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Dermatoglyphs in Leukaemia

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Ridge differentiation takes place in the foetus during the third and fourth months of intrauterine life, and as a result certain disturbances of foetal growth during this period are recorded by modifications in the ridge configurations. The best example of such prenatal disturbance of ridge pattern is found in Down's syndrome in which there is retardation of growth affecting most parts of the body (Holt, 1961a).

The study of the patterns of the epidermal ridges of fingers, palm, and sole can serve as an aid in the diagnosis of a number of disease conditions, particularly those caused by chromosomal aberrations, but also in others both genetically and non-genetically determined. Findings in exogenous embryopathies following maternal rubella or thalidomide intake are examples of the latter. In clinical conditions generally caused by autosomal trisomy, e.g. Down's syndrome, dermatoglyphic evidence can often be accepted as definite confirmation of a tentative clinical diagnosis. In sex chromosome aberrations, increased frequencies of certain patterns and deviations in ridge counts have been found. Several disorders due to a single abnormal gene have been reported to show dermatoglyphic features significantly varying from normal as have other conditions not definitely shown to be genetically determined. Leukaemia comes into the latter category, and Aleksandrowicz, Schiffer, and Debski (1966), Menser and Purvis-Smith (1969), Rosner (1969), and Wertelecki, Plato, and Fraumeni (1969) have reported their findings.

Patients and Methods

People studied were unrelated British whites and consisted of 110 patients with leukaemia (68 males, 42 females) and 158 normal controls (76 males, 82 females), composed of medical and nursing personnel. A different control group composed of medical, nursing, and administrative hospital staff was used for investigation of abnormal palmar creases (80 males, 80 females), and control values for total finger ridge count (Holt, 1961b) and *a–b* ridge count (Holt, 1968a) are not my own. The age range of the leukaemia patients was 3–89 years, but only 10 were 16 years old or younger; the latter 10 all had acute leukaemia, lymphoblastic in 7 cases, and myeloblastic in the other 3 cases. No patients studied had Down's syndrome. The study was conducted between January 1968 and June 1969. Features studied were percentage frequencies of finger pattern types, total finger ridge count, configuration in the second, third, and fourth interdigital areas of the palm, *a–b* ridge count (i.e. the number of ridges occurring in the second interdigital area between the digital triradii *a* and *b*), and other findings have also been recorded. The total finger ridge count is an inherited metrical character in which a number of perfectly additive genes are concerned and in which environment plays a comparatively small part (Holt, 1968b). The *a–b* ridge count has also been shown to be genetically controlled (Fang, 1950).

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Findings

Frequencies of pattern types of fingers (Tables I–IV and Fig. 1 and 2). Tables I and II show the frequencies of pattern types. It is clear that the only main group with a substantial difference from the controls is the males with acute leukaemia, who show an increased frequency of whorls and a decreased frequency of ulnar loops.

A preliminary analysis by means of the χ^2 test shows that in neither acute nor chronic female leukaemia groups is there a significant difference in the distribution of pattern types from the controls. In the males, however, the difference is highly significant ($p < 0.001$) in acute leukaemia and significant ($p < 0.01$) in chronic leukaemia. In acute leukaemia the main difference is between the proportion of whorls (41·9% in patients and 27·2% in controls) and ulnar loops (49·7% in patients and 62·6% in controls). In chronic leukaemia the main difference is between the proportions of radial loops (7·8% in patients and 4·9% in controls) and arches (1·4% in patients and 5·3% in controls).

These statistical results in males cannot be accepted uncritically as the χ^2 test assumes complete independence of pattern types between prints

TABLE I

FREQUENCIES OF FINGER PATTERN TYPES IN 49 CASES OF ACUTE LEUKAEMIA (M32, F17), 61 CASES OF CHRONIC LEUKAEMIA (M36, F25), AND 158 CONTROLS (M76, F82)

