The Incidence of Minor Physical Anomalies in Adult Male Schizophrenics

by James D. Guy, Lawrence V. Majorski, Charles J. Wallace, and Margaret P. Guy

Abstract

The Waldrop anomaly score was used to assess the incidence of minor physical anomalies among 40 adult male Caucasian schizophrenics. This sample had a higher group mean anomaly score than that reported for the normal population ($p < .001$). Those with higher anomaly scores evidenced poorer premorbid adjustment ($p < .05$). A significant relationship also existed between anomaly scores and the subset of premorbid adjustment, Wechsler Adult Intelligence Scale Vocabulary scores, and the Neurological Impairment Index ($p < .05$). These results suggest that first trimester developmental abnormalities, as reflected by the high incidence of anomalies, may parallel some form of central nervous system disturbance which may, in some cases, predispose toward the eventual development of schizophrenia. Implications of these findings, along with suggestions for further research, are considered.

Mounting evidence suggests that genetically related organic factors of various types contribute to the etiology of some forms of schizophrenia (Wender 1972). The incidence of schizophrenia among biological relatives of schizophrenics has been found to be significantly higher than that for 2d among the general population (Rosenthal et al. 1971). The closer the biological relationship, the higher is the incidence (Kety et al. 1968). Although the exact hereditary mechanism is unknown, the most promising theories concerning the transmission of schizophrenia posit a complex, multifactorial, and interactional process, in which both recessive and dominant genes predispose the individual toward the development of schizophrenia in the presence of sufficient environmental stressors (Kallmann 1946; Rosenthal et al. 1977). However, some theorists even go so far as to suggest that genetic transmission alone is sufficient to produce several forms of schizophrenia, regardless of the nature of environmental influences (Matthysse and Kidd 1976).

In addition to genetic factors, a growing body of research suggests that nongenetic biological factors may also contribute to the development of schizophrenia. Teratogenic insults to the fetus during the first trimester of embryonic development, such as maternal infection or disease (Wender 1972) or dietary deficiency (Warkany 1951), have been implicated in the etiology of schizophrenia among children (Campbell et al. 1978). Difficulties in later pregnancy and delivery have also been identified as possible contributing factors (Mednick 1970).

While numerous biochemical theories exist which identify chemical abnormalities as possible underlying mechanisms of schizophrenia, some evidence suggests that other central nervous system (CNS) deficits related to neurological impairment in brain structure or function may also be involved in the development of schizophrenia (Brackbill 1956; Mirsky 1969; Klonoff, Fibiger, and Gutton 1970). The extensive longitudinal studies of Mednick and Schulsinger (1968), Fish (1957, 1959, 1960, 1971), Zubin (1975), and Zubin and Spring (1977) have provided further evidence of CNS deficits in some forms of schizophrenia, and a high incidence of neurological and neuropsychological impairment has been found in both child and adult schizophrenics. For example, schizo-
phrenic patients evidence more abnormalities on physical neurological exams (Pollin et al. 1966; Rockford et al. 1970), electroencephalograms (Small et al. 1972; Tucker et al. 1965), pneumocencephalograms (Haug 1963), and histopathic studies of the brain (Wildi, Linder, and Costoulos 1967) than both normals and other psychiatric patients. Many schizophrenic patients also evidence an abundance of soft neurological signs (Quitkin, Rifkin, and Klein 1976). Elkes (1961) suggests that disturbances in basic information processing related to neurological impairment are frequently found among schizophrenics. Others have found evidence of abnormal subcortical modulating mechanisms (Josiassen 1978). Heath and Krupp (1967) reported that some schizophrenics showed structural abnormalities in the septal region of the brain, while Mednick (1970) found structural damage in the hippocampus. Unfortunately, the precise role of these numerous CNS deficits in the etiology of schizophrenia is unknown.

