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# Multivariate analysis of dermatoglyphics of severe mental retardates: an application of the constellation graphical method for discriminant analysis.

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## Abstract

We studied the dermatoglyphics of 353 severe mental retardates (excluding those with chromosomal abnormalities and major limb malformations), using multivariate analysis, to determine how early intrauterine factors are related to the etiology of mental retardation. First, dermatoglyphics were compared between 140 individuals with undefined prenatal factors and 700 normal controls. After 6 and 9 dermatoglyphic traits were chosen as discriminative variables for males and females, respectively, the data were subjected separately for each sex to the constellation graphical method for discriminant analysis. The same formula as obtained in the idiopathic group was subsequently applied to data from cases in other etiological categories. When the misclassification rate was 0.03, the rates of correct classification of the male patients into the etiological categories of undefined prenatal, defined prenatal, perinatal, postnatal and unknown (no anamnestic data available) categories were 19.7% (13/66), 20.0% (3/15), 8.8% (5/57), 5.0% (1/20) and 7.7% (2/26), while the correct classification rates of females were 24.3% (18/74), 42.1% (8/19), 18.9% (7/37), 5.1% (1/16) and 13.0% (3/23), respectively. The results suggest that early intrauterine factors such as those producing dermatoglyphic deviations may contribute to the pathogenesis of severe mental retardation not only in patients with undefined prenatal etiological factors but also in those with perinatal factors, especially those of the female sex.

**KEYWORDS:** mental retardation, dermatoglyphics, multivariate analysis, constellation graphical method

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# Multivariate Analysis of Dermatoglyphics of Severe Mental Retardates: An Application of the Constellation Graphical Method for Discriminant Analysis

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We studied the dermatoglyphics of 353 severe mental retardates (excluding those with chromosomal abnormalities and major limb malformations), using multivariate analysis, to determine how early intrauterine factors are related to the etiology of mental retardation. First, dermatoglyphics were compared between 140 individuals with undefined prenatal factors and 700 normal controls. After 6 and 9 dermatoglyphic traits were chosen as discriminative variables for males and females, respectively, the data were subjected separately for each sex to the constellation graphical method for discriminant analysis. The same formula as obtained in the idiopathic group was subsequently applied to data from cases in other etiological categories. When the misclassification rate was 0.03, the rates of correct classification of the male patients into the etiological categories of undefined prenatal, defined prenatal, perinatal, postnatal and unknown (no anamnestic data available) categories were 19.7% (13/66), 20.0% (3/15), 8.8% (5/57), 5.0% (1/20) and 7.7% (2/26), while the correct classification rates of females were 24.3% (18/74), 42.1% (8/19), 18.9% (7/37), 5.1% (1/16) and 13.0% (3/23), respectively. The results suggest that early intrauterine factors such as those producing dermatoglyphic deviations may contribute to the pathogenesis of severe mental retardation not only in patients with undefined prenatal etiological factors but also in those with perinatal factors, especially those of the female sex.

*Key words :* mental retardation, dermatoglyphics, multivariate analysis, constellation graphical method

Epidemiological studies have shown that the frequency of severe mental retardation (IQ below 50), about 0.3 to 0.4% of the general population is similar in various countries (1). Though pathological conditions affecting the central nervous system prevail more often in severely than in mildly retarded patients, elucidation of the exact etiology is difficult in many cases and complicated by the genetic/environmental interaction or multiple factors. Epidermal ridges and palmar creases are formed during early intrauterine life, and their configurations are considered to be influenced by both genetic and nongenetic factors at that time (2). This period

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of skin morphogenesis corresponds to the stage of rapid structural organization of the primordial brain. Analysis of dermatoglyphic patterns in retarded patients may provide a clue to the role of early intrauterine factors in the pathogenesis of mental retardation.

Previous dermatoglyphic studies (3-9) suffered from two methodological shortcomings. First, they only concentrated on the comparison of respective dermatoglyphic features between patients and normal controls. Since there are neither abnormal dermatoglyphic nor palmar flexion crease patterns which in themselves indicate a pathological condition, the difference or deviation should be evaluated in terms of distributions or combinations of the same dermatoglyphic features as seen in normal individuals (10). Second, the criticism always remains that the difference could be a bias due to the inclusion of patients with chromosomal abnormalities identifiable by modern cytogenetic techniques. The present paper describes results of multivariate analyses on dermatoglyphics of institutionalized mentally retarded (excluding those with chromosomal abnormalities). We discuss how early intrauterine factors, such as those which produce dermatoglyphic deviation from the normal, may be related to the pathogenesis of mental retardation.