	Left					Right					Total	Per Cent	χ^2	p
	V	IV	III	II	I	V	IV	III	II	I				
Acute leukaemia (males)														
W	7	16	10	15	12	10	20	11	17	16	134	41·9		
UL	24	16	17	8	18	21	11	18	10	16	159	49·7	22·85	< 0·001
RL	0	0	1	6	0	0	0	1	3	0	11	3·4		
A	1	0	4	3	2	1	1	2	2	0	16	5·0		
Chronic leukaemia (males)														
W	3	13	9	9	13	6	13	12	13	14	105	29·3		
UL	32	23	24	12	22	30	22	23	10	22	220	61·5	13·20	< 0·01
RL	0	0	1	14	0	0	0	1	12	0	28	7·8		
A	0	0	2	1	1	0	0	0	1	0	5	1·4		
Controls (males)														
W	11	27	13	23	24	12	32	11	20	32	205	27·2		
UL	63	49	53	24	50	63	42	57	30	41	472	62·6	—	—
RL	0	0	1	18	0	0	0	2	15	1	37	4·9		
A	1	0	9	9	2	1	0	6	10	2	40	5·3		
Acute leukaemia (females)														
W	1	6	0	5	7	3	7	1	9	10	49	28·8		
UL	16	10	14	5	9	14	10	14	4	6	102	60·0	2·48	> 0·05 (NS)
RL	0	1	0	5	0	0	0	0	2	0	8	4·7		
A	0	0	3	2	1	0	0	2	2	1	11	6·5		
Chronic leukaemia (females)														
W	3	8	8	9	7	2	8	5	12	6	68	27·7		
UL	19	17	15	8	15	20	16	17	7	16	150	61·2	7·38	> 0·05 (NS)
RL	0	0	0	5	0	0	0	0	2	0	7	2·9		
A	1	0	2	2	2	2	1	3	4	3	20	8·2		
Controls (females)														
W	8	27	12	26	28	10	31	11	29	31	213	26·0		
UL	70	55	66	26	51	71	48	68	30	49	534	65·2	—	—
RL	2	0	0	18	0	1	2	0	14	0	37	4·5		
A	2	0	4	11	3	0	1	3	9	2	35	4·3		

χ^2 values are determined for leukaemia patterns against controls; number of degrees of freedom is 3.

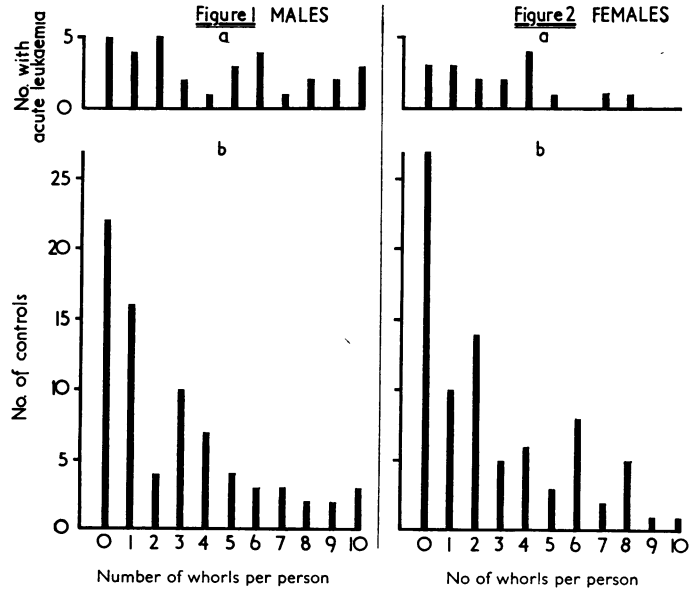

FIG. 1 and 2. Histograms comparing the number of whorls per person in patients with acute leukaemia and in controls.

TABLE II

FREQUENCIES OF FINGER PATTERN TYPES

	Acute Leukaemia	Acute Myeloblastic Leukaemia	Lymphoblastic Leukaemia	Chronic Leukaemia	Chronic Myelocytic Leukaemia	Chronic Lymphocytic Leukaemia	Controls
No. of male subjects	32	15	13	36	14	22	76
No. of readable prints	320	150	130	358	139	219	754
W	134 (41·9%)	62 (41·3%)	60 (46·2%)	105 (29·3%)	48 (34·5%)	57 (26·0%)	205 (27·2%)
UL	159 (49·7%)	76 (50·7%)	57 (43·8%)	220 (61·5%)	78 (56·1%)	142 (64·8%)	472 (62·6%)
RL	11 (3·4%)	2 (1·3%)	8 (6·2%)	28 (7·8%)	9 (6·5%)	19 (8·7%)	37 (4·9%)
A	16 (5·0%)	10 (6·7%)	5 (3·8%)	5 (1·4%)	4 (2·9%)	1 (0·5%)	40 (5·3%)
No. of female subjects	17	9	3	25	13	12	82
No. of readable prints	170	90	30	245	125	120	819
W	49 (28·8%)	31 (34·4%)	4 (13·3%)	68 (27·7%)	36 (28·8%)	32 (26·7%)	213 (26·0%)
UL	102 (60·0%)	52 (57·8%)	23 (76·7%)	150 (61·2%)	77 (61·6%)	73 (60·8%)	534 (65·2%)
RL	8 (4·7%)	2 (2·2%)	3 (10·0%)	7 (2·9%)	2 (1·6%)	5 (4·2%)	37 (4·5%)
A	11 (6·5%)	5 (5·6%)	0	20 (8·2%)	10 (8·0%)	10 (8·3%)	35 (4·3%)