Several recent studies have found a relationship between a high incidence of minor physical anomalies and the development of schizophrenia (Campbell et al. 1978; Goldfarb and Botstein 1967; Walker 1977). A minor physical anomaly is a slight physical defect, a deviation in appearance from essential physical characteristics (Evans, Goldstein, and Rodnick 1973). These abnormalities include “electric hair,” abnormal-sized head, epicanthus, hypertelorism, low-seated ears, adherent ear lobes, malformed or asymmetrical ears, high-steepled mouth, furrowed tongue, curved fifth finger, single transverse palmar crease, syndactyly of the toes, gaps between the first and second toes, and unusually long third toes. Table 1 contains the Waldrop Scale (Waldrop, Pederson, and Bell 1968), a widely used standardized scoring system for assessing the incidence of minor physical anomalies. With the possible exception of head size, these minor physical anomalies develop during the first trimester, the most critical period of fetal growth (Moore, cited in Smith 1976; Waldrop 1979). All of the essential ectodermal derivatives develop at this time, such as the brain, heart, liver, somites, arms, legs, ears, nose, and eyes. This is also a particularly critical phase in general CNS development (Mussen, Conger, and Kagan 1979). Minor physical anomalies developing at this time sometimes result from genetic abnormalities such as Down’s syndrome, Trisomy 13, Seckel’s syndrome, and a variety of complex interacting polygenic abnormalities (Rapoport and Quinn 1975; Smith 1970; Smith, cited in Rosenthal 1970). Several family studies have further established the role of genetic abnormalities in the etiology of anomalies (Firestone, Lewy, and Douglas 1976; Firestone et al. 1978; Oettinger, Evans, and Harris 1979; Smith 1970; Smith, cited in Rosenthal 1970). In addition to these genetic factors, a variety of teratogenic agents, such as rubella, infection, anoxia, bleeding, fetal distress, dietary deficiency, and toxemia, may also cause a number of anomalies (Pasamanick and Rogers 1956; Warkany, Monroe, and Sutherland 1961; Waldrop and Halverson 1972; Rapoport, Quinn, and Lamprecht 1974). In addition to causing the formation of minor physical anomalies, these same genetic and teratogenic agents may also alter critical CNS development occurring during this first trimester period, resulting in varying degrees of neurological impairment (Quinn and Rapoport 1974; Steg and Rapoport 1975). In some cases, the greater the severity of the fetal trauma, the greater are both the number of anomalies and the severity of CNS deficit (Kawi and Pasamanick 1958; Rosenberg and Weller 1973). Thus, the presence of multiple anomalies suggests the likelihood of general developmental deviation, and CNS deficits in particular. Recent research has demonstrated that the incidence of minor physical anomalies is associated with various forms of CNS-related pathology, such as speech and hearing impairment, mental retardation, academic failure, learning disabilities, poor motor coordination, and hyperactivity (Waldrop 1979).

In view of the mounting evidence of genetic and teratogenic factors in the etiology of schizophrenia, as well as the suggestive evidence of the role of neurological impairment of both structure and function in the development of schizophrenic symptoms, it is not surprising that initial studies have found a relationship between the incidence of minor physical anomalies and the development of schizophrenia and related disorders. Goldfarb and Botstein (1967) found that a group of schizophrenic children had the highest mean number of anomalies as compared to both normal and nonpsychotic behavior-disordered children. The schizophrenic group also had the highest number of children with multiple anomalies, with 24 percent having five or more. Only ¼ percent of the other groups combined had five or more anomalies. Of related interest, Steg and Rapoport (1975) found that autistic children also tended to have more anomalies than either psycho-neurotics or normals (p < .05). Walker (1977) reported that autistic
children had more anomalies than normal children (p < .001). Finally, Campbell et al. (1978) reported that autistic children had more anomalies than both their siblings and normal children (p < .01).

The purpose of this study was to investigate the relationship between the incidence of minor physical anomalies and schizophrenia in adult males, as related to age of onset, process vs. reactive status, chronic vs. acute course, good vs. poor premorbid functioning, and neurological impairment vs. non-impairment. It was felt that such a study would be useful for several reasons. By more fully exploring the relationship between the incidence of anomalies and various features of schizophrenia, it was hoped that additional evidence concerning the role of organic factors in the nature and severity of schizophrenia would be obtained. Furthermore, such a study might provide data about neuropsychological impairment in schizophrenic patients, as related to evidence of first trimester fetal trauma. Finally, anomaly examinations might be useful in identifying "at risk" individuals, as well as in differentiating between organic and functional forms of etiology, if a relationship between the incidence of minor physical anomalies and certain forms of schizophrenia were to be demonstrated.