## Subjects and Methods

The subjects were 353 patients, 184 males and 169 females, who were cared for at Asahigawa-Jidoin, Asahigawa-Gakuen and Aiikuryo, Okayama, which are residential institutions for the mentally retarded. The age of the subjects ranged from 4 to 46 years, and averaged 18.6 years. The majority of the patients (84.1%) had severe or profound mental retardation. A wide variety of clinical diagnoses was given to them, mainly on the basis of phyiscal examinations and anamnestic data. The etiologies can be classified into 5 major categories: undefined (39.7%) and defined prenatal (9.6%), perinatal (26.6%), postnatal (10.2%) and unknown (no anamnestic data available) (13.9%) (Table 1). The subclassification did not

**Table 1** Clinical diagnosis of the patients

| Clinical diagnosis | Number of patients | | |
|---|---|---|---|
| | Males | Females | Total |
| Undefined origins | 66 | 74 | 140 |
| Defined prenatal origins: | | | |
| Multiple congenital anomalies/mental retardation (MCA/MR) syndrome[a] | 6 | 4 | 10 |
| Cerebral malformations | 3 | 7 | 10 |
| Progressive degenerative disease of central nervous system | 2 | 4 | 6 |
| Phacomatosis | 2 | 1 | 3 |
| Phenylketonuria | 1 | 1 | 2 |
| Ectodermal dysplasia | 1 | 1 | 2 |
| Congenital rubella syndrome | 0 | 1 | 1 |
| Perinatal origins including birth asphyxia *etc.* | 57 | 37 | 94 |
| Postnatal origins: | | | |
| Encephalitis or encephalopathy | 13 | 10 | 23 |
| Purulent meningitis | 5 | 3 | 8 |
| Intracranial bleeding | 1 | 2 | 3 |
| Others | 1 | 1 | 2 |
| Unknown (no anamnestic data available) | 26 | 23 | 49 |

*a*: This category includes de Lange syndrome (2 male and 2 female patients), Rubinstein-Taybi syndrome (3 male patients), Prader-Willi syndrome without apparent deletion of 15q (2 female patients) and Noonan syndrome (1 male patient).

take electroencephalographic or computerized tomographic findings into consideration. Cases with chromosomal abnormalities and major limb malformations were excluded from the study. Seven hundred healthy individuals (350 males and 350 females), ranging from 18 to 51 years in age, served as normal controls. All subjects were Japanese.

*Dermatoglyphic analysis.* Prints were obtained of fingers, palms and hallucal areas with a Hollister's foot printer. Dermatoglyphic traits studied were fingertip patterns; axial triradii; thenar/first interdigital patterns; second, third and fourth interdigital patterns; hypothenar patterns; transverse palmar flexion creases; hallucal patterns; mean a-b ridge counts; and total finger ridge counts. These traits were analyzed according to the Memorandum on Dermatoglyphic Nomenclature (11). Transverse palmar flexion creases were simply classified as normal or abnormal (complete and aberrant forms of simian and Sydney line) as described previously (12). Data were evaluated separately for the two sexes and the right and left sides, since sex and side are important variables influencing the frequencies of certain dermatoglyphic traits. Statistical comparisons were made with the chi-square test without continuity correction and Student's '$t$' test.

*Multivariate analysis.* Dermatoglyphic traits, which were shown to be related to mental retardation at least at the 5% nominal level of statistical significance by univariate analysis were chosen as discriminative variables, and the dermatoglyphic data of the patients were further studied by multivariate analysis. The constellation graphical method (13), representing method for multi-dimensional data on a two-dimensional plane, was used for discriminant analysis. As schematically shown in Fig. 1, the method is composed of two procedures: numerical quantification of data and weighting of each variable. Briefly, assume $n$ pairs of data with $k$ variables as follows:

$$(X1\alpha,\ X2\alpha,\cdots,Xk\alpha),\quad (\alpha=1,2,\cdots,n). \qquad [1]$$

The data [1] are first transformed into certain angles ($\xi j\alpha$) by $k$ real valued functions $f1, f2,\cdots, fk$ as follows:

$$\xi j\alpha = fj(Xj\alpha),\quad (j=1,2,\cdots,k,\ \alpha=1,2,\cdots,n) \qquad [2]$$

where $fj(j=1,2,\cdots,k)$ is assumed to satisfy the conditions: (a) $0 \leqq fj(Xj\alpha) \leqq \pi/2$, and (b) $fj$ is a strictly monotone function. In the case of continuous data, the function is as follows:

$$fj(Xj\alpha) = \frac{X_{j\alpha}-X_{jl}}{X_{ju}-X_{jl}}\times\frac{\pi}{2}\ , \qquad [3]$$

where

$$Xju = \max_{1\leq\alpha\leq n} Xj\alpha,\ Xjl = \min_{1\leq\alpha\leq n} Xj\alpha;\quad (j=1,2,\cdots,k).$$

