many specimens have to be taken and but few are positive. The number of cases, however (now amounting to thirteen), from which actinomyces has been obtained from the excised spleen or blood in acholuric jaundice, splenic anaemia, and Banti's disease makes it probable that the present organism bears an aetiological relation to the disease.

This is the first occasion on which a known pathogenic species of actinomyces has been isolated from cases of jaundice. It also appears to be the first occasion on which *Actinomyces caprae* has been found in a human case. *Actinomyces caprae* was first isolated from the lung of a goat by Silberschmidt (1899). Galli-Valerio (1912) obtained it from the same source.

An attempt has been made to identify agglutinin to *Actinomyces caprae* in the blood of the patient. The organism is particularly difficult to emulsify. A thin emulsion, however, made by continual shaking of a twenty-four-hour broth culture showed no agglutination as tested against the serum of Case I. The serum of Case I added to a broth culture of the organism increased the rate of growth and brought out more prominently the characteristic colour of the organism as compared with six normal control sera. Again, the siderotic nodules in the spleen on first exposure were a buff colour, but on being incubated in culture tubes under aerobic conditions they assumed the same tint as an old culture on agar.

The patients come of peasant stock and were born in a cottage which had been part of an old farm; it is said to have been very insanitary, and has since been pulled down. A neighbour used to keep ten to twelve goats, and occasionally these used to stray into the garden of the cottage occupied by the patients. There is no history of their having drunk goats' milk.

Summary

Two cases of acholuric jaundice are described in which splenectomy had the effect of completely relieving the jaundice, restoring the haemoglobin and number of red corpuscles to normal, and diminishing the fragility. From the spleen of one of these patients an organism was isolated which gave the characteristic reactions of *Actinomyces caprae*. It is concluded that this organism was an essential pathogenic agent in the production of the disease.

[Note.—Since the above was written a sister of the patients (aged 17), referred to in the text as normal, has come under observation with jaundice, splenomegaly, and a haemoclastic crisis.]

REFERENCES

- Galli-Valerio, B. (1912). *Zbl. Bakt.*, **63**, 555.
Silberschmidt (1899). *Ann. Inst. Pasteur*, **13**, 841.

On June 16 King Gustaf V of Sweden will celebrate his eightieth birthday, and a national subscription has been opened to celebrate the occasion. In accordance with the King's own desire the funds collected will be used for research into and for combating infantile paralysis and rheumatic diseases. Subscription lists have been issued throughout the country and also in Swedish Legations and Consulates abroad, thus giving an opportunity to Swedes, whether at home or in foreign countries, to contribute. A similar appeal on the occasion of King Gustaf's seventieth birthday brought in the splendid total of just over 5 million kronor (£275,000 at par). The fund on that occasion was earmarked for cancer research and the establishment of cancer clinics. Large sums from this fund have been spent on purchase of radium, and some of the projected clinics have already been opened.

FAMILIAL CLUBBING OF THE FINGERS AND TOES

BY

D. R. SEATON, M.A., M.B.

House-physician, St. James's Hospital, Leeds

Although it is not uncommon to find clubbing of the fingers in the absence of apparent cause (I have seen two instances during the past year), reports of such cases are infrequent, but they have been described by Mangelsdorf (1885), West (1897), and Lewy (1921). Decloux and Lippmann (1902) recorded the occurrence of clubbed fingers and toes in a man aged 55 and in his sister aged 30; there were sixteen other brothers and sisters, who were said to be unaffected, as were their parents and the man's children. None of these was seen. The affected pair said that the condition had been present since birth; neither had any pulmonary or cardiac disease. Weber (1919) put on record the cases of twin brothers aged 25 with clubbed fingers; another brother, aged 30, was not seen, but was said to be likewise affected. All were quite healthy.

Cases occurring in Two Successive Generations

Cases occurring in two successive generations have been recorded in six instances. Fraentzel (1888) described a healthy man showing "drumstick" fingers, his father and his sister having the same condition. Freytag (1891), in his monograph on clubbed fingers, mentioned two instances of father and son being affected in the absence of disease. Weber (1919) found clubbing of the fingers and toes of a man aged 25 and his father; three brothers and a sister had a like condition. Crouzon and Gutmann (1931) reported clubbed fingers in a healthy man aged 30; a radiograph of his hands showed normal bones. The father had been similarly affected, but had suffered from a chronic "broncho-pulmonary" condition. Kayne (1933) published a record of a family in which two brothers, a sister, and the father had clubbed fingers and slightly clubbed toes; the mother and four sisters were normal. The patient's Wassermann reaction was negative, as was a radiograph of his chest. Radiography of his hands showed only slight splaying of the terminal phalanges. In the same year Ragins and Freilich (1933) described a family in which a man, his sister, father, and a paternal uncle all had clubbed fingers and toes.

Witherspoon (1936) reviewed the articles on the subject and reported a negro family in which four brothers and their father had clubbed fingers and toes. One of the brothers had two children, aged 2 months and 17 months, who were found to have pronounced thickening of the volar pads of the fingers and thumbs, but no true clubbing. A maternal half-brother was said to be affected. The mother and four sisters had normal fingers and toes. Wassermann reactions were negative, and radiographs of the chest were normal in all cases examined. Neurath (1932) described four families in which clubbing of the thumbs and big toes was inherited as a simple Mendelian dominant. The clubbing in these cases was due to a bony malformation of the terminal phalanges, and was a special variety of brachyphalangia; for this reason it is not comparable with the present series.

Anomalous Inheritance

In the foregoing reports the inheritance is straightforward, from father to son; but there are two families described in which the inheritance is apparently anomalous.