**Methods**

Goldstein (1978) proposed a multileveled model as the most useful way of studying various forms of schizophrenia. By the incorporation of such a model, the following hypotheses were formulated:

- Hypothesis I: Extensive normative data have established that the

<table>
<thead>
<tr>
<th>Table 1. Waldrop list of minor physical anomalies with scoring weights</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anomaly</strong></td>
</tr>
<tr>
<td>-------------</td>
</tr>
<tr>
<td><strong>Head</strong></td>
</tr>
<tr>
<td>Fine electric hair:</td>
</tr>
<tr>
<td>Very fine hair that will not comb down</td>
</tr>
<tr>
<td>Fine hair that is soon awry after combing</td>
</tr>
<tr>
<td>Two or more hair whorls</td>
</tr>
<tr>
<td>Head circumference outside normal range:</td>
</tr>
<tr>
<td>&gt;1.5'</td>
</tr>
<tr>
<td>&gt;1.0 &lt;1.5'</td>
</tr>
<tr>
<td><strong>Eyes</strong></td>
</tr>
<tr>
<td>Epicanthus:</td>
</tr>
<tr>
<td>Where upper and lower lids join the nose, point of union is:</td>
</tr>
<tr>
<td>Deeply covered</td>
</tr>
<tr>
<td>Partly covered</td>
</tr>
<tr>
<td>Hypertelorism:</td>
</tr>
<tr>
<td>Approximate distance between tear ducts:</td>
</tr>
<tr>
<td>&gt;1.5'</td>
</tr>
<tr>
<td>&gt;1.0 &lt;1.5'</td>
</tr>
<tr>
<td><strong>Ears</strong></td>
</tr>
<tr>
<td>Low-seated ears:</td>
</tr>
<tr>
<td>Point where ear joins head, not in line with the corner of eye and nose bridge:</td>
</tr>
<tr>
<td>Lower by &gt;0.5 cm</td>
</tr>
<tr>
<td>Lower by &lt;0.5 cm</td>
</tr>
<tr>
<td>Adherent ear lobes:</td>
</tr>
<tr>
<td>Lower edge of ears extend:</td>
</tr>
<tr>
<td>Upward and back toward crown of head</td>
</tr>
<tr>
<td>Straight back toward rear of neck</td>
</tr>
<tr>
<td>Malformed ears</td>
</tr>
<tr>
<td>Asymmetrical ears</td>
</tr>
<tr>
<td>Soft and pliable ears</td>
</tr>
<tr>
<td><strong>Mouth</strong></td>
</tr>
<tr>
<td>High-steepled palate:</td>
</tr>
<tr>
<td>Roof of mouth:</td>
</tr>
<tr>
<td>Definitely steepled</td>
</tr>
<tr>
<td>Flat and narrow at the top</td>
</tr>
<tr>
<td>Furrowed tongue (one with deep grooves)</td>
</tr>
<tr>
<td>Tongue with smooth-rough spots</td>
</tr>
<tr>
<td><strong>Hands</strong></td>
</tr>
<tr>
<td>Curved fifth finger:</td>
</tr>
<tr>
<td>Markedly curved inward toward other fingers</td>
</tr>
<tr>
<td>Slightly curved inward toward other fingers</td>
</tr>
<tr>
<td>Single transverse palmar crease</td>
</tr>
</tbody>
</table>

Continued on next page.
Table 1. Waldrop list of minor physical anomalies with scoring weights—Continued

<table>
<thead>
<tr>
<th>Anomaly</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feet</td>
<td></td>
</tr>
<tr>
<td>Third toe longer than second:</td>
<td></td>
</tr>
<tr>
<td>Definitively longer than second toe</td>
<td>2</td>
</tr>
<tr>
<td>Appears equal in length to second toe</td>
<td>1</td>
</tr>
<tr>
<td>Partial syndactylia of two middle toes</td>
<td>1</td>
</tr>
<tr>
<td>Big gap between first and second toes</td>
<td>1</td>
</tr>
</tbody>
</table>

1 SD from mean based on age.

incidence of minor physical anomalies among the general population ranges between 0 and 4 (Waldrop 1975, 1979). These scores remain stable from birth onward (Campbell et al. 1978; Waldrop 1979). Waldrop scores of 5 or greater have consistently been shown to correlate with various forms of psychopathology (Waldrop 1979). It was hypothesized that adult male schizophrenics would tend to have Waldrop scores of 5 or greater, with a group mean of 5 or greater.