The transformation of nominal data must be based on another function. For this purpose, we referred to the log score of the index frequency (14) for each characteristic of the $j$th variable in patients/controls. Suppose that the $j$th variable has $m$ characteritics $Q_{j1}, Q_{j2},\cdots, Q_jm$ and the frequency of each characteristic is $P_{j1}, P_{j2},\cdots, P_jm$ for the controls and $P'_{j1}, P'_{j2},\cdots, P'_jm$ for the patients, the function is as follows:

$$\xi j\alpha = fj(Qj\beta),$$
$$fj(Qj\beta) = \frac{\log P'_{j\beta}/P_{j\beta} - \log P'_{jl}/P_{jl}}{\log P'_{ju}/P_{ju} - \log P'_{jl}/P_{jl}}\times\frac{\pi}{2}\ , \qquad [4]$$

where

$$P'_{ju}/P_{ju} = \max_{1\leq\beta\leq m} P'_{j\beta}/P_{j\beta},$$
$$P'_{jl}/P_{jl} = \min_{1\leq\beta\leq m} P'_{j\beta}/P_{j\beta};\ (j=1,2,\cdots,k).$$

As the result of the transformation, the data [1] take the following form:

$$(\xi 1\alpha,\ \xi 2\alpha,\cdots,\xi k\alpha),\ (\alpha=1,2,\cdots,n)\ . \qquad [5]$$

If a certain length of weight ($Wj$) is assigned to the $j$th variable, positions of the data ($Z\alpha$) will be represented as end points derived from $k$ linked vectors whose arguments and lengths are $\xi j\alpha$ and $Wj(j=1,2,\cdots,k)$, respectively. The formula is as follows:

$$Z\alpha = \sum_{j=1}^{k} Wj(\cos\xi j\alpha + i\sin\xi j\alpha),\quad (\alpha=1,2,\cdots,n), \qquad [6]$$

where

$$i=\sqrt{-1}$$

and the following are assumed to be satisfied:

$$\sum_{j=1}^{k} Wj = 1\ ;\ Wj \geqq 0,\ (j=1,2,\cdots,k). \qquad [7]$$

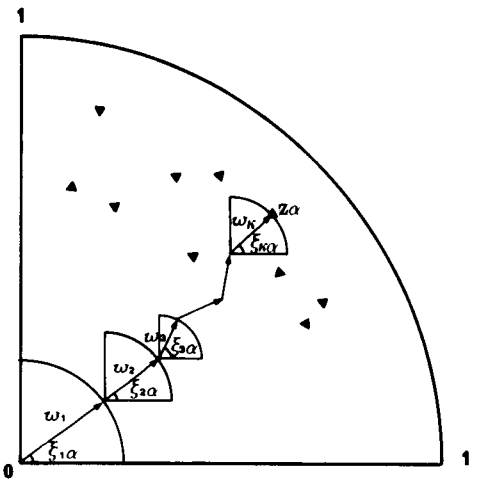


**Fig. 1** Schematic illustration of the constellation graphical method. Triangles ($Z\alpha$) indicate coordinate points of the $\alpha$th individual with $k$ multi-dimensional data on a quarter-circle graph. $Z\alpha$ is represented as an end point derived from $k$ linked vectors whose arguments and lengths are $\xi j\alpha$ and $Wj$ ($j = 1, 2, \cdots, k$, $\alpha = 1, 2, \cdots, n$), respectively. $\xi j\alpha$ is an angle into which each $j$-th variable is transformed, and $Wj$ is a weighting length given to the $j$-th variable.

$Wj$ values fulfilling [7] may be determined in the following two ways. First, the transformed data [5] are subjected to the traditional linear discriminant function, and values of coefficients obtained in the function serve as guidelines for setting the $Wj$ values. Second, $Wj$ values fulfilling [7] are randomly selected from the table of random numbers, and the set of values that can distinguish the two study groups most effectively on the resulting constellation graph is explored. In the present study, we adopted the former method, with the aid of a computer program (Discriminant Analysis, Kyoritsu Press, Tokyo).

