology</u>. Springfield, Illinois: Charles Thomas.

Golden, C. (1987). <u>The brief Luria-Nebraska neuropsychological battery</u>. Los Angeles: Western Psychological Services.

Golden, C., & Hammeke, T. (1986). <u>The Luria-Nebraska Neuropsychological battery (revised)</u>. Los Angeles, Western Psychological Services.

Golden, C., Hammeke, T., & Purisch, A. Berg, R., Moses, J., Newlin, T Wilkening, G., & Puente, A. (1982). <u>Item interpretation of the Luria-Nebraska Neuropsychological battery</u>. Lincoln, Nebraska: University of Nebraska Press.

Golden, C., Moses, J., Zelazowski, R., Graber, B., Zatz, L., Horwarth, T., & Berger, P. (1980). Cerebral ventricular size and neuropsychological impairment in young chronic schizophrenics. <u>Archives of General Psychiatry</u>, <u>37</u>, 619.

Green, P., Hallett, S., & Hunter, M. (1983). Abnormal interhemispheric integration and hemispheric specialization in schizophrenics and high risk children. In P. Flor-Henry & J. Gruzelier (Eds.), <u>Laterality and Psychopathology</u> (pp. 443-469). Amsterdam: Elsevier.

Guilford, J. (1929). Results of training in understanding of facial expressions of emotion. Cited in Woodworth, R. (1938). Experimental Psychology. New York: Holt.

Halberstadt, A. (1983). Family expressiveness styles and

nonverbal communication skills. <u>Journal of Nonverbal Behavior</u>, <u>8</u>, 14-27.

Hays, W. (1981). Statistics. New York: Holt, Rinehart and Winston.

Haggard, E., & Isaacs, K. (1966). Micro-momentary facial expressions as indicators of ego mechanisms in psychotherapy. In L. Gottschalk & A. Auerbach (Eds.), <u>Methods of research in psychotherapy</u> (pp. 213-235). New York: Appleton-Century Croft.

Ingvar, D., & Franzen, G. (1974). Abnormalities of cerebral blood flow in patients with chronic schizophrenia. <u>Acta Psychiatrica Scandanavia</u>, <u>10</u>, 425-446.

Janis, I. (1972). <u>Victims of groupthink</u>. Boston: Houghton-Mifflin.

Jensen, A., & Rohwer, W. (1966). The Stroop color word test: a review. <u>Acta Psychologica</u>, <u>25</u>, 36-93.

Johnson, J., & McCown, W. (submitted). Meta-analysis of affect recognition deficits in clinical syndromes. manuscript submitted for publication.

Jordan, R. (1969). <u>Faces: a history of physiognomy and science</u>. Topeka: Emporia Press.

Kanner R. (1931). Judging emotions from facial expressions. <u>Psychology Monograph,</u> <u>41</u>, (3, Whole No. 186).

Kirk, R. (1982). <u>Experimental design</u>. Belmont, California: Brooks/Cole.

Langfeld, R. (1918). The judgment of emotions from facial expressions. <u>Journal of Abnormal and Social Psychology</u>, <u>13</u>, 172-184.

Ley, R., & Bryden, M. (1979). Hemispheric differences in processing emotions and faces. <u>Brain and Language</u>, <u>7</u>, 127-138.

Lezak, M. (1983). <u>Neuropsychological assessment</u>.New York: Oxford Press.

Livesay, J. (1981). Inconsistent interpersonal judgment in thought-disordered schizophrenia. <u>Psychological Reports</u>, <u>49</u>, 179-182.

Luria, A. (1963). <u>Restoration of function after brain injury</u>. New York: Macmillan.

Luria, A. (1973). <u>The working brain: an introduction to neuropsychology</u>. London: Penguin.

Mandal, H. (1986). Judgement of facial affect in depressives and schizophrenics. <u>British Journal of Clinical Psychology</u>, <u>25</u>, 87-94.

McCown, W. (1988). Methods of affect recognition presentation and their interaction with primary personality factors. Unpublished manuscript. New Orleans VA Medical Center Psychology Service.

McCown, W., Johnson, J., & Austin, S. (1986). Inability of delinquents to decode facial expressions of emotion. <u>Journal of Personality and Social Behavior</u>, 1, 487-496.

McCown, W., Johnson, J., & Austin, S. (in press). Patterns of errors in delinquents' abilities to decode facial affects. Journal of Social Behavior and Personality.

McCown, W., Johnson, J., Shefsky, M., & Austin, S. (in press).Deficits in parents of schizophrenics abilities to decode facial expressions: clinical implications. Psychotherapy and Private Practice.

Mednick, S. (1974). Breakdown in individuals at high risk for schizophrenia: possible predispositional factors. In J. Neale, G. Davison, & K. Price (Eds.), Contemporary Readings in Psychopathology (pp. 209-222). New York: Wiley.

Morrison, R., & Bellack, A. (1987). Social functioning of schizophrenic patients: clinical and research issues. Schizophrenia Bulletin, 13, 715-725.

Mukari, D., & Bates, M. (1977). Judgments of emotion and chronic schizophrenia. Journal of Clinical Psychology, 33, 662-666.