- Hypothesis II: Early onset of schizophrenia has been found to be related to possible insults to the brain (Gittleman and Birch 1968; Mednick 1970). Several studies have also demonstrated that the incidence of minor physical anomalies is related to early onset of several forms of psychopathology (Waldrop 1979). Since the incidence of anomalies was also found to be related to CNS deficits, one might expect to find a higher incidence of anomalies among schizophrenic patients with early onset. It was hypothesized that those with earlier onsets of identifiable psychiatric symptoms would have higher Waldrop scores than those with later onsets.

- Hypothesis III: Patients with process schizophrenia show more evidence of neurological impairment and CNS pathology than patients with reactive schizophrenia (Lilliston 1970; Parsons and Klein 1970). Since CNS pathology correlates with the incidence of anomalies, it was hypothesized that patients with process schizophrenia would have higher Waldrop scores than patients with reactive schizophrenia.

- Hypothesis IV: Chronic schizophrenic patients tend to evidence greater neurological impairment than acute schizophrenic patients (Weiner 1966; Goldstein 1978; Heaton, Baade, and Johnson 1978). Since the incidence of anomalies has been found to be related to neurological impairment, it was hypothesized that chronic schizophrenic patients would have higher Waldrop scores than acute schizophrenic patients.

- Hypothesis V: Poor premorbid functioning has been found to be related to neurological impairment in schizophrenia (Lilliston 1970). Patients with poor premorbid functioning also tend to be those with process and chronic schizophrenia (Evans, Goldstein, and Rodnick 1973; Goldstein 1978). Thus, it was hypothesized that those evidencing poor premorbid functioning would have higher Waldrop scores than those with good premorbid functioning.

- Hypothesis VI: Because of the numerous studies which found a relationship between schizophrenia and some degree of neurological impairment, as well as those demonstrating a relationship between anomalies and neurological impairment, it was hypothesized that those evidencing neurological impairment would have higher Waldrop scores than those without impairment.

Subjects. Subjects were selected at random from the inpatient population at Camarillo State Hospital, Camarillo, California, and the day treatment population at Harbor-UCLA Medical Center, Torrance, California. All subjects were between 18 and 65 years of age, as verified by hospital records. Because males tend to evidence more genetic and teratogenic abnormalities (Halverson and Victor 1976), as well as a greater incidence of anomalies (Waldrop 1979), only males were included to eliminate gender-related variance. Most published studies on neurological impairment in schizophrenia included only males, and due to subject availability, males were also selected for this study. Since previous anomaly studies were limited to Caucasians, only Caucasians were included to eliminate possible variations in anomaly scores due to racial differences (Waldrop 1975). In an effort to eliminate variance due to the effects of long-term use of psychiatric medications and institutionalization upon cognitive and perceptual abilities,
subjects who had been hospitalized for more than 3 of the last 5 years were excluded (Strauss and Carpenter 1972; Goldstein 1978). All subjects had been hospitalized or under the direct care of a physician for at least 10 days before testing, to eliminate variance due to recently prescribed medications or florid psychosis (Trouton and Eysenck 1961; Owen 1970; Baker 1968). Twenty-five subjects at Camarillo and 15 at Harbor-UCLA fulfilled these criteria and agreed to participate, providing a reasonably balanced representation of the schizophrenic population (Goldstein 1978). Due to the extensive normative data on the incidence of minor physical anomalies among the general population, and because this study was primarily a within-group comparison, a control group was not included.

