DERMATOGLYPHICS IN CONGENITAL HEART MALFORMATIONS

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Dermatoglyphics were analyzed in 141 patients with congenital heart malformations not associated with major non-cardiac malformations, and 100 control children. The results were compared with and, where possible, combined with those of other series. In the combined series, the aortic stenosis group had an increase in whorls and arches, and a decrease in ulnar loops; patent ductus arteriosus a decrease in whorls and an increase in arches; and pulmonic stenosis an increase in arches. However, these differences are too small to be diagnostically useful, and may reflect inadequate matching with controls. Other differences reported as significant in previous studies disappeared in the combined series. In particular there were no significant differences in finger patterns in cases with tetralogy of Fallot, transposition of the great vessels, ventricular septal defect, atrial septal defect and coarctation of the aorta. There was a probable increase in the height of the palmar triradius in cases of tetralogy of Fallot, and a highly significant increase in hypothenar patterns in cases of pulmonic stenosis, aortic stenosis and transposition of the great vessels.

DERMATOGLYPHICS IN CONGENITAL HEART MALFORMATIONS

by

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LIST OF TABLES

TABLE OF CONTENTS

INTRODUCTION

Previous reports in the literature have suggested that the frequencies of certain dermatoglyphic configurations in patients with congenital heart disease differ from those of normal individuals or patients with acquired heart disease. This study compares the dermatoglyphics of patients with various types of congenital heart malformations with those of the normal population and reviews the findings of other workers (Alter and Schulenberg, 1970; Burguet and Collard, 1968; Cascos, 1964, 1965; Ceccarelli et al., 1968; Emerit et al., 1968; Fried and Heel, 1962; Hale et al., 1961; Kontras and Bodenbender, 1965; Laurenti, 1969; Saksena and Kumar, 1968; Saller and Glowatzki, 1967; Takashina and Yorifuji, 1966; Weninger et al., 1966).

MATERIALS AND METHODS

Dermatoglyphic patterns of 141 patients with congenital heart disease were recorded at The Montreal Children's Hospital. The diagnosis in all cases was made by cardiac catheterization and/or operation. Patients with other major congenital anomalies were not included. The diagnostic groups and their abbreviations are shown in Table I.

The control group was 100 children admitted to hospital for complaints not related to any congenital condition. Both groups were heterogeneous as to racial origin, and excluded negroes. Analysis of the controls and CHD patients was done by one individual.

The following comparisons were made between all CHD patients and the controls and between the various types of CHD and controls:

1. Finger pattern distribution. The patterns were classified as whorls, ulnar loops, radial loops or arches according to the number of triradii present.

2. Frequency of a simian crease on the palm. A single transverse palmar crease or two transverse creases joined by a branch line were classified as simian creases.

3. Axial triradius. The height was expressed as a percentage of the total height from the distal wrist crease to the proximal crease at the base of digit three as described by Walker[18]. t is defined as a height of 0-14%, t' 15-39%, and t" greater than 40% of the total height. When more than one triradius was present the distal one was used for the calculation. The presence of multiple axial triradii

was also recorded.

4. Frequency of a pattern (loop or whorl) in the hypothenar area.

5. Frequency of a pattern in the thenar/first interdigital space (thenar/I_1).

6. Frequency of a loop in the third interdigital space.

7. Frequency of a loop in the fourth interdigital space.

8. Hallucal area pattern. The patterns were classified as tibial arch, fibular arch, proximal arch or tented arch; small distal loop ($\leq$ 21 ridges), large distal loop, fibular loop; tibial loop; or whorl. The various arch patterns were combined for statistical analysis.

RESULTS

The results are presented in Tables II-V. Except for Table II, only figures for TF and VSD are presented individually; the other groups were too small to analyze individually. There are no significant differences between the CHD and control groups in patterns on all fingers combined (Table II); frequency of palmar simian crease (6.4% of CHD and 3% of control cases had a simian crease on at least one hand); frequency of high t' and t" distal palmar triradius; frequency of multiple axial triradii; pattern in the hypothenar area; pattern in the thenar/I_1 space, third and fourth interdigital spaces (Table IV); or hallucal area pattern (Table V). The VSD group had an increase and the AS group a decrease in whorls, significant by the heterogeneity Chi square test (Table II).

TABLE I

Diagnostic groups and their abbreviations.