from the same individual. It seems reasonable to expect that some individuals may have a tendency towards particular finger patterns. A more valid test of the significance of the finding of a higher proportion of whorls in acute leukaemics than in controls (and a corresponding decrease in ulnar loops) can be derived by comparing the number of individuals with 0, 1, 2, 3, etc. whorls in the two groups. Fig. 1 shows the distribution of individuals classified in this way; there are fewer males with less than 2 whorls in the leukaemia group than in the controls, otherwise no clear-cut conclusion emerges (possibly because of the relatively small number of individuals). The averages of these distributions are compared in Table III. Proper comparison is impossible owing to the non-normality of the distributions, but the fact that the differences between the mean number of whorls and ulnar loops per person in patients and controls only just reach the 5% level of significance supports the suggestion that the distribution of a particular pattern among the 10 fingers of each individual is not independent.

On the other hand, a study of individual finger proportions (Table I) shows that there is an increased proportion of whorls on *all* fingers in acute leukaemia compared to controls and a decreased proportion of ulnar loops on all fingers in acute leukaemia patients compared to controls.

It may be concluded that while these findings offer a strong suggestion of a real difference in finger pattern frequencies between the male acute leukaemics and normal controls, it would need a larger sample to show conclusively that this is a true characteristic of the disease.

A study of the comparable histograms for the females with acute leukaemia (Fig. 2) shows one feature in common with the males; there are fewer women without any whorls in the controls than in the leukaemics. The difference in the means is however not significant (Table III).

TABLE III

MEAN NUMBERS OF WHORLS AND ULNAR LOOPS PER PERSON IN ACUTE LEUKAEMIA AND CONTROLS

	Mean No. ± SE	Significance
Number of whorls/person		
Males {Acute leukaemia	4·19 ± 0·60	t = 2·18
Males {Controls	2·70 ± 0·33	p < 0·05
Females {Acute leukaemia	2·88 ± 0·56	t = 0·44
Females {Controls	2·60 ± 0·30	p > 0·05 (NS)
Number of ulnar loops/person		
Males {Acute leukaemia	4·97 ± 0·53	t = 2·03
Males {Controls	6·21 ± 0·31	p < 0·05
Females {Acute leukaemia	6·00 ± 0·47	t = 0·93
Females {Controls	6·51 ± 0·29	p > 0·05 (NS)

TABLE IV

DISTRIBUTION AND MEANS FOR RADIAL LOOPS AND ARCHES IN MALE CHRONIC LEUKAEMICS AND CONTROLS

	Total No. of Persons	No. of Arches per Person											Mean ± SE	Significance
		0	1	2	3	4	5	6	7	8	9	10		
Chronic leukaemia	36	33	2	0	1	0	0	0	0	0	0	0	0·14 ± 0·09	t = 2·29
Controls	76	58	7	6	2	1	1	1	0	0	0	0	0·53 ± 0·14	p < 0·05
	Total No. of Persons	No. of Radial Loops per Person											Mean ± SE	Significance
		0	1	2	3	4	5	6	7	8	9	10		
Chronic leukaemia	36	18	10	6	2	0	0	0	0	0	0	0	0·78 ± 0·15	t = 1·71
Controls	76	48	21	5	5	2	0	0	0	0	0	0	0·49 ± 0·08	p > 0·05 (NS)

A similar analysis may be applied to the other main findings, i.e. the apparent increase in radial loops and decrease in arches among male chronic leukaemics compared with controls. Here we are dealing with much less common pattern types and it is even more difficult to draw firm conclusions. Table IV gives the distribution and means for radial loops and arches in male chronic leukaemics and controls. It appears from this analysis that the prevalence of arches is significantly decreased among chronic leukaemics; this may, however, be a reflection of an abnormally high rate (5·3%) in the controls. Thus, in a sample of 500 British males (Holt, 1964) the percentage frequency of arches was 4·3% as compared to 5·7% in a sample of 500 British females. Females do tend to have a higher frequency of arches than males so that in this respect my male control sample is atypical.