Procedure. Patients diagnosed as schizophrenic by attending psychiatrists were randomly selected and approached to obtain consent for initial interviewing. Those consenting were interviewed using the Present State Examination (Wing, Cooper, and Sartorius 1974). Those satisfying both the requirements of the New Haven Schizophrenia Index (Astrachan et al. 1972) and DSM-III (American Psychiatric Association 1980) for a diagnosis of schizophrenia were then included as subjects. Forty-five potential subjects were interviewed to obtain the necessary 40 schizophrenic subjects who fulfilled both sets of diagnostic criteria and provided consent. Interrater reliability on the exact scoring of the New Haven was \( r = .78 \) and \( r = 1.00 \) on the diagnosis of schizophrenia, as obtained by eight videotaped interviews. Those consenting to testing were examined for anomalies and Waldrop scores were obtained (Waldrop, Pederson, and Bell 1968). Interrater reliability based on three videotaped and seven live exams was \( r = .92 \), well within the range of \( .70 \) to \( .96 \) obtained in past studies (Waldrop 1979). Because all interviewing, examining, and testing were conducted by one examiner, all anomaly exams were done before any data gathering and testing to reduce rater bias (Rosenberg and Weller 1973; Walker 1977).

Following the anomaly exam, each subject was interviewed, using the oral versions of the Ullmann-Giovannoni Self-Report Process-Reactive Questionnaire (Ullmann and Giovannoni 1964) and the Abbreviated Phillips Premorbid Adjustment Scale (Harris 1975). Interrater reliability of \( r = .90 \) was obtained on eight videotaped interviews. At this point, each subject was given a battery of tests, including the Memory for Designs Test (Graham and Kendall 1968), the Benton Visual Retention Test (Benton 1955), the Trail-Making Test (Reitan 1956), and the Vocabulary Subtest of the Wechsler Adult Intelligence Scale (WAIS) (Wechler 1955). To control for practice effects, each of the possible 10 orders of administration were randomly assigned four times. The first three tests were chosen because of their correct "hit rates" of 68 to 84 percent in differentiating organic and nonorganic psychiatric patients, with correct hit rates of 69 to 77 percent among schizophrenics when long-term chronic were excluded, as they were from the present study (Goldstein 1978). The WAIS Vocabulary Subtest was included to assess verbal memory, primarily a left hemisphere function, since the other tests tend to assess primarily right hemisphere functioning.

After testing was completed, each subject's charts and medical records were reviewed. Those hospitalized for a total of less than 12 months, either consecutively or with multiple admissions, were identified as acute, while those hospitalized for 12 or more months were identified as chronic (Ricks and Berry 1970; Goldstein 1974; Goldstein and Halperin 1977). Age of onset was also determined by a comprehensive review of all records. Where no age of onset of identifiable psychiatric symptomatology could be clearly found, age of first hospitalization was used (Goldberg et al. 1977). All 40 subjects qualifying for inclusion in the study successfully completed the interviews and testing.

Results
The experimental variables were quantified into raw data for comparison purposes. Waldrop Scores were the total number of points obtained using the Waldrop Anomaly Scale (Waldrop, Pederson, and Bell 1968). The higher the score, the greater were the number and severity of the anomalies present. Ages were expressed in total years, rounded off to the nearest year. Process-reactive was the total number of points obtained on the Ullmann questionnaire (Ullmann and Giovannoni 1964). The higher the score, the more reactive was the schizophrenia. Chronic-acute was determined by the method described earlier, and a value of "0" was assigned to patients with acute schizophrenia, while patients with chronic schizophrenia were given a "1." Premorbid functioning was the total number of points on the Abbreviated Phillips Scale (Harris 1975). The higher the score, the poorer was the level of premorbid adjustment. WAIS vocabulary was the total
number of points obtained for correct responses on the Vocabulary Subtest of the WAIS. Impairment index was derived from the scores earned on the Memory for Designs, Benton Visual Retention, and Trail-Making tests, converted into linear $t$ scores, and averaged together to obtain a mean $t$ score. This mean $t$ score was the impairment index. The greater the score, the greater was the neurological impairment.

Table 2 presents the mean, standard deviation, and the range for each variable. Table 3 presents the univariate correlation matrix. Table 4 presents the $t$ test comparison of mean anomaly scores.