Type of defect	Abbreviation	N	♂ : ♀
Tetralogy of Fallot	TF	34	19 : 15
Ventricular septal defect	VSD	30	13 : 17
Pulmonary stenosis	PS	17	11 : 6
Atrial septal defect	ASD	15	7 : 8
Transposition of the great vessels	TGV	13	10 : 3
Aortic stenosis	AS	8	5 : 3
Patent ductus arteriosus	PDA	6	3 : 3
Coarctation of aorta	CA	5	2 : 3
Other cardiac anomalies	"Others"	13	6 : 7
TOTAL CHD	CHD	141	76: 65
CONTROL		100	57: 43

TABLE II

Percentage pattern types on all fingers in controls and CHD patients.

	W	UL	RL	A
Control	25.0	64.7	6.0	4.3
All CHD	27.1	64.3	5.2	3.4
TF	29.4	60.9	5.0	4.7
VSD**	36.7**	56.7	4.0	2.6
PS	27.1	62.9	8.8	1.2
ASD	16.0	73.3	4.0	6.7
TGV	31.5	60.8	5.4	2.3
AS*	13.7*	75.0	10.0	1.3
PDA	21.7	66.7	5.0	6.6
CA	26.0	68.0	4.0	2.0
"Others"	18.5	76.1	3.1	2.3

* significant at the 5% level

** significant at the 1% level

TABLE III

Percentage distribution of individual finger patterns for control (C); all congenital heart disease patients (CHD); Tetralogy of Fallot (TF); and ventricular septal defect (VSD).

		Whorl				Ulnar loop				Radial loop				Arch			
		C	CHD	TF	VSD	C	CHD	TF	VSD	C	CHD	TF	VSD	C	CHD	TF	VSD
1	R	40	32	27	47	59	66	73	53	0	1	0	0	1	1	0	0
	L	31	30	23	50	65	67	71	47	0	0	0	0	4	3	6	3
2	R	31	36	35	47	39	34	44	30	21	22	15	17	9	8	6	6
	L	26	35	32	47	41	33	27	30	24	24	29	17	9	8	12	6
3	R	14	16	23	23	79	77	71	74	3	2	0	3	4	5	6	0
	L	11	16	15	27	75	76	76	60	5	2	0	3	9	6	9	10
4	R	36	48	59	57	57	50	35	43	4	1	3	0	3	1	3	0
	L	35	37	47	47	62	59	44	53	2	1	3	0	1	3	6	0
	R	16	9	15	7	83	91	85	93	0	0	0	0	1	0	0	0
	L	10	11	18	17	87	89	82	83	1	0	0	0	2	0	0	0

TABLE IV

Percentage frequency of palmar patterns in control and CHD patients.

		Control	CHD	TF	VSD
	N	100	141	34	30
Height of axial triradius	L t'	47.0	41.1	38.2	53.3
	L t"	7.0	12.8	17.7	10.0
	R t'	54.0	45.4	50.0	56.7
	R t"	7.0	12.8	17.7	16.6
Multiple axial triradii	L	17.0	17.0	20.6	20.0
	R	14.0	17.0	17.6	10.0
	Bil.	9.0	9.2	11.8	6.7
Hypothenar	L	29.0	43.3	38.2	43.3
	R	31.0	34.0	32.4	26.7
	Bil.	21.0	27.0	26.5	20.0
Thenar/I_1	L	7.0	10.6		
	R	6.0	4.3		
Interdigital space	3 L	30.0	30.5		
	3 R	49.0	51.1		
	4 L	47.0	53.9		
	4 R	43.0	39.0		

TABLE V

Percentage frequency of hallucal area patterns.

		N	A	SDL	LDL	L fib.	L tib.	W
Left	Control	100	4.0	9.0	33.0	5.0	6.0	43.0
	CHD	141	4.3	17.7	34.7	0.0	12.1	31.2
	TF	34	5.9	20.6	29.4	0.0	8.8	35.3
	VSD	30	0.0	23.3	33.4	0.0	13.3	30.0
Right	Control	100	7.0	10.0	43.0	1.0	7.0	32.0
	CHD	141	7.8	16.3	35.5	0.0	11.3	29.1
	TF	34	0.0	17.6	41.2	0.0	14.7	26.5
	VSD	30	13.3	20.0	26.7	0.0	13.3	26.7

DISCUSSION

1. Finger pattern distributions.

(a) Considering all 10 digits together.