Causal factors may be heterogeneous in any etiological category of mentally retarded patients. If dermatoglyphic study were undertaken with overly heterogeneous etiological factors, the cancelling effect could obscure a deviation which might be characteristic of one of its components. The result obtained would be too confounding to interpret. In view of this problem and the fact that dermatoglyphic patterns are formed during early fetal life, we first submitted data from 140 patients with undefined prenatal etiological factors (probably the least heterogeneous group) and 700 normal controls to the discriminant analysis (constellation graphical method), separately for each sex. The same formula of the graph obtained in the undefined prenatal group was subsequently applied to the patients in other etiological categories, in order to determine whether or not dermatoglyphic abnormalities specific to the undefined prenatal group existed in other groups.

## Results

*Dermatoglyphic analysis.* Tables 2 and 3 summarize results of the univariate analysis in the 140 patients with undefined prenatal etiological factors and 700 normal controls. We comment herein on dermatoglyphic traits which differed significantly between the two groups.

Pattern percentages in females were similar between the two groups on all fingers except the right third finger, where more arches and radial loops and fewer ulnar loops were found in the patients ($\chi_3^2 = 11.1$, $0.01 < P < 0.025$).

Mean a-b ridge counts were significantly lower in the patients than in the controls for both sexes (males: $t = 3.3$, $P < 0.001$, females: $t = 2.1$, $0.025 < P < 0.05$). Total finger ridge counts were reduced in the male patients ($t = 2.0$, $0.025 < P < 0.05$).

True patterns appeared more frequently on the right thenar/first (Th/1st) ($\chi_1^2 = 5.3$, $0.01 < P < 0.025$) and the right 2nd interdigital ($\chi_1^2 = 9.1$, $0.001 < P < 0.005$) regions in the male patients, and on the left Th/1st interdigital region in the female patients ($\chi_1^2 = 5.0$, $P = 0.025$). More true patterns were found on both hypothenar regions in the female patients than in the controls ($\chi_3^2 = 10.2$, $0.01 < P < 0.025$; right: $\chi_3^2 = 8.3$, $0.025 < P < 0.05$), while fewer true patterns were observed on the left hypothenar region in the male patients than in the controls ($\chi_1^2 = 5.8$, $0.01 < P < 0.025$).

Abnormal palmar flexion creases were

more frequently seen on both hands in the female patients (left: $\chi_1^2 = 13.4$, $P < 0.001$; right: $\chi_1^2 = 9.4$, $0.001 < P < 0.005$) than in the controls. The female patients were found to have higher axial t positions on both hands (left: $\chi_3^2 = 14.9$, $0.001 < P < 0.005$, right: $\chi_3^2 = 17.5$, $P < 0.001$).

Comparisons of hallucal patterns showed significant differences in the right sole for males ($\chi_5^2 = 11.5$, $0.025 < P < 0.05$) and the left for females ($\chi_6^2 = 20.6$, $0.001 < P < 0.005$). The differences were due to the increase of tibial and fibular arches in the patients of both sexes.

*Multivariate analysis.* Univariate analyses of dermatoglyphics between the undefined prenatal group and the normal controls revealed statistically significant differences ($P < 0.05$) in 6 and 9 dermatoglyphic traits for males and females, respectively. These