Hypothesis I. Even after a value of 4 was selected to represent the mean anomaly score of the general population, which is actually the upper limit of the 0–4 range consistently reported (Waldrop 1979), the schizophrenic patients in this study evidenced significantly more than the expected number of anomalies ($p < .001$; see table 4). The group mean was 6.89; only 25 percent had fewer than 5 anomaly points, and no schizophrenic patient had fewer than

### Table 2. Univariate statistics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Standard deviation</th>
<th>Smallest value</th>
<th>Largest value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>29.625</td>
<td>8.666</td>
<td>19</td>
<td>51</td>
</tr>
<tr>
<td>Onset</td>
<td>18.8</td>
<td>5.254</td>
<td>12</td>
<td>33</td>
</tr>
<tr>
<td>Process-reactive</td>
<td>11.275</td>
<td>3.282</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>Chronic-acute</td>
<td>4.25</td>
<td>.501</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Premorbid function</td>
<td>5.125</td>
<td>2.399</td>
<td>1</td>
<td>12</td>
</tr>
<tr>
<td>WAIS Vocabulary</td>
<td>42.35</td>
<td>18.328</td>
<td>14</td>
<td>77</td>
</tr>
<tr>
<td>Impairment index</td>
<td>49.933</td>
<td>7.985</td>
<td>40.8</td>
<td>78.3</td>
</tr>
<tr>
<td>Waldrop score</td>
<td>6.875</td>
<td>2.884</td>
<td>2</td>
<td>16</td>
</tr>
</tbody>
</table>

### Table 3. Correlation matrix

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Onset</th>
<th>Process-reactive</th>
<th>Chronic-acute</th>
<th>Premorbid function</th>
<th>WAIS Vocabulary</th>
<th>Impairment index</th>
<th>Waldrop score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.00</td>
<td>.51$^*$</td>
<td>1.00</td>
<td>.53$^*$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset</td>
<td>.51$^*$</td>
<td>1.00</td>
<td>.17</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Process-reactive</td>
<td>.38$^2$</td>
<td>-.03</td>
<td>.021</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic-acute</td>
<td></td>
<td></td>
<td>.32$^2$</td>
<td>-.496$^*$</td>
<td>-.003</td>
<td>.394$^*$</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Premorbid function</td>
<td>-.20</td>
<td>-.32$^2$</td>
<td></td>
<td>.21</td>
<td>-.394$^*$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WAIS Vocabulary</td>
<td>.29</td>
<td>.07</td>
<td>.32$^2$</td>
<td>.21</td>
<td>-.459$^*$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impairment index</td>
<td>.21</td>
<td>.01</td>
<td>-.142</td>
<td>.266</td>
<td>.462$^*$</td>
<td>-.459$^*$</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Waldrop score</td>
<td>-.001</td>
<td>-.12</td>
<td>-.21</td>
<td>.11</td>
<td>.314$^2$</td>
<td>.21</td>
<td>-.10</td>
<td>1.00</td>
</tr>
</tbody>
</table>

$^*$ $p < .01$.

$^2$ $p < .05$. 
2 anomaly points. Hypothesis I was confirmed.

Hypotheses II–VI. With the use of an "all possible subsets" multiple regression design, computer program BMDP-P9R, the bivariate relationships between the Waldrop anomaly scores and each variable were examined. Table 5 summarizes the data for hypotheses II–VI. Only hypothesis V was confirmed. Schizophrenics with poorer levels of premorbid functioning had higher anomaly scores \( (p < .05) \). When variance due to all other variables was partialed out, premorbid functioning accounted for about 12 percent of the variance in Waldrop scores \( (p < .05) \).

Table 6 presents the \( F \) ratios for those subsets which accounted for the greatest amount of variance in Waldrop anomaly scores. One subset was computed for each incremental number of possible variables. \( F \) ratios were computed for both the variance due to the subset, and increment of variance accounted for by adding a new variable to the subset. The best possible subset was premorbid functioning and WAIS Vocabulary, as related to Waldrop anomaly scores. This subset accounted for 23 percent of the variance in anomaly scores \( (p < .01) \). Adding WAIS Vocabulary scores to the comparison of premorbid functioning and anomaly scores added significantly to the explanation of variance in anomaly scores, over and above that of premorbid functioning \( (p < .01) \). Although three other subsets also reached statistical significance, the addition of each remaining variable to the above subset did not add significantly to the explanation of variance in Waldrop scores beyond that already provided by the subset premorbid functioning and WAIS Vocabulary scores, i.e., the increments were not significant.