Females with leukaemia and, in particular, chronic leukaemia, showed an increased frequency of arches, but in fact only 3 of the 17 females with acute leukaemia showed any arches and only 6 of the 25 females with chronic leukaemia showed arches. It can be seen in Table I that only one whorl (5·9%) was present on digit III in the 17 patients with acute leukaemia. Corresponding numbers and percentages of whorls in the 82 normal female controls for digit III were left 12 (14·4%) and right 11 (13·4%).

Other findings on fingers.

(a) *Six or more arches:* One male with acute myeloblastic leukaemia showed 8 and one female with acute myelomonocytic leukaemia 6. One female with chronic lymphocytic leukaemia showed 8 and another with chronic myelocytic leukaemia 7.

In the control group of 158, one male had 6 arches and one female 7 arches. It is well known that 6 or more arches may be found in normal persons and in several different conditions. Thus, in the population of the United Kingdom 1 out of 1000 males and 1 out of 250 females have arches on all 10 fingers. A further 0·6% of males and 1·7% of females have 7, 8, or 9 arches each (Holt, 1968b). Five of 104 patients with alopecia areata described by Verbov (1968) had 6 or more arches. In trisomy 17 or 18 nearly all the finger patterns are simple arches, many having 10 arches (Uchida, Patau, and Smith, 1962; Penrose, 1963) and a high frequency of arches on fingers has also been found in cases of undifferentiated mental deficiency (Hoefnagel *et al.*, 1963).

(b) *Ten ulnar loops.* One male with acute myeoblastic leukaemia and three with chronic lymphocytic leukaemia. In the controls 15 people (M, 7; F, 8) had 10 ulnar loops.

(c) *Ten whorls.* Three males with acute leukaemia (lymphoblastic (2) and myeloblastic (1)). Four controls (M, 3; F, 1) had 10 whorls.

Mean total finger ridge count (Table V). The values for total ridge count found in the British population range from 0 to about 300 and the differences in mean total ridge count of the order found (Table V) are not significant (at the 5% level).

TABLE V

TOTAL FINGER RIDGE COUNT

	No. of Subjects	Mean Total Finger Ridge Count and Standard Error of Mean	Range of Count
Males			
Acute leukaemia	29	140·9 ± 9·4	37–220
Chronic leukaemia	33	133·3 ± 6·5	53–209
Controls (Holt, 1961b)	825	144·98 ± 1·78	—
Females			
Acute leukaemia	16	125·3 ± 10·7	29–180
Chronic leukaemia	22	122·4 ± 10.7	7–199
Controls (Holt, 1961b)	825	127·23 ± 1·83	—

Palmar configurations (Table VI).

(a) *Second interdigital area (I_2) in 101 patients.* All the patterns were loops and all had an accessory

TABLE VI

FREQUENCIES OF PATTERNS IN SECOND, THIRD, AND FOURTH INTERDIGITAL AREAS OF PALM (OPEN FIELDS, ABSENT TRIRADIUS c, AND VESTIGIAL PATTERNS CONSIDERED 'NO TRUE PATTERNS')

Dermatoglyphic Area	Sex	Acute Leukaemia		Chronic Leukaemia		Controls	
		Left	Right	Left	Right	Left	Right
Second interdigital area (I_2)	M	2 (6·5%)	2 (6·5%)	1 (2·9%)	2 (5·9%)	0	1 (5·3%)
Subjects studied: Acute leukaemia (M31, F16) Chronic leukaemia (M34, F20) Controls (M19, F26)	F	0	3 (18·8%)	1 (5·0%)	2 (10·0%)	0	1 (3·8%)
Third interdigital area (I_3)	M	13 (40·6%)	21 (65·6%)	9 (27·3%)	15 (45·5%)	6 (28·6%)	12 (57·1%)
Subjects studied: Acute leukaemia (M32, F17) Chronic leukaemia (M33. F25) Controls (M21, F27)	F	7 (41·2%)	6 (35·3%)	7 (28·0%)	12 (48·0%)	12 (44·4%)	13 (48·1%)
Fourth interdigital area (I_4)	M	19 (59·4%)	13 (40·6%)	23 (69·7%)	17 (51·5%)	27 (56·3%)	15 (31·3%)
Subjects studied: Acute leukaemia (M32, F17) Chronic leukaemia (M33, F25) Controls (M48, F38)	F	7 (41·2%)	10 (58·8%)	18 (72·0%)	14 (56·0%)	25 (65·8%)	22 (57·9%)

triradius; patterns were more common in right hands. The above is the normal situation found in the second interdigital area. There was, however, an increased frequency of patterns in leukaemic females (36 patients) on the right, viz. 13·9% compared with controls (26 females), viz. 3·8%, but the difference is not statistically significant.