**Table 2** Finger pattern[a] percentages, total finger ridge count, and a-b ridge count

| Fingers | Pattern | Normal controls (%) | | | | Patients (%) | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Males | | Females | | Males | | Females | |
| | | Left | Right | Left | Right | Left | Right | Left | Right |
| I | W | 52.0 | 62.0 | 51.2 | 50.8 | 59.1 | 59.1 | 51.3 | 54.0 |
| | $L^u$ | 46.3 | 36.3 | 45.4 | 46.3 | 37.9 | 37.9 | 39.2 | 41.9 |
| | $L^r$ | 0 | 0.3 | 0 | 0.6 | 1.5 | 0 | 0 | 0 |
| | A | 1.7 | 1.4 | 3.4 | 2.3 | 1.5 | 3.0 | 9.5 | 4.1 |
| | | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| II | W | 43.1 | 45.7 | 43.4 | 40.9 | 47.0 | 42.4 | 47.3 | 54.0 |
| | $L^u$ | 39.7 | 39.7 | 39.4 | 48.0 | 39.4 | 37.9 | 35.1 | 36.5 |
| | $L^r$ | 13.2 | 11.4 | 12.0 | 7.7 | 9.1 | 16.7 | 10.8 | 4.1 |
| | A | 4.0 | 3.2 | 5.2 | 3.4 | 4.5 | 3.0 | 6.8 | 5.4 |
| | | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| III | W | 37.2 | 32.3 | 36.3 | 29.7 | 47.0 | 36.4 | 32.4 | 32.4* |
| | $L^u$ | 59.1 | 65.1 | 58.0 | 68.0 | 45.5 | 62.1 | 56.8 | 59.4* |
| | $L^r$ | 1.1 | 1.7 | 2.0 | 0.3 | 3.0 | 0 | 4.1 | 4.1* |
| | A | 2.6 | 0.9 | 3.7 | 2.0 | 4.5 | 1.5 | 6.7 | 4.1* |
| | | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| IV | W | 54.8 | 64.0 | 55.4 | 59.1 | 62.1 | 71.2 | 55.4 | 58.1 |
| | $L^u$ | 44.3 | 35.7 | 44.3 | 40.9 | 36.4 | 28.8 | 43.2 | 40.5 |
| | $L^r$ | 0 | 0 | 0 | 0 | 0 | 0 | 1.4 | 0 |
| | A | 0.9 | 0.3 | 0.3 | 0 | 1.5 | 0 | 0 | 1.4 |
| | | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| V | W | 30.3 | 36.8 | 29.4 | 31.6 | 27.3 | 31.8 | 35.1 | 36.5 |
| | $L^u$ | 68.8 | 62.9 | 68.9 | 66.6 | 72.7 | 68.2 | 63.5 | 60.7 |
| | $L^r$ | 0.6 | 0 | 1.1 | 0.9 | 0 | 0 | 1.4 | 1.4 |
| | A | 0.3 | 0.3 | 0.6 | 0.9 | 0 | 0 | 0 | 1.4 |
| | | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| TFRC[b] | | 142.5 ± 41.5 | | 127.9 ± 42.3 | | 131.1 ± 40.1* | | 125.5 ± 47.4 | |
| Mean a-bRC[c] | | 38.0 ± 4.7 | | 37.3 ± 4.6 | | 35.9 ± 4.7* | | 36.1 ± 3.8* | |

*a*: W: whorl, $L^u$: ulnar loop, $L^r$: radial loop, A: arch.
*b*: Total finger ridge count.
*c*: Mean a-b ridge count.
*: Statistically significant ($P < 0.05$).

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variables were employed for discriminant analysis. The linear discriminant functions obtained were as follows: in males,

$$Z = -0.0917X_1 - 0.0616X_2 - 0.0608X_3 - 0.0358X_4 - 0.0833X_5 - 0.0130X_6 + 2.87,$$

where $X_1$ denotes the true pattern in the right Th/1st interdigital region, $X_2$ the true pattern in the right 2nd interdigital region, $X_3$ the true pattern in the left hypothenar region, $X_4$ the pattern on the right hallucal area, $X_5$ the mean a-b ridge count and $X_6$ the total finger ridge count, and in females,

$$Z = -0.0190X_1 - 0.0154X_2 - 0.0100X_3 - 0.0115X_4 - 0.0160X_5 - 0.0099X_6 - 0.0133X_7 - 0.0262X_8 - 0.0316X_9 + 3.20,$$

where $X_1$ denotes the pattern on the right

**Table 3** Percentages of patterns[a] of palmar and interdigital (ID) areas, axial triradii and hallucal area and frequencies of abnormal palmar flexion crease

