

He married a normal, and of their children six were normal and two others had several undeveloped teeth. A normal sister had eighteen children, one of whom was without teeth or hair. Fürst (*Arch. f. Rass.- u. Gesells.-biol.*, 1905, xvi, 310) describes another case of anodontia.

Any possible connexion of rickets in the etiology of this condition need not be considered here, as a hereditary genetic defect displayed in three successive generations cannot possibly be due to such or to any other form of malnutrition or vitamin deficiency.

From all the foregoing cases this family group is essentially different, in that the primordia of the teeth are not suppressed, being present in the gums even when dentition is either greatly delayed or never completed. Sweating is normal here, and the hair is as abundant in all the affected members as it is in ordinary members of the general community. It is just conceivable that as the teeth are epiblastic appendages, and the corneal epithelium and the lens itself are also epiblastic in origin, the eye defect in this family and its inherited dental condition may be associated characters, joined or linked together in the process of heredity. Whether this is so or not must at present be left an open question, though the association itself is certainly suggestive.

I have to thank Dr. Veitch Clark, the medical officer of health, and Dr. Maude Duckworth, at whose infant welfare centre under the Manchester Corporation the infant was seen, for permission to publish notes of the above case; and to acknowledge to R. R. Gates's *Heredity in Man* (1929) my indebtedness for references to the literature.

HEREDITY AND DUPUYTREN'S CONTRACTION

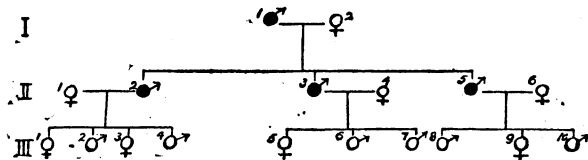
BY

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The hardening and contraction of the palmar fascia, resulting in flexor contraction of the fingers named after Dupuytren, has been known for many years to have had a hereditary factor in its etiology. This factor has been thought usually to be of a gouty nature, influenced by occupation. It is rare to come across three brothers affected with the deformity, whose father was also affected, and whose mother was affected by double-bent little finger at the proximal interphalangeal joint, but whose other fingers were normal.

The father died at the age of 73, and the mother at the age of 63. All the affected followed different occupations. The father was a grocer, and the brothers were a baker, clerk, and general labourer, their present ages being 72, 70, and 68 respectively. There is no history of gout in the family. The pedigree is as follows (a female infant in Generation II, who died in infancy, is not shown):



Description of Members of Pedigree

I 1: Both hands affected; third and fourth fingers contracted; began at about the age of 50.

I 2: Both little fingers (fourth) bent at proximal interphalangeal joint; said to be from birth. Other fingers unaffected.

II 1, 4, 6: Unaffected.

II 2: Both hands affected; right first and fourth fingers, noticed first at the age of 57; left first, second, third, and

fourth, noticed first at the age of 37, after receiving injury to second finger.

II 3: Both hands affected; noticed first about the age of 40. Right first, second, and third fingers; left second and third fingers. Both fourth or little fingers are bent at the interphalangeal joints similar to his mother's.

II 5: Both hands affected; began at the age of 48. Right third and fourth fingers; left second, third, and fourth fingers.

III 6: Died in infancy.

III 7: Died, aged 23, unaffected.

III 1, 2, 3, 4, 5, 8, 9, 10: All unaffected—ages from 41 to 33.

It will be observed that there is some slight variation in the degree of deformity. II 2 and II 5 have the greatest deformity in the left hand, while in II 3 it is the right hand which is most affected. In all three the fourth or little finger is affected, and it is difficult to decide whether the maternal character as well as the paternal is shown, or whether the maternal factor has reinforced the paternal factor so as to produce the very marked deformity in all three brothers.

So far the deformity has not appeared in the third generation, but this generation has hardly yet reached the age when it begins to be noticed. The late period in life—advanced maturity—when the deformity appears and continues to progress, illustrates the potency of the hereditary factor which lies dormant during childhood, adolescence, and maturity.

There may be other defects or diseases appearing late in life which have a hereditary basis, but are not so easily recognizable clinically as Dupuytren's contraction. Owing to the difficulty of disentangling and analysing the various factors these defects or diseases are attributed chiefly to environmental causes.

Senescent hereditary defects or diseases are more difficult to study with the completeness of those occurring in earlier years, because many members of families do not live long enough for the defect to appear, and develop to such a degree as to be recognizable.

DISSEMINATED ENCEPHALO-MYELITIS FOLLOWING SPINAL ANAESTHESIA

BY

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Although spinal anaesthesia was introduced by Bier of Bonn in 1889 the method has been generally employed only since the discovery of stovaine in 1904. Toxic effects at the time when stovaine was on trial, and its known action in lowering the blood pressure and depressing the cardio-vascular and respiratory systems, discouraged many surgeons for a time from the employment of spinal anaesthesia. For many years it has been successfully used by those aware of its dangers and of the technique of administration; and in a large field of surgery it now appears to be the anaesthetic of choice. Those who employ this method have recorded series of thousands of cases without any immediate ill effects or sequelae, and the majority of deaths which have occurred under spinal anaesthesia have been due to the state of the patient prior to operation, shock, cardiac disease, and to loss of blood, etc.—conditions which would make the operation more hazardous if performed under chloroform or ether.

Headache, paraesthesiae, and even pain in the trunk and limbs, not uncommonly follow spinal anaesthesia. More rarely paralysis of an ocular muscle occurs, but recovery is usually complete within a few weeks. More