Table VI summarizes the results of this and previous studies on series of CHDs without regard to type of malformation. Cascos (1964) reported a significant difference in his total series as compared to 50 controls. He quotes a p value of 0.001, but we calculate a p value of .975-.99 for his series, using a Chi square test for heterogeneity. Emerit et al. (1968), on the other hand, found a significant decrease in the frequency of arches in their CHD patients ($p = 0.001$). Inspection of Table VI shows no consistent trends in frequencies of patterns associated with the presence of congenital heart malformations, and there is no significant difference in finger pattern distribution between 680 CHD patients and 877 controls in the combined data of Emerit, Cascos, Saksena and Kumar, Laurenti and the present series. It would seem therefore, that the significant differences reported by others are the result either of sampling error or incorrect use of tests of significance.

(b) Considering each digit separately.

Many series did not report figures for individual digits. Emerit et al. (1968) reported a significant increase in whorls on the left second digit, decreased ulnar loops on the right second digit, and increased ulnar loops and decreased whorls on left digit 5. When their data were combined with ours, none of these differences were significant.

(c) Considering specific types of cardiac malformation.

There is no reason to expect all the various types of cardiac malformation, which may be etiologically heterogeneous, to show the same deviations from the normal distribution of finger patterns. Thus, considering the group of cardiac malformations as a whole may obscure any pattern abnormalities characteristic of a particular type.

Table VII summarizes the data for finger patterns, height of axial triradius and frequency of hypothenar patterns for the various groups of CHD.

In the case of Tetralogy of Fallot, although Cascos, Laurenti, Kontras and Bodenbender, and the M.C.H. study reported an increase in whorls, the combined series of Cascos, Laurenti and M.C.H. (which were the only ones for which the data was suitable for pooling) did not differ significantly from the combined controls (29.7% in 109 patients vs. 26.1% in 457 controls).

For Pulmonic Stenosis two series reported an increase in arches, and, even though the M.C.H. series showed a decrease in arches, the combined series of Cascos, Laurenti and M.C.H. (59 patients) showed an increase in arches (8.6% vs. 5.1%; p = .01-.005), significant at the 1% level. Emerit et al. (1968) did not distinguish between cases of isolated CHD and those with multiple malformations; they also report a significant increase in arches. In the study by Weninger et al. there was no increase in arches in their 27 patients.

Table VIII presents the frequency of patients with many, few or no patterns of particular types in the combined series of Cascos,

Laurenti and M.C.H. The combined results indicate an increase of PS patients with 3-10 arches (12% vs. 6%), and with 0 whorls (34% vs. 25%) but the difference is not significant. Cascos (1965) concluded that "a patient with congenital heart disease having more than two arches in their finger-prints was very likely to have pulmonary stenosis". In the combined series the AS and PDA groups also have 12% of cases with more than two arches. Clearly, Cascos' conclusion is not generally valid. A larger series is necessary to confirm whether there is a real increase in arches and, if so, whether patients with arches have other features which distinguish them from other PS patients. For instance, David (1969) claims on the basis of two cases, that familial cases of CHD may show an increase in arches.

For Aortic Stenosis the combined results of Cascos, Laurenti and M.C.H. (40 patients) compared to the combined control gives a p value of .01-.005 with an increase in whorls (32% vs. 26.1%) and arches (8.0% vs. 5.1%) and a decrease in ulnar (55.6% vs. 63.8%) and radial (4.5% vs. 5.0%) loops. Weninger (1966) reported increased whorls but decreased arches for 15 male patients. Emerit et al. (1968) did not find an increase. If there are real differences in this group they are small ones. Cascos (1965) proposed that a patient "with more than four whorls probably had either aortic stenosis, coarctation of the aorta, or Fallot's tetralogy". In the combined series (Table VII) the VSD group had the highest frequency of cases with more than four whorls (30%) and the control value of 24% was comparable with those for AS (25%), CA (26%) and TF (24%).

For patent ductus arteriosus the combined results of Cascos, Laurenti and M.C.H. (34 patients) show a highly significant difference

($p = .005-.001$) with decreased whorls (18.5% vs. 26.1%) and increased arches (8.8% vs. 5.1%). However, Weninger's series show an increase in whorls.