| Pattern | Normal controls (%) | | | | Patients (%) | | | |
|---|---|---|---|---|---|---|---|---|
| | Males | | Females | | Males | | Females | |
| | Left | Right | Left | Right | Left | Right | Left | Right |
| True palmar pattern | | | | | | | | |
| Thenar/1st ID | 2.0 | 0.9 | 2.0 | 1.1 | 4.5 | 4.5* | 6.8* | 2.7 |
| 2nd ID | 0.9 | 0.9 | 0.3 | 0.6 | 1.5 | 6.1* | 0 | 0 |
| 3rd ID | 13.1 | 32.0 | 12.0 | 30.0 | 10.6 | 34.8 | 8.1 | 29.7 |
| 4th ID | 64.3 | 61.7 | 73.7 | 65.4 | 64.2 | 60.6 | 79.7 | 62.2 |
| | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| Hypothenar pattern | | | | | | | | |
| Open | 76.0 | 78.3 | 81.7 | 85.4 | 89.4* | 83.3 | 73.0* | 78.3* |
| $L^r$ or $L^d$ | 18.9 | 17.4 | 15.2 | 12.0 | 9.1* | 13.7 | 16.2* | 12.2* |
| $L^u$ or $L^c$ | 3.7 | 2.6 | 2.6 | 2.0 | 1.5* | 1.5 | 6.8* | 8.1* |
| Complex pattern | 1.4 | 1.7 | 0.6 | 0.6 | 0 * | 1.5 | 4.0* | 1.4* |
| | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| Abnormal palmar flexion crease | 6.0 | 7.1 | 4.6 | 4.3 | 10.6 | 9.1 | 16.2* | 13.5* |
| Axial t position | | | | | | | | |
| t | 59.7 | 63.7 | 54.6 | 55.4 | 51.5 | 53.0 | 32.4* | 36.5* |
| t' | 40.0 | 35.7 | 44.6 | 44.0 | 48.5 | 45.5 | 63.5* | 58.1* |
| t'' | 0 | 0.6 | 0.6 | 0.3 | 0 | 0 | 2.7* | 4.1* |
| $t^b$ | 0.3 | 0 | 0.3 | 0.3 | 0 | 1.5 | 1.4* | 1.4* |
| | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| Hallucal pattern | | | | | | | | |
| $L^d$ | 55.1 | 54.9 | 52.8 | 52.6 | 45.5 | 42.4* | 45.9* | 55.4 |
| $L^t$ | 8.3 | 11.1 | 9.4 | 9.4 | 9.1 | 15.1* | 12.2* | 17.6 |
| $L^f$ | 0.3 | 0 | 0.9 | 0.6 | 0 | 0 * | 1.4* | 1.4 |
| W | 24.3 | 26.0 | 27.1 | 28.0 | 24.3 | 22.7* | 24.3* | 20.3 |
| $A^t$ | 3.4 | 2.6 | 0.6 | 0.9 | 7.6 | 9.1* | 6.7* | 0 |
| $A^f$ | 0.3 | 0.6 | 0.9 | 1.7 | 1.5 | 1.5* | 4.1* | 2.7 |
| Others | 8.3 | 4.8 | 8.3 | 6.8 | 12.1 | 9.1* | 5.4* | 2.7 |
| | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

*a*: $L^r$: radial loop, $L^u$: ulnar loop, $L^c$: cubital loop, $L^d$: distal loop, $L^t$: tibial loop, $L^f$: fibular loop, W: whorl, $A^t$: tibial arch, and $A^f$: fibular arch

*: Statistically significant ($P < 0.05$).

3rd finger, $X_2$ the position of the left axial triradius, $X_3$ the position of the right axial triradius, $X_4$ the true pattern in the left Th/1st interdigital region, $X_5$ the true pattern in the left hypothenar region, $X_6$ the abnormal flexion crease on the left palm, $X_7$ the abnormal flexion crease on the right palm, $X_8$ the pattern on the left hallucal area and $X_9$ mean a-b ridge count.

Transformed angles ($\xi j\alpha$) for characteristics of each variable and weighting lengths ($Wj$) for each variable are shown in Tables 4 (for males) and 5 (for females). The end points of the subjects were calculated and illustrated on quarter-circle graphs by the use of a personal computer (PC-9801, NEC) (Fig. 2). If the quarter-circle was divided by a certain radius ($Y=0.75X$ for males, and $Y=0.51X$ for females), 19.7% (13/66) of the male patients and 24.3% (18/74) of the female patients were discriminated in a sector bounded by the $Y$ axis and the radius where 2.9% (10/350) of the male and female controls were assigned (misclassification rate of 0.03, a figure representing a limit of normality in various anthropometric characters). The above formulas of the constellation graphs and the borderlines were applicable to data from the patients in other etiological categories (data not shown). Correct classification rates in the male patients with defined prenatal, perinatal, postnatal

**Table 4** Transformed angles ($\xi j\alpha$) for respective characteristics of dermatoglyphic traits in males[a]

| Variables | Angles for respective characteristics | Weighting length |
|---|---|---|
| 1. True pattern in the right Th/1st ID region | (−): 0, (+): 90 | 0.26 |
| 2. True pattern in the right 2nd ID region | (−): 0, (+): 90 | 0.18 |
| 3. True pattern in the left hypothenar region | (+): 0, (−): 90 | 0.18 |
| 4. Pattern on the right hallucal area | $L^d$: 0, W: 7, $L^t$: 17, $L^f$: 33, $A^t$: 90 Others: 55 | 0.10 |
| 5. Mean a-b ridge count | 52 (maximum): 0, 20.5 (minimum): 90 | 0.24 |
| 6. Total finger ridge count | 236 (maximum): 0, 9 (minimum): 90 | 0.04 |

a: For abbreviations see Table 3.