In 1902 Newton and Mercellis published the record of a Hungarian family in which a man aged 33, his four children, his brother, and his paternal grandfather had clubbing of the fingers and toes. The patient's father, who had been asthmatic, had died of cholera at the age of 36, and was said to have had normal fingers. On the paternal side the family had suffered from cough and shortness of breath, and had as a rule died of some lung complaint. The patient, who at this time was suffering from an indolent axillary abscess, said that his fingers and toes had been clubbed since birth. He had contracted a venereal sore at the age of 31, and had been refused admission to the Army because of a "weak chest"; he had a chronic morning cough, otherwise his health was good. Three of his four children had died in infancy, but all were said to be affected. The remaining child, a girl aged 4, was found to have clubbed fingers and toes, and also "acute endocarditis." In 1911 von Eiselsberg described a man aged 30 with clubbed fingers and toes, which he had had "since birth"; a grandfather, an uncle, and a sister were said to be similarly affected. Nothing was said about the man's parents or other relatives.

The following report of a case admitted to St. James's Hospital, Leeds, shows the condition to have been present in three successive generations.

Case Record

A man (I, 2 in the pedigree) aged 42, a piano-tuner, was admitted on April 10, 1937, suffering from a left hemiplegia, which had come on suddenly the previous day. Before this he had enjoyed good health, apart from occasional attacks of pain and swelling in his right great toe, which he had been told was gout, and several momentary bouts of dizziness during the month preceding the stroke. On examination it was found that his heart was enlarged, the apex beat was $4\frac{1}{2}$ inches from the mid-sternal line in the fifth left interspace, his blood pressure was 230/150 mm. Hg, the Wassermann reaction was negative, and the urine was normal. Apart from the hemiplegia there was nothing of note except an unusual degree of clubbing of the fingers and toes. Questioned regarding these, he said that they had started to enlarge at the age of 21, and had quickly attained their present size, which merited the epithet "drumstick"; the nails had a livid tinge, and were curved like an inverted watch-glass. A radiograph of the chest showed left ventricular hypertrophy and normal lung fields; radiography showed no changes in the bones of the hands. The patient was aware of his abnormality, and said that his elder brother and his father had the same condition. His brother (I, 1 in the pedigree), aged 53, was next interviewed. He had lost three fingers of one hand in an accident, but the remaining fingers and his toes showed the same degree of clubbing as his brother's. He said that he had had the condition as long as he could remember. His health has always been good apart from recent slight winter cough, and he has no physical signs of disease at present. The third brother (I, 3) was not seen, but was said to be unaffected; he has been out of touch with the family for twenty years. The father (AI) died at an advanced age of cancer of the throat; both of his sons agreed that he had clubbed fingers. Nothing was remembered of the grandfather (AII), but a battered photograph of a family group showed quite plainly that both he and the father (AI) were affected. The patient's niece (DI) aged 29, was seen and was found to be normal.

This is the second family to be described in which the deformity runs directly through three generations. In the case of a hereditary condition this is sufficient to show that the abnormality is probably inherited as a simple Mendelian dominant: all the previously reported instances conform to this hypothesis, with the exception of those which I have called anomalous. In the family described by Newton and Mercellis the patient said that his paternal grandfather was affected, but that his father was not.

Assuming that he was not mistaken, it is possible that the father had died before the anomaly appeared, as in one of the cases that I have described the condition was not evident until the age of 21, and in one of West's cases not until after 50, though this was not shown to be familial. Alternatively the clubbing may have arisen out of the lung

The patient's hands, showing the degree of clubbing of the fingers.

Genealogical table, showing presence of clubbing in three successive generations.

diseases to which this family was subject, although this seems unlikely. In von Eiselsberg's report no information is given except the bare facts abstracted above, so it does not afford enough material for discussion; but the argument that the patient's father or mother may have died before their fingers became altered applies here also.

Conclusion

As in most cases the normal members of the families have not been recorded it is impossible to compare their numbers with the numbers of affected persons; in the fully reported families the ratio of affected to unaffected persons is 15:10, which is as near to the 1:1 ratio of Mendelian dominants as one can expect with such a small number. At all events it can be said that extreme clubbing of the fingers and toes may occur in the absence of organic disease, and that the probability is that in some of these cases the condition is inherited as a simple Mendelian dominant.

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REFERENCES

- Crouzon, O., and Gutmann (1931). *Bull. Mém. Soc. méd. Hôp. Paris*, **47**, 1765.
 Decloux and Lippmann, A. (1902). *Ibid.*, **19**, 80.
 Fraentzel, O. (1888). *Dtsch. med. Wschr.*, **14**, 651.
 Freytag, A. (1891). *Über die Trommelschlagfinger und Knochenveränderungen bei chronischen Lungen- und Herzkrankheiten*, Bonn.
 Kayne, G. G. (1933). *Proc. roy. Soc. Med.*, **26**, 270.
 Lewy, E. (1921). *Med. Klinik*, **17**, 845.
 Mangelsdorf, J. (1885). *Wien. med. Wschr.*, **35**, 361.
 Neurath, R. (1932). *Wien. klin. Wschr.*, **45**, 1210.
 Newton, R. C., and Mercellis, E. (1902). *Int. Clin.*, 11th s., **4**, 153.
 Ragins, O. B., and Freilich, E. B. (1933). *Ann. intern. Med.*, **6**, 946.
 von Eiselsberg (1911). *Münch. med. Wschr.*, **58**, 1591.
 Weber, F. Parkes (1919). *British Medical Journal*, **2**, 379.
 West, S. (1897). *Trans. clin. Soc. Lond.*, **30**, 60.
 Witherspoon, J. T. (1936). *Arch. intern. Med.*, **57**, 18.