The combined results of Laurenti and M.C.H. for Transposition of the Great Vessels (30 patients) show a difference of borderline significance from the combined controls ($p = .05-.025$) with an increase in whorls (33.4% vs. 26.2%).

In the combined series of Cascos, Laurenti and M.C.H. there were no significant differences from the control in 71 ventricular septal defects, 52 atrial septal defects and 27 coarctation of the aorta.

TABLE VI

Summary of results comparing all CHD to controls for various series.

	No. of CHD pts.	Isol. CHD	I Finger pattern distribution U	R	W	A	II Bil. simian crease	III Axial triradius height	mult.	IV Hypothenar pattern	V IDS different from normal
Hale et al.	157	No						+*	+*	+	
Fried & Neel	50	?						+			
Cascos	150	?	-	+	+	+		+*			
			*(see text)								
Kontras & Bodenbender	69	?						+			
Takashina & Yorifuji	44	?						+	* +		
Weninger et al.	165	?						0		Uni 0 Bil +*	
Ceccarelli et al.	90	?						+*		0	No
Saller & Glowatzki	91	?	U & R								
				+	+	-		+			
Emerit et al.	174	Yes	+	+	+	-*	+	+	+	+*	
	156	No	+	-	-	-	+*	+*	+*	+*	
Saksena & Kumer	32	?						+	* +	+*	
Burguet	98	?					0	+*			
Laurenti	183	Yes	-	-	+	+			+	+	No
Alter & Schulenberg	225	Yes	0	0	0	0	0	+*		+	No
M.C.H.	141	Yes	-	-	+	-	+	+	+	+	No

Legend: (+) increase; (-) decrease; ( ) no data; (0) no change or non-significant change for which direction of change is not known; (*) statistically significant.

TABLE VII

Dermatoglyphic features of various categories of CHD.

Type of defect	Series	No. of cases	Isolated CHD	Finger patterns U	R	W	A	Height of axial triradius	Hypothenar L	R	Bil.	L&R
TF	Cascos	34	?	-	+	+	-	+*				
	Burguet	10	?					+				
	Laurenti	41	Yes	-	-	+	-					+
	Alter	36	Yes					+*				
	M.C.H.	34	Yes	-	-	+	+	+	+	+	+	+
VSD	Cascos	21	?	+*	+	-	-	+				
	Burguet	11	?					+*				
	Laurenti	20	Yes	-	-	+	+					+
	Alter	42	Yes					+*				
	M.C.H.	30	Yes	-	-	+*	-	+	+	-	-	+
PS	Cascos	20	?	+	-	-*	+*	+				
	Burguet	13	?					-				
	Laurenti	22	Yes	+	-	+	+					+
	Alter	17	Yes					+				
	M.C.H.	17	Yes	-	+	+	-	+	+	+	+	+
ASD	Cascos	14	?	+	+	-	+	+				
	Burguet	22	?					+				
	Laurenti	23	Yes	-	-	+	-					-
	Alter	32	Yes					+				
	M.C.H.	15	Yes	+	-	-	+	-	-	-	-	-
AS	Cascos	15	?	-*	-	+*	+	+				
	Weninger	15♂	?			+*					0	
	Laurenti	17	Yes	-	-	+	+					+
	Alter	20	Yes					+				
	M.C.H.	8	Yes	+	+	-*	-	-	-	+	-	+
PDA	Cascos	12	?	+	+	-	+	+				
	Weninger	31	?			+*						+
	Burguet	15	?					+*				
	Laurenti	16	Yes	+	-	-	+					-
	Alter	16	Yes					+*				
	M.C.H.	6	Yes	+	-	-	+	0				+
		11	?	-	+	+*	-	-				