Noummenna , T. (1964). The language of the face. Jvuslvidna, Findland: Jyvaskylan Yilopistoyhdistys.

Osgood, C. (1966). Dimensionality of the semantic space for communication via facial expressions. Scandinavian Journal of Psychology, 7, 121.

Petty, R., & Cacioppo, J. (1981). Attitudes and per- suasions: classic and contemporary approaches. Dubuque, Iowa: William Brown Company.

Piderit, T. (1859). <u>Mimik und Physiognomik</u>. Universitaadt Munchen.

Reitan R. (1957). <u>Manual for the Halstead-Reitan neuropsychological battery</u>. Author.

Reitan, R., & Davison, L. (1974). <u>Clinical neuropsychology: current status and applications</u>. Washington: Winston.

Rosenthal, R. (1976). <u>Experimenter effects in behavioral research</u>. New York: Irving Publishers, Halstead Press Division of Wiley.

Rosenthal, R. (1981). Pavlov's mice, Pfungst's horse, and Pygmalion's PONS: Some models for the study of interpersonal expectancy effects. In T. A. Sebeok & R. Rosenthal (Eds.), <u>The Clever Hans phenomena</u>. Annals of the New York Academy of Sciences, No.364.

Rosenthal, R. (1984). <u>Meta-analytic procedures for the social sciences</u>. Beverly Hills, California: Sage.

Rosenthal, R., & Benowitz, L. (1986). Sensitivity to nonverbal communication in normal, psychiatric and brain damaged samples. In E. Black, R. Buck, & R. Rosenthal (Eds.), <u>Nonverbal communication in the clinical context</u>. Harrisonburg Pennsylvania: University Press, pp. 223-256.

Rosenthal, R., Hall, J., Dimatteo, M., Rogers, P., & Archer, D. (1979). <u>Sensitivity to nonverbal communication: The PONS test</u>. Baltimore: The Johns Hopkins University Press.

Russell, E., Neuringer, C., & Goldstein, G. (1970). <u>Assess-</u>

ment of brain damage: A neuropsychological key approach. New York: Wiley.

Schlosberg, H. (1954). Three dimensions of emotion. Psychological Review, 61, 81-88.

Seemen, M. (1985). Schizophrenia and laterality. In M. Menuck & M. Seeman, (Eds.), New perspectives in schizophrenia (pp. 61-70). New York: Macmillan.

Silverstein, M. (1988, January). Neuropsychological dysfunction and clinical status in schizophrenia and depression. Paper presented at the 16th annual International Neuropsychological Science Association, New Orleans, Louisiana.

Smith, A. (1982). Symbol Digit Modalities Test. Los Angeles: Western Psychological Press.

Sperry, R. (1968). Mental unity following surgical disconnection of the cerebral hemispheres. Harvey Lectures, 62, 293-323.

Stuss, B., & Benson, W. (1986). The frontal lobes. Amsterdam: Elsevier.

Taylor, M. (1981). The neuropsychiatric mental status examination. Jamaica, New York: Spectrum.

VandenBerg, S., & Mattson, E. (1961). The interpretation of facial expressions by schizophrenics, other mental patients, normal adults and children. Acta Psychologica, 12, 495-496.

Walker, E,. Marwit, S., & Emory, E. (1980). A cross-section-

al study of emotion recognition in schizophrenics. <u>Journal of Abnormal Psychology</u>, <u>89</u>, 428-436.

Wechsler, D. (1958). <u>The measurement and appraisal of adult intelligence</u> (4th ed.). Baltimore: Williams & Wilkins.

Weinberger, D., Torrey, E., Neophytides, A., & Wyatt, R. (1979). Lateral cerebral ventricular enlargement in chronic schizophrenia. <u>Archives of General Psychiatry</u>, <u>36</u>, 361-374.

Whitaker, L. (1980). <u>Objective measurement of schizophrenic thinking: a practical and theoretical guide to the Whitaker Index of Schizophrenic Thinking</u>. Los Angeles: Western Psychological Services.

Winer, B. <u>Statistical principles in experimental design</u> (2nd ed.). New York: McGraw-Hill.

Woodworth, R. (1938). <u>Experimental Psychology</u>. New York: Holt.

Wolf, F. (1986). <u>Meta-analysis: Quantitative methods for research synthesis</u>. Sage University Paper series on quantitative applications in social sciences. Series No. 07-059.

Zec, R., & Weinberger, D. (1988, January). Residual neuropsychological impairments in chronic schizophrenia. Paper presented at the 16th annual International Neuropsychological Science Association, New Orleans, Louisiana.

APPROVAL SHEET

The dissertation submitted by William McCown has been read and approved by the following committee:

> Dr. Alan Dewolfe, Director
> Professor of Psychology, Loyola
>
> Dr. Richard Maier
> Professor of Psychology, Loyola
>
> Dr. John Shack
> Professor of Psychology, Loyola

The final copies have been examined by the director of the dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the dissertation is now given final approval by the Committee with reference to content and form.

The dissertation is therefore accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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Date

Alan S. Dewolfe, Ph.D.
Director's Signature