(b) *Third and fourth interdigital areas (I_3 and I_4) in 107 patients.* The differences in pattern frequencies in the third interdigital area in patients and controls were not statistically significant. In both acute and chronic leukaemia patients there were a number of loops with accessory triradii and double patterns noted in the fourth interdigital area, particularly in males, but this did not differ significantly from the findings in the controls, viz. double patterns in the fourth interdigital area were seen in 1 male and 1 female with acute leukaemia and in 3 males and 1 female with chronic leukaemia. The corresponding 86 control subjects showed double patterns in 2 males and 2 females. Males with chronic leukaemia showed an increased frequency of patterns in the right fourth interdigital area compared to controls, but the difference was only of borderline significance (χ^2 3·42). In females with acute leukaemia (17 females), the left fourth interdigital area showed a low incidence of patterns and a correspondingly high incidence of 'no true patterns' compared with the controls (38), but the frequency of patterns compared to controls was not statistically significant (χ^2 2·94).

***a–b* ridge count (i.e. sum of values for both hands** (Table VII). In England the range of *a–b* counts in the general population is from 47–122 ridges. It is normal to find a slightly lower *a–b* count on the right hand than on the left, and the findings in Table VII are in keeping with this. The differences in mean *a–b* ridge count in males between controls and chronic leukaemias, and in females, between controls and acute leukaemias is significant ($p < 0.01$) at the 5% level.

TABLE VII

a–b RIDGE COUNT

	No. of Subjects	Mean *a–b* Ridge Count and Standard Error of Mean	Range of Count	Mean Ridge Count on Left Hand	Mean Ridge Count on Right Hand
Males					
Acute leukaemia	28	82 ± 2·6	50–126	41·3	40·7
Chronic leukaemia	25	80·6 ± 1·5	65–92	40·9	39·7
Controls (Holt, 1968a)	250	85·49 ± 0·66	—	—	—
Females					
Acute leukaemia	15	78·7 ± 1·9	66–98	39·9	38·8
Chronic leukaemia	13	81·2 ± 2·2	67–100	40·8	40·4
Controls (Holt, 1968a)	250	84·88 ± 0·65	—	—	—

Frequency of abnormal flexion creases (Table VIII). Flexion creases are not components of dermatoglyphs but they are often considered with them. The simian line is a modified, distal, transverse, flexion crease coursing continuously from radial to ulnar margins of the palm, which occurs occasionally in normal persons (Cummins and Midlo, 1961) but is often present in Down's syndrome.

The Sydney line is another palmar crease which

TABLE VIII
FREQUENCY OF ABNORMAL FLEXION CREASES

Palmar Crease	Males		Females		Total	
	Leukaemia (63)	Controls (80)	Leukaemia (37)	Controls (80)	Leukaemia (100)	Controls (160)
Simian	0	5 (6·25%)	1 (2·7%)	0	1 (1%)	5 (3·1%)
Sydney	7 (11·1%)	6 (7·5%)	4 (10·8%)	9 (11·25%)	11 (11%)	15 (9·4%)

was described only recently by Purvis-Smith and Menser (1968). It is described as the proximal transverse palmar crease which, instead of terminating approximately over the axis of the 4th digit as is usual, extends to the ulnar margin of the palm. Purvis-Smith and Menser (1968) believe this extended proximal transverse palmar crease to be atypical in a normal population. However, Dubowitz (1969) found Sydney lines in 5 of 14 newborn (and presumably normal) infants. Recently Menser and Purvis-Smith (1969) have said that they consider that Sydney and simian lines may be significant as markers for leukaemia following their studies on 25 children with leukaemia. Wertelecki *et al.* (1969) found an increased frequency of Sydney creases but not of simian creases in their patients with acute lymphocytic leukaemia compared to controls.

My findings (Table VIII) suggest that the incidence of Sydney and simian lines is not significantly different in leukaemia (excluding Down's syndrome) from that in the normal population.