TF	Cascos	34	?	-	+	+	-	+*					
	Burguet	10	?					+					
	Laurenti	41	Yes	-	-	+	-						+
	Alter	36	Yes					+*					
	M.C.H.	34	Yes	-	-	+	+	+	+	+	+	+	
VSD	Cascos	21	?	+*	+	-	-	+					
	Burguet	11	?					+*					
	Laurenti	20	Yes	-	-	+	+						+
	Alter	42	Yes					+*					
	M.C.H.	30	Yes	-	-	+*	-	+	+	-	-	+	
PS	Cascos	20	?	+	-	-*	+*	+					
	Burguet	13	?					-					
	Laurenti	22	Yes	+	-	+	+						+
	Alter	17	Yes					+					
	M.C.H.	17	Yes	-	+	+	-	+	+	+	+	+	
ASD	Cascos	14	?	+	+	-	+	+					
	Burguet	22	?					+					
	Laurenti	23	Yes	-	-	+	-						-
	Alter	32	Yes					+					
	M.C.H.	15	Yes	+	-	-	+	-	-	-	-	-	
AS	Cascos	15	?	-*	-	+*	+	+					
	Weninger	15♂	?			+*					0		
	Laurenti	17	Yes	-	-	+	+						+
	Alter	20	Yes					+					
	M.C.H.	8	Yes	+	+	-*	-	-	-	+	-	+	
PDA	Cascos	12	?	+	+	-	+	+					
	Weninger	31	?			+*							+
	Burguet	15	?					+*					
	Laurenti	16	Yes	+	-	-	+						-
	Alter	16	Yes					+*					
	M.C.H.	6	Yes	+	-	-	+	0					+
CA	Cascos	11	?	-	+	+*	-	-					
	Burguet	8	?					+					
	Laurenti	11	Yes	+	-	+	-						-
	Alter	16	Yes					+					
	M.C.H.	5	Yes	+	-	+	-	-					-
TGV	Laurenti	17	Yes	-	-	+	-						+
	Alter	9	Yes					+					
	M.C.H.	13	Yes	-	0	+	-	+					+

TABLE VIII

The percentage frequencies of patients with no, few and many prints of each type combining the results of Cascos, Laurenti and M.C.H.

Pattern	No. of these patterns per hand	Cascos and M.C.H. only Control 150	VSD (71)	TF (109)	PS (59)	ASD (52)	AS (40)	PDA (33)	CA (27)
A	0	74	75	76	64	71	80	55	67
	1-2	20	22	19	24	25	8	33	33
	3-10	6	3	5	12	4	12	12	0
UL	0	0	0	6	7	10	10	0	0
	1-4	21	25	21	15	17	20	12	26
	5-10	79	75	73	78	73	70	88	74
RL	0	58	69	69	63	67	65	61	70
	1 or 2	42	31	31	37	33	35	39	30
W	0	25	28	21	34	25	23	30	22
	1-4	51	42	55	47	56	52	61	52
	5-10	24	30	24	19	19	25	9	26

The biological significance of the small, but statistically significant differences reported above remains unclear. They may represent no more than inadequate matching of control groups, e.g., for sex or racial background. It would seem, therefore, that finger print patterns are of no value as an aid to diagnosis or clue to etiology.

2. Frequency of simian crease on the palms.

Our findings confirm those of Emerit et al. (1968), who found no significant increase in simian creases in their patients with isolated CHD. In the combined series there is also no significant increase in bilateral simian creases. Burguet and Collard (1968) and Alter and Schulenberg (1970) also report no significant differences in the frequency of simian crease.

3. Height and position of the axial triradius on the palms.

In those series where cases with non-cardiac malformations were excluded, Emerit (1968) found a significant increase in t' triradius but not t". Alter and Schulenberg (1970) report a highly significant increase in the atd angle in their CHD series although this difference would be somewhat less significant had the controls been matched to the CHDs for age. In our series there is a non-significant decrease in t' and increase in t".

In series which did not specify their exclusion of cases with associated malformations, Cascos (1964) found the axial triradius to be significantly more distal and radial than in controls with

p = 0.001 by a X^2 heterogeneity test (though we calculate a p value from his figures of only 0.05–0.02), Burguet and Collard (1968), with CHDs and controls of similar age, and Ceccarelli et al. (1968) likewise report a significant increase in the height of axial triradius for CHDs. Takashina and Yorifuji (1966) and Saksena and Kumar (1968) found a significant increase in cases with either distal displacement or multiple axial triradii. On the other hand, Fried and Neel (1962) who matched their CHD and controls for age did not find the increase to be significant in atd angles greater than 57° in the CHD cases. Weninger et al. (1966) and Saller and Glowatzki (1967) also report no significant increase.

In the series which included cases with associated malformations, a significant increase in distally located axial triradii was reported by Hale et al. (1961) and Emerit et al. (1968).

Since there seems to be some evidence for an increased height or number of axial triradii it becomes of interest to examine the data for specific types of cardiac malformation.

a) Tetralogy of Fallot.