**Table 5** Transformed angles ($\xi j\alpha$) for respective characteristics of dermatoglyphic traits in females[a]

| Variables | Angles for respective characteristics | Weighting length |
|---|---|---|
| 1. Pattern on the right 3rd finger | $L^u$: 0, W: 7, A: 27, $L^r$: 90 | 0.12 |
| 2. Position of the left axial triradius | t: 0, t': 35, t'': 90, $t^b$: 62 | 0.10 |
| 3. Position of the right axial triradius | t: 0, t': 20, t'': 90, $t^b$: 58 | 0.07 |
| 4. True pattern in the left Th/1st ID region | (−): 0, (+): 90 | 0.08 |
| 5. True pattern in the left hypothenar region | (−): 0, $L^r$ or $L^d$: 8, $L^u$ or $L^c$: 47, Other complex pattern: 90 | 0.10 |
| 6. Abnormal flexion crease on the left palm | (−): 0, (+): 90 | 0.07 |
| 7. Abnormal flexion crease on the right palm | (−): 0, (+): 90 | 0.09 |
| 8. Pattern on the left hallucal area | $L^d$, W: 19, $L^t$: 29, $L^f$: 35, $A^f$: 65, $A^t$: 90, Others: 0 | 0.17 |
| 9. Mean a-b ridge count | 65 (maximum): 0, 20.5 (minimum): 90 | 0.20 |

a: For abbreviations see Table 3.

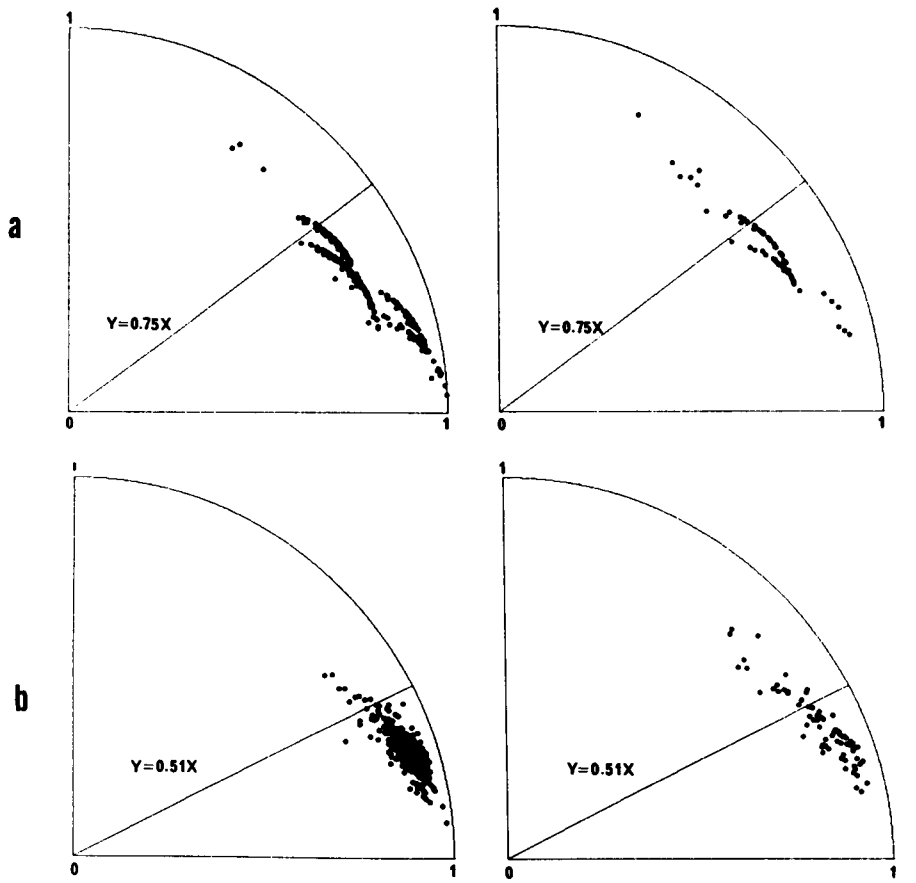


**Fig. 2** Distributions of end points (closed circles) of the subjects on the constellation graph. The normal controls are shown in the left panels and the patients with undefined prenatal etiological factors in the right panels (a: males, b: females). The application of a borderline $Y = 0.75X$ and $Y = 0.51X$ (a misclassification rate of 0.03) leads to a correct classification rate of 19.7% for the male patients and 24.3% for the female patients.