### Discussion

In this investigation, adult male schizophrenic patients had a higher group mean anomaly score than that found among the general population \( (p < .001) \). Seventy-five percent of these schizophrenics had Waldrop scores above the upper limits of those obtained in the general population. These findings are consistent with previous research which found relationships between anomaly scores and both childhood schizophrenia (Goldfarb and Botstein 1967) and childhood autism (Steg and Rapoport 1975; Walker 1977; Campbell et al. 1978). The results of this study give further evidence of some type of developmental abnormality in the first trimester which may predispose toward the development of schizophrenia. These findings are also consistent with previous studies which suggest the influence of organic factors in the etiology of some forms of schizophrenia.

<table>
<thead>
<tr>
<th>Variable</th>
<th>( r ) zero-order correlation</th>
<th>( r^2 ) variance</th>
<th>( r^2 ) semi-partialled</th>
<th>( F ) ratio of semi-partialled ( r^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>-.122</td>
<td>.01485</td>
<td>.00073</td>
<td>.03253</td>
</tr>
<tr>
<td>Process-reactive</td>
<td>-.210</td>
<td>.04423</td>
<td>.01428</td>
<td>.63640</td>
</tr>
<tr>
<td>Chronic-acute</td>
<td>.109</td>
<td>.01183</td>
<td>.00321</td>
<td>.14305</td>
</tr>
<tr>
<td>Premorbid function</td>
<td>.314†</td>
<td>.09840</td>
<td>.11393</td>
<td>5.07700†</td>
</tr>
<tr>
<td>WAIS Vocabulary</td>
<td>.207</td>
<td>.04285</td>
<td>.04060</td>
<td>1.81000</td>
</tr>
<tr>
<td>Impairment index</td>
<td>-.100</td>
<td>.01005</td>
<td>.03739</td>
<td>1.66600</td>
</tr>
</tbody>
</table>

\( p < .05 \).
The Abbreviated Phillips Premorbid Adjustment Scale (Harris 1975) assesses the level of interpersonal, sexual, and general social adjustment during childhood, adolescence, and early adulthood. Past research demonstrated a relationship between neurological impairment and poor premorbid functioning (Lilliston 1970). A similar relationship was found in this study. Schizophrenics with poor premorbid adjustment obtained low scores on the WAIS Vocabulary Subtest and high scores on the impairment index (p < .01; see table 3). Perhaps neurological impairment hinders early interpersonal relationships and social competency. Previous research has also found a relationship between poor premorbid adjustment and chronic vs. acute, and process vs. reactive, schizophrenia (Evans, Goldstein, and Rodnick 1973; Goldstein 1978). Similar relationships were found in this study. Those with poorer levels of premorbid functioning tended to have process schizophrenia (p < .01) with early ages of onset (p < .05). A significant relationship was also found between Waldrop scores and the subset of premorbid adjustment, WAIS Vocabulary scores, and the impairment index, which accounted for 25 percent of the variance in anomaly scores (p < .05). This suggests that first trimester aberrations leading to high Waldrop scores may have resulted in some type of neurological impairment which predisposed these individuals toward poor social competency and eventual schizophrenia.

When the implications of these findings are considered, it is important to remember that the incidence of minor physical anomalies is a nonspecific predictor of potential psychopathology. Previous studies have found relationships between Waldrop scores and hyperactivity, autism, childhood schizophrenia, epilepsy, learning disabilities, mental retardation, speech and hearing impairments, and poor motor coordination (Waldrop 1979). While the presence of multiple anomalies indicates early fetal trauma which may have altered CNS development, the exact nature of the potential psychopathology cannot be predicted by Waldrop scores. Thus, the presence of multiple anomalies in this population of schizophrenic patients indicates only that a first trimester aberration has occurred.