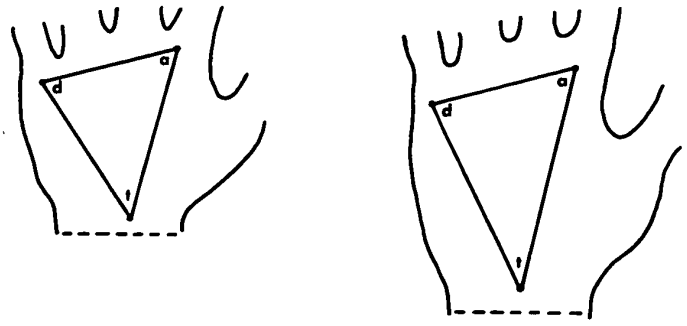
Cascos reports a highly significant difference between his TF and control group, with the triradius in the former being more radial and distal. However, there is no indication that the patients were matched to controls for age, and Holt (1968) has shown that the atd angle is age-dependent ranging from 95.0 in children under 5 years to 85.5 in persons over 14 years.

In order to compare our results with those of Cascos we have measured the angles formed by joining points a t and d on the palm.

The axial triradius was classified as t^{o}, t' and t" from the atd angle, and t^{u} (ulnar), t^{m} (middle) or t^{r} (radial) from the tda angle according to the following criteria used by Cascos.

An atd angle of less than 46^{o} was classified as t^{o}, 46^{o}-70^{o} as t' and more than 70^{o} as t". A tda angle of more than 85^{o} was classified as t^{u}, 76-85^{o} as t^{m} and less than 76^{o} as t^{r}. The triradius can then be assigned to one of nine categories: tu^{o}, tm^{o}, tr^{o}, t'u, t'm, t'r and t"u, t"m and t"r.

Figure 1



Change in atd and tda angle with increasing age of the child.

Figure 1 shows that the atd angle will become smaller and the tda angle larger as the length of the hand increases. Thus, by the above criteria the triradius will be more proximal and ulnar in the older hand and more distal radial in the younger hand.

This can be shown also by plotting the tda angle in the controls against age; 43% of those under six years have a t^r triradius whereas only 17% of those over six have a t^r triradius.

Table IX gives the per cent frequencies of the various types of triradii in 34 TF and 34 matched controls. The 30 VSD cases have a slightly younger age distribution.

TABLE IX

Position and height of axial triradius in cases of TF, VSD and controls.

	t^0u	t^0m	t^0r	t'u	t'm	t'r	t"r
Control	7.5	35.0	7.5	1.5	24.5	22.5	1.5
TF	3.0	33.5	12.0	0.0	16.0	26.5	9.0
VSD	6.5	35.0	10.0	1.5	15.0	25.5	6.5

The TF groups have somewhat more distal-radial triradii than the controls; however the difference is not significant though it would have been if the groups were not age-matched.

In our series, the difference between the VSD and TF groups is insignificant and we can therefore conclude that TF cannot be distinguished from at least one other type of CHD on the basis of the position of the axial triradius. It seems not unlikely that some of the difference reported by Cascos results from failure to match the TF group with the control for age.

Burguet and Collard (1968) report a non-significant increase in the atd angles of their 10 TF patients and Alter and Schulenberg (1970) report a highly significant increase in their 36 patients. Again, however, it is likely that the difference would be less significant had the controls been matched for age.

Nevertheless, the bulk of the evidence suggests that the axial triradius is elevated in TF. The degree of this increase, however, cannot be accurately estimated without matching for age when the atd angle is used as a measurement. In any case, the increase is not great enough to be of any diagnostic value.

b) Ventricular septal defect.

Cascos (1965) reports a significant increase in radial axial triradii at the 5% level. The increase is not significant in our series. The height of the axial triradius is not significantly increased in the series by Cascos, Kontras and Bodenbender, or M.C.H., but is in the series of Burguet and Collard (1968) and Alter and Schulenberg (1970) when measured by atd angle. In the latter series this increase would probably not be significant had the controls and patients been matched for age. There is, then, no convincing evidence that the axial triradius is higher than normal in cases of VSD.

c) Patent ductus arteriosus.

