combination of these, as the clinical signs accompanying the clubbing varied considerably. In the case reported above there was, clinically, obstruction of the vessels as shown by oedema, venous dilatation, pulse, and blood pressure. Subsequently, pressure on the nerves gave rise to pain.

Ogle (1859) thought that the clubbing was due to the presence of an increased amount of venous blood in the affected limb and that this was caused by interference with the axillary circulation. Other observers thought it was due to interference with the sympathetic nerves running with the vessels, but as it is now known that sympathetic nerve fibres do not run down the whole length of the arteries this theory seems untenable.

Mendlowitz (1938) investigated the circulation in the arms of 4 cases of unilateral clubbing and found no evidence of a circulatory cause. He compared the maximum heat elimination and blood pressure gradient from brachial to digital areas in both the normal and affected arms and found that no significant changes in the blood flow had occurred in the affected finger-tips. However, only 4 cases were investigated, and the results of the experiments were not very conclusive. Local anoxaemia due to circulatory embarrassment still remains a very probable cause of clubbing.

Chemical causes, such as toxæmia, seem very probable when bilateral cases are considered. Thus, bronchiectasis, a disease with general toxæmia, is commonly accompanied by bilateral clubbing. However, if general toxæmia were always the cause, unilateral clubbing should never occur. There remains the possibility that the localized pressure, for instance from an aneurysm, might precipitate incipient clubbing on one side, and that the other side might eventually show clubbing if the patient lived long enough. It is interesting to note that cases of an aneurysm causing pressure and pronounced clubbing in one hand have sometimes shown slight clubbing of the other hand (Béclère, 1901; Hatzieganu, 1923). It seems unlikely that pressure on the nerves of the arm could be the cause of clubbing, as although nervous symptoms are common in unilateral cases they do not occur in bilateral cases.

Finally, it may be noted that in 2 of the reported cases the clubbing disappeared wholly or partially after operation on the aneurysm (Hatzieganu, 1923; Högler, 1920). This is convincing evidence that pressure is the primary factor in unilateral cases. Local circulatory embarrassment follows this pressure, causing anoxaemia and, hence, probably the clubbing. Anoxaemia from circulatory embarrassment or general toxæmia in bilateral cases may well account for the clubbing, so conforming with the final factor in unilateral cases.

Summary

A case of unilateral clubbing of the fingers is described.

The cases previously reported are tabulated.

The difficulty of explaining the exact causation is stressed and the possibilities are discussed.

I wish to thank Prof. E. J. Wayne for permission to publish this case, and Dr. L. C. D. Hermitte for the photographs.

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Medical Memoranda

Cirrhosis of Liver and Perforated Gastric Ulcer in an Infant of 6 Months

The only type of infantile cirrhosis which I can trace in medical literature is one which occurs in India. It was first described by Twining in 1832, and its morbid anatomy was investigated by Gibbons in 1891. A full account of this disease is given by Sir Upendranath Brahmachari and Phanindranath Brahmachari in volume 8 of the *British Encyclopaedia of Medical Practice*. I can find no record of a similar condition in this country, nor of any case in which the terminal event was a perforated gastric ulcer, though Brahmachari refers to gastric and intestinal haemorrhage as a common complication. A non-syphilitic cirrhosis, originating as an intralobular and progressing to a multilobular cirrhosis, occurring in an infant of this age and terminating in a perforated pyloric ulcer at 6 months must be very uncommon in this country; and if the brother's liver was also cirrhotic the condition must be, as in the Indian cases, familial. Two other children appear to be healthy.

CASE RECORD

A male child aged 5½ months was sent to me by Dr. Bruce of Newport. The history was that he weighed 9 lb. at birth, and that, beyond a somewhat intense icterus which persisted for some days, the puerperium was normal. It was found, however, that the infant did not thrive on the breast. Dried milk preparations did not result in improvement, and on these the stools were fatty in appearance. On sweetened condensed milk he thrived for a time, but on resuming dried milk he lost weight. It was found that the liver was very large, extending to the umbilicus. No free fluid was detected in the abdomen, and there was no enlargement of the spleen. There had been no jaundice since the neo-natal period. A former child of the same family had also had a much enlarged liver, and had died of pneumonia at 2 years of age. A provisional diagnosis of von Gierke's (glycogen) disease was made.

The blood sugar was first investigated. The fasting blood sugar was 100 mg. per 100 c.cm.; one hour after a feed of 5 oz. ostermilk it was 225 mg., and after two hours 160 mg. Next the effect of adrenaline was observed, for in von Gierke's disease this drug does not raise the blood sugar. On this occasion the fasting blood sugar was 140 mg.; 5 oz. of adrenaline 1 in 1,000 raised this to 160 mg. The urine contained glucose and acetone. The blood count was: red cells, 3,700,000 per c.mm.; haemoglobin, 80%; white cells, 12,400 per c.mm. (polymorphs 29%, lymphocytes 70%, large mononuclears 1%). The Wassermann reaction was negative. I concluded that this was not a case of von Gierke's disease, but a true diabetes mellitus with hepatomegaly, and decided to try the effect of 3 units of soluble insulin twice a day. On the second day the infant suddenly collapsed and died at 11.15 a.m.—four hours after his third dose of insulin. Just before death he passed a quantity of blood per rectum. At no time did he seem to have any pain.

Post-mortem Examination.—The abdomen contained about 8 to 10 oz. of cloudy fluid. The liver had the typical appearance of multilobular cirrhosis. On raising the liver I discovered the cause of the cloudiness of the fluid in the abdomen, for a perforated ulcer of the pylorus on the greater curvature was exuding partially digested milk, and this, mingling with the straw-coloured ascitic fluid, produced the turbid appearance. The small and large intestines were full of blood. The spleen was normal in size. The liver weighed 22 oz. The pathological report on the liver was as follows: "Sections show considerable cirrhosis; this is to some extent of multilobular type, but numerous delicate fibrous bands are seen surrounding most of the individual lobules, and conforming more to a biliary (Hanot's) cirrhosis, though there is no evidence of active bile-duct regeneration."

My thanks are due to Dr. Bruce for his account of the history of this case, and to Dr. Douglas Thornton for his report on sections of the liver, as also to his staff for pathological investigation.

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