The rear end of the upper jaw surrounding the hinge of the clip was defective, and broke when the clip was opened after removal of the spring. The manufacturers (Rocket) examined the clip and confirmed that the pivot was broken. Nevertheless, they thought that this would not have made the device fail, since the pin and pivot are only mechanical contrivances to apply the clip. The spring function was within accepted limits. The clip failed in the approximation of the two jaws, but whether this was secondary failure due to distortion of the moulding after the pivot had been broken could not be determined.

Comment

Sterilisation with Hulka-Clemens clips is well established. It is relatively simple to perform, avoids the dangers of diathermy, and minimises tubal damage, which is important if reversal of sterilisation is to be considered.² Pregnancies may occur after sterilisations of all types, and after surgical division or diathermy pregnancy is usually due to formation of tuboperitoneal fistulae.³ Failure of the spring has caused failed clip sterilisation.¹ This case shows that the plastic jaws are susceptible to breakage or distortion.

Transmigration of sperm with fertilisation and implantation in the contralateral tube is another interesting feature of this case. This phenomenon has been doubted,⁴ but transmigration of sperm⁵ or ova was the only satisfactory explanation of pregnancy in three other reported cases. If tubal pregnancy follows any sterilisation procedure both Fallopian tubes should be considered as possibly patent and so religated, preferably by the Irving technique, which has the lowest failure rate³ of methods of tubal sterilisation.

¹ Hulka, J F, *et al*, *Fertility and Sterility*, 1975, **26**, 1122.

² Lieberman, B A, *et al*, *Journal of Reproductive Medicine*, 1977, **18**, 241.

³ Garb, A E, *Obstetrical and Gynecological Survey*, 1957, **12**, 291.

⁴ Jeffcoate, N, *Principles of Gynaecology*, 4th edn, p 215. London, Butterworths, 1975.

⁵ Cortes, P J, *Acta Ginecológica*, 1972, **23**, 671.

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Dermatoglyphics in children with febrile convulsions

It is widely accepted that dermatoglyphics are inherited in a polygenic manner¹ and that the final dermatoglyphic pattern is set by about the 19th week of gestation.² Any environmental factor accounting for an increased familial prevalence of dermatoglyphic abnormalities must therefore operate in early intrauterine life. The presence of dermatoglyphic abnormalities in children with febrile convulsions and in their parents would indicate the presence of genetic factors and possibly shed new light on the genetic mechanisms. This is the first report of such an investigation.

Subjects, methods, and results

The dermatoglyphics of 513 Chinese parents and children were studied. They comprised 400 schoolchildren (200 boys and 200 girls) who served as controls; 76 children with febrile convulsions admitted to the university department of paediatrics (54 with up to four episodes recorded, and 22 with more than four episodes recorded, often with afebrile seizures as well); and 37 parents of children with febrile convulsions. Patterns in the thenar and first to fourth interdigital areas were classified as proposed by Plato and Wertelecki,³ except that in the fourth interdigital area only unusual patterns were studied (patterns coded 0-4 were excluded).

The children with up to four episodes of febrile convulsions showed increased prevalences of several patterns (see table). A pattern was present in the third interdigital area in 16 (29.6%) of these children compared with 66 (16.5%) of the controls ($P < 0.05$); in 14 cases the pattern was a radial loop. Unusual patterns in the fourth interdigital area were found in 11 (20.4%) of

Percentage prevalences of dermatoglyphic patterns in subjects studied

	Controls (n = 400)	Group with febrile convulsions		
		Children with ≤ 4 episodes (n = 54)	Children with > 4 episodes (n = 22)	Parents (n = 37)
Fingertip patterns				
Whorls	40.2	42.0	44.5	43.2
Ulnar loops	54.4	54.5	50.9	53.3
Radial loops	3.4	2.4	3.2	2.4
Arches	2.0	1.1	1.4	1.1
Palmar dermatoglyphics				
Thenar/1st interdigital area, presence of pattern	0	1.9	0	0
2nd interdigital area, presence of pattern	3.0	3.7	4.5	0
3rd interdigital area:				
Presence of pattern	16.5	29.6*	13.6	32.4*
Radial loop	15.5	25.9	13.6	29.7
4th interdigital area:				
Presence of unusual pattern	5.8	20.4**	9.1	8.1
Radial/accessory loop	3.8	16.7*	9.1	5.4
Accessory triradius	1.3	3.7	0	2.7
Hypothenar area:				
Presence of pattern	13.5	7.4	4.5	29.7*
Radial loop	9.5	5.6	4.5	18.9
Absence of c triradius	13.0	13.0	4.5	10.8
Accessory t triradius	1.3	0	0	8.1
Distal t triradius ($\geq 30^\circ$ palmar height)	6.0	5.6	9.1	18.9*
Simian crease	9.8	13.0	36.4*	16.2
Sydney crease	2.0	9.3	4.5	5.4
Bilateral distal border termination of distal transverse crease	12.5	29.6**	13.6	13.5
Radial border termination of thenar crease	8.3	13.0	13.6	16.2

Significance of difference from controls: * $P < 0.05$; ** $P < 0.01$.

the children with up to four episodes but in only 23 (5.8%) of the controls ($P < 0.01$). The radial/accessory loop was the most common pattern (nine patients (16.7%) compared with 15 (3.8%) of the controls; $P < 0.05$). Bilateral distal border termination of the distal transverse crease was present in 16 (29.6%) of the children with up to four episodes compared with 50 (12.5%) of the controls ($P < 0.01$). The only abnormality found in the children with more than four episodes of febrile convulsions was a greater prevalence of the simian crease than in the controls (eight cases (36.4%) compared with 39 (9.8%); P close to 0.01).

The parents of the children with febrile convulsions also showed a greater prevalence of a pattern in the third interdigital area (12 cases (32.4%)) than the controls (66 cases (16.5%)) ($P < 0.05$). Again the most common pattern was the radial loop (11 cases). Two unexpected dermatoglyphic abnormalities were also found—namely, an increased prevalence of a pattern in the hypothenar area (11 cases (29.7%) compared with 54 (13.5%) among the controls; $P < 0.05$), in most cases (7) the pattern being a radial loop; and an increased prevalence of distal location of the axial t triradius (30% or more of palmar height) (seven cases (18.9%) compared with 24 (6%) among the controls; $P < 0.05$).

Comment

The significantly increased prevalence of dermatoglyphic abnormalities in children with febrile convulsions and in their parents suggests a polygenic mode of inheritance. Thus the view that febrile convulsions are inherited by a single dominant gene with incomplete penetrance⁴ needs to be re-examined. The difference in dermatoglyphic patterns between children with up to four and more than four episodes of febrile convulsions (often with afebrile seizures as well) suggests that there are two types of febrile convulsions—namely, non-epileptic and epileptic. This is supported by recent work on the oculocardiac reflex in febrile convulsions.⁵ The view that most febrile convulsions have epileptic mechanisms also needs to be re-examined.

Further work on the dermatoglyphics of children with febrile convulsions may well result in a clinical method of distinguishing non-epileptic from epileptic varieties.

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² Mulvihill, J A, and Smith, D W, *Journal of Pediatrics*, 1969, **75**, 579.

³ Plato, C C, and Wertelecki, W, *American Journal of Physical Anthropology*, 1972, **37**, 97.

⁴ Frantzen, E, *et al*, *Neurology*, 1970, **20**, 909.

⁵ Stephenson, J B P, *British Medical Journal*, 1978, **2**, 726.

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