and unknown etiological factors were 20.0% (3/15), 8.8% (5/57), 5.0% (1/20) and 7.7% (2/26), while the values in the females patients were 42.1% (8/19), 18.9% (7/37), 5.1% (1/16) and 13.0% (3/23), respectively. As compared to the normal controls, dermatoglyphic deviation was found to be significantly higher in the undefined prenatal ($P < 0.001$), defined prenatal ($P = 0.005$) and perinatal ($0.025 < P < 0.05$) groups of the male patients, and in the undefined prenatal ($P < 0.001$), defined prenatal ($P < 0.001$), perinatal ($P < 0.001$) and unknown ($P = 0.05$) groups of the female patients. Clinical diagnoses of the discriminated patients were Rubinstein-Taybi syndrome (2 male patients), de Lange syndrome (one male and 2 female patients), Prader-Willi syndrome (one female patient), phenylketonuria (one female patient) and microcephaly (4 female patients) in the defined prenatal group; neonatal asphyxia (5 male and 6 female patients) and kernicterus (one female patient) in the perinatal group; and head trauma (one female patient) and encephalitis (one male patient) in the postnatal group.

## Discussion

In terms of a combination of unusual der-

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matoglyphic traits, the severely mentally retarded patients with undefined prenatal etiological factors were shown to have 7 to 8 times as much dermatoglyphic deviation as did the normal controls. Genetic analysis on the basis of family and twin data has demonstrated that the epidermal ridge and palmar flexion crease patterns are under genetic influence (15-16). Certain dermatoglyphic traits are claimed to be transmitted as dominant (total finger ridge count), incompletely dominant (absence of *c* triradius), or recessive (suppression of axial triradius *t*) gene(s), but it is generally acknowledged that the mode of inheritance in most dermatoglyphic features is compatible with a polygenic (multifactorial) system, with individual genes contributing a small additive effect. The deviation seen in the severe retardates, therefore, may reflect biases of genetic as well as environmental factors acting during the formation of volar ridge patterns and flexion crease.

Patients with so-called 'idiopathic' mental retardation constitute the largest group among moderately to severely retarded individuals. Although the exact causes underlying idiopathic mental retardation are unknown, prenatal factors are thought to be involved in the pathogenesis because of the high incidence of associated congenital malformations (17) and the frequent neuroradiological demonstration of brain structural defects by examinations (18). The fact that dermatoglyphic deviation was present in the undefined prenatal group of patients but not in those with postnatal etiological factors suggests that early intrauterine factors, genetic or nongenetic, play an important role in the pathogenesis of 'idiopathic' mental retardation. The dermatoglyphics of most cases of Rubinstein-Taybi and de Lange syndromes, for which early intrauterine factors are suspected to be responsible (19), conformed to the formula of the graph, further supporting our hypothesis.

Dermatoglyphic abnormalities were not restricted to the group of prenatal etiological factors, but were also observed in the group of perinatal origins, especially patients of the female sex. This result suggests that multiple factors including early prenatal ones are actually involved in the pathogenesis of mental retardation in a substantial number of the patients with presumed perinatal factors. As none of those discriminated had any major or minor malformations, early intrauterine factors might predispose them to birth asphyxia or kernicterus through the immaturity or maldevelopment of vital organs. Dermatoglyphic deviation was found to be more frequent in the female than in the male patients. Since neonatal mortality is less frequent in females than in males (20-21), it is tempting to assume that female patients who develop severe mental retardation due to perinatal etiologies may bear a potent predisposition to brain dysfunction.

The same speculation as in the perinatal group with severe mental retardation has been proposed for in the pathogenesis of epilepsy and cerebral palsy. A dermatoglyphic study of epileptics showed deviations in patients with symptomatic (posttraumatic and alcohol-related) as well as genetic and idiopathic epilepsy, indicating that genetic predisposition to epilepsy is present even in the symptomatic group (22). Further evidence of genetic predisposition to epilepsy is provided by the high incidence of epileptiform EEG abnormalities in close relatives of patients with symptomatic epilepsy (23). In cerebral palsy, too, a sequential multivariate analysis of risk factors has revealed that intrapartum or postpartum factors play only a minor role in the etiology, while defects intrinsic to a fetus are major etiological factors (24).

These reports, coupled with the present study, lead us to suppose that in an appre-

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ciable proportion of patients with developmental disabilities, whose brain dysfunction is apparently due to perinatal or postnatal events, the causes of brain dysfunction may in fact be determined genetically or arise very early in fetal life. Interestingly, despite the recent improvements in obstetric and neonatal care, there has been no consistent decrease in the incidence of cerebral palsy over the past decade or two (25). This fact poses a serious challenge to the researcher to gain a thorough knowledge of responsible factors in order to prevent disabilities such as mental retardation, epilepsy and cerebral palsy.

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