Cascos (1965) reports a non-significant increase in the height as measured by atd angle. Burguet and Collard (1968) and Schulenberg (1970) report a significant increase. It would seem especially important in the latter series to have matched the controls for age since the mean of the PDA groups is only 7.6 years. Our 6 patients, when matched to controls for age show no significant increase in atd angle, but would if they were not age-matched.

d) Other types of cardiac malformation.

No significant increases in height of palmar triradii were found in the case of pulmonary stenosis, atrial septal defect, aortic stenosis, coarctation of the aorta, or transposition of the great vessels.

4. Hypothenar patterns on the palm.

Weninger et al. (1966) found no significant increase in hypothenar patterns in all CHDs as compared to controls but did report a significant increase in bilateral hypothenar patterns in their CHDs when they excluded their 11 patients with TF and 22 with ASD who did not conform to the other groups. However, this is not a statistically valid procedure. Emerit et al. (1968) reported a significant increase in hypothenar patterns for both their isolated CHD group and those with CHD as a part of multiple malformations as compared to controls. Alter and Schulenberg (1970) report a non-significant increase.

The combined results of the studies in which the CHD patients have no other major congenital anomalies and data available for pooling are shown in Table IX. The difference between CHDs and controls is significant with p value .0010-.0005.

TABLE IX

Percentage palms with hypothenar patterns.

	Emerit		Laurenti		M.C.H.		Combined	
	N	%	N	%	N	%	N	%
CHD	174	39.4	183	39.1	141	38.7	498	39.1
Control	200	26.0	101	29.3	100	30.0	401	27.8

Considering individual types of cardiac malformation separately, only Laurenti and the present study provide appropriate data. In the combined series, there is an increase in frequency of hypothenar patterns for pulmonary stenosis (48.7% of 39 cases vs. 29.6% of 201 controls; p = 0.005-0.001), for aortic stenosis (48% of 25 cases; p = 0.025-0.01) and for transposition of the great vessels (50% of 30 cases; p =.005-.001). Non significant increases were found for 75 cases of Tetralogy of Fallot (p = .10-.05), 50 cases of ventricular septal defect, 38 cases of atrial septal defect, and a non-significant decrease in 21 cases of patent ductus arteriosus, and 16 cases of coarctation of the aorta.

CONCLUSION

In conclusion, it appears that in spite of many claims that characteristic dermatoglyphic abnormalities occur with congenital heart malformations, there are relatively few well established associations. It is interesting that the most striking association, an increase in hypothenar patterns, occurs in pulmonic and aortic stenosis and transposition of the great vessels, all of which may result from errors of rotation (Altshuler, 1970). In the tetralogy of Fallot group the increase was not quite statistically significant, and further data are needed.

Cascos (1968) suggested that patients with unusually high frequency of a particular pattern might represent a "genetic fraction". He then compared this group of patients with the rest with respect to correlation of ridge count and atd angle and found that the "genetic fraction" showed higher correlations than the rest. He did not, however, do a similar comparison for a control group. It seems to us that there is no reason to call cases with unusual dermatoglyphic patterns "genetic"; any relevant developmental disturbance could also be environmental. Furthermore, unusual patterns may themselves be familial, though not related to the heart malformation. In any case, the differences in correlation coefficient have such large standard deviations that they are clearly not statistically significant.

Finally, the hope expressed by Cascos, David and others that unusual patterns may be useful in diagnosis is not born out, since none of the differences occurred so often in cases of CHD and so rarely in the normal population that their presence in a patient would be diagnostically helpful.

SUMMARY

Dermatoglyphic patterns were analyzed in 141 patients with congenital heart malformations not associated with major non-cardiac malformations, and 100 control children. The results were compared and, where possible, combined with those of other series. In the combined series, the aortic stenosis group had an increase in whorls and arches, and a decrease in ulnar loops. Patent ductus arteriosus cases showed a decrease in whorls and an increase in arches. However these differences are so small that they are not diagnostically useful, and may represent nothing more than inadequate matching with controls. There were no significant differences in finger patterns in cases with tetralogy of Fallot, transposition of the great vessels, ventricular septal defect, atrial septal defect and coarctation of the aorta. There was no significant difference in frequency of bilateral simian crease in congenital heart disease. A probable increase in height of the axial palmar triradius was found in cases of tetralogy of Fallot, and there was a highly significant increase in hypothenar patterns in cases of pulmonic stenosis, aortic stenosis and transposition of the great vessels.

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