Table 6. Multiple regression matrix for $r^2$ increments

<table>
<thead>
<tr>
<th>No. of variables in subset</th>
<th>Best subset</th>
<th>$r^2$ variance</th>
<th>$F$ for subset</th>
<th>$F$ for increment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6</td>
<td>.09840</td>
<td>4.147</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>6,7</td>
<td>.22783</td>
<td>5.459</td>
<td>6.202</td>
</tr>
<tr>
<td>3</td>
<td>6,7,8</td>
<td>.25472</td>
<td>4.100</td>
<td>1.299</td>
</tr>
<tr>
<td>4</td>
<td>5,6,7,8</td>
<td>.26596</td>
<td>3.171</td>
<td>.536</td>
</tr>
<tr>
<td>5</td>
<td>2,4,6,7,8</td>
<td>.27669</td>
<td>2.602</td>
<td>.501</td>
</tr>
<tr>
<td>6</td>
<td>2,4,5,6,7,8</td>
<td>.28136</td>
<td>2.158</td>
<td>.325</td>
</tr>
<tr>
<td>7</td>
<td>2,3,4,5,6,7,8</td>
<td>.28209</td>
<td>1.796</td>
<td>.033</td>
</tr>
</tbody>
</table>

$^1 p < .05.$

Subsets:
2: Age
3: Age of onset
4: Process-reactive
5: Chronic-acute
6: Premorbid function
7: WAIS Vocabulary
8: Impairment index
does not provide clues to the actual role of organic factors in the etiology of schizophrenia. One is left to speculate on the nature of this relationship. Perhaps the genetic or teratogenic agent responsible for the multiple anomalies may also have interrupted normal CNS development, resulting in aberrancies in neurotransmitter function, inhibitory mechanisms, information processing, or signal detection. Perhaps the resultant CNS abnormality somehow predisposed the individual toward the development of schizophrenia. The rather strong relationship between Waldrop scores and premorbid functioning and neurological impairment encourages further speculation about the role of organic impairment in predisposing one toward a lower level of social competency, poorer social adjustment, and eventual schizophrenia. Perhaps these data give further support to an interactional model which attributes the etiology of schizophrenia to a variety of organic and functional factors, at least in those schizophrenics who evidence high Waldrop anomaly scores.

Several reasons may exist for the fact that four of the basic hypotheses were not confirmed, although all outcomes were in the predicted direction and most approached statistical significance. Because of the narrowly defined and restrictive inclusion criteria, the resultant subject pool was somewhat homogeneous. Scores on the Ullmann-Giovannoni Questionnaire and the impairment index, in particular, did not follow normal distributions. The subject pool was not large enough to compensate for some of this homogeneity, thereby reducing statistical significance somewhat. Some question also remains about operationalizing chronic vs. acute schizophrenia as the total length of hospitalization, due to variance resulting from factors related to changes in social policy, funding, and chemotherapy. Eliminating the long-term chronic patients further weakened the validity of this comparison. Finally, the use of medical records to determine age of onset and length of hospitalizations is likely to have introduced variance due to reporting or recording error.

The speculative nature of this study requires that it be viewed as an initial attempt, at best, to explore the relationship between first trimester fetal trauma, as evidenced by Waldrop anomaly scores, and the development of schizophrenia. The need for further research is obvious. For example, it would be interesting to determine whether blood relatives of schizophrenics with multiple anomalies also have high Waldrop scores, as well as evidencing schizophrenic or nonpsychotic psychopathology. It might also be valuable to determine if there are differential responses to chemotherapy among schizophrenics with varying Waldrop scores. Perhaps there are different mean Waldrop scores among the various subgroups of schizophrenic patients, such as paranoid, undifferentiated, or catatonic. It might also be interesting to determine the relationship between Waldrop scores and the various affective disorders, as compared to schizophrenia. Because anomaly studies have thus far focused on Caucasians, anomaly examinations which focus on other racial groups are badly needed.

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Finally, including Waldrop scores in studies of neurological impairment in schizophrenia would provide further data regarding the role of early fetal trauma in neurological impairment and the development of schizophrenia.

In view of the increased emphasis upon early detection and prevention of psychopathology, the value of the 5- to 10-minute Waldrop Anomaly Exam as an added research assessment tool is apparent. Its inclusion would likely add to the mounting evidence regarding the role of organic factors play in predisposing toward the eventual development of psychopathology.

References


Fish, B. The detection of schizophrenia in infancy. Journal of Nervous and Mental Disease, 125:1–24, 1957.


Fish, B. Involvement of the central nervous system in infants with schizophrenia. Archives of Neurology, 2:115–121, 1960.


Waldrop, M. "Minor Physical Anomalies—A Predictor of Problem


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