Discussion

It is unlikely that leukaemia is a single disease but a group of diseases perhaps with many different causes. Chromosome studies have been carried out in various types of leukaemia, and chromosomal abnormalities have been found particularly in cases of chronic granulocytic leukaemia and acute leukaemias. Chromosomal abnormalities are not, however, found in all cases of leukaemia by any means. It has been suggested that chromosome 21 may possess one or more gene loci affecting leukopoiesis (Nowell and Hungerford, 1964). Fitzgerald *et al.* (1966) found an inherited chromosomal abnormality in 1 of 12 families with a multiple occurrence of leukaemia and related disorders. This family showed a high incidence of chronic lymphocytic leukaemia. The authors commented that the finding raised the possibility that inherited chromosome abnormality might be an important predisposing factor to leukaemia in some families. However, with regard to the Philadelphia chromosome (Ph^1), work on identical twins suggests that environmental factors are more important than hereditary factors in the development of this autosomal abnormality. Thus, Goh and Swisher (1965) and Woodliff, Dougan, and Onesti (1966) have both reported a pair of identical twins, one of whom had chronic granulocytic leukaemia and the other did not. The Philadelphia chromosome was found neither in cells cultured from peripheral blood nor in bone-marrow in the unaffected twins, but was present in some cells cultured from peripheral blood in the affected twin described by Goh and Swisher (1965) and both in cells cultured from peripheral blood and in bone-marrow cells in the affected patient described by Woodliff *et al.* (1966). These findings provide evidence suggesting that the Philadelphia chromosome is an acquired chromosomal abnormality.

It is fair to say that genetic influence may play a part in the aetiology of some leukaemias, and it is possible that some cases are genetically determined. The increased incidence of leukaemia among patients with mongolism provides some support for genetic factors being involved in the aetiology of leukaemia in view of the chromosomal aberration in Down's syndrome. Miller (1963) found in a survey of sibship histories of 519 leukaemic children that Down's syndrome was more common than usual not only among the leukaemic children but also among their sibs.

Familial leukaemia occurs and many cases of both acute and chronic types have now been reported. For instance, acute leukaemia occurring in three successive generations in a family has been described (Heath and Moloney, 1965), and Fraumeni, Vogel, and DeVita (1969) studied a family with chronic lymphocytic leukaemia in three sibs, the progeny of a consanguineous mating. Their findings led them to suggest that a genetically controlled immune mechanism is involved in familial clusters of chronic lymphocytic leukaemia. However, from investigations among relatives of leukaemia sufferers no definite tendency for leukaemia to recur in families has been shown. Steinberg (1960), in one such investigation, attempted to estimate the role of

heredity in the causation of acute leukaemia in children. He investigated the families of the sufferers finding no evidence of an increased frequency of leukaemia among the patients' relatives.

Aleksandrowicz *et al.* (1966) reported finger-print patterns in 175 patients of unspecified age with various types of leukaemia. They found an increased incidence of radial loops in their male patients but they also found an increased frequency of whorls in females. They did not distinguish the different types of leukaemia in their report.

Rosner (1969) in a study of 188 cases of leukaemia found that females with chronic lymphocytic leukaemia had significantly more arches on their fingers than controls, but his findings in males with chronic lymphocytic leukaemia did not differ from those of normal men. Menser and Purvis-Smith (1969) in a study of 25 children, 24 of whom had acute leukaemia, noted an increased incidence of arches and a decreased incidence of ulnar loops in their patients.

Wertelecki *et al.* (1969) mentioned that they found no significant differences in finger pattern frequencies between a group of patients with acute lymphocytic leukaemia and controls, though the percentage frequency of whorls in their male leukaemics was 37·5 compared to 28·6 in controls.

Many genes are concerned in determining the details of finger- and palm-prints, and the above investigation on dermatoglyphs in leukaemia has been carried out to see whether patients with leukaemia show any characteristic dermatoglyphic features which could perhaps incriminate genetic factors in the aetiology of this condition.

Summary

Dermatoglyphic findings in 110 patients with leukaemia are described. This is the first British series of patients investigated, and there is a breakdown into various types of leukaemia. Pattern types on fingers, total ridge count, palmar configurations, *a–b* ridge count, abnormal palmar creases, and other findings are considered.

Finger pattern frequencies in males with leukaemia were found to differ from those of a normal control group, and the more important findings were an increased frequency of finger whorls and a decreased frequency of ulnar loops in acute leukaemia, and an increased frequency of radial loops and a decreased frequency of arches in chronic leukaemia and in particular chronic lymphocytic leukaemia.

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