No part of the bunion itself is removed. When the patient is returned to the ward, the end of the bed is raised on 6-inch blocks, and a cradle placed over the operated foot.

AFTER-TREATMENT

This is of great importance. On the second day, strips of adhesive plaster, half an inch wide, are applied to the dorsal and plantar aspects of the big toe, and their long ends are carried up and fixed to the cross-bar of the cradle to act as an extension to pull the toe straight and make certain that there will be an appreciable space between the base of the phalanx and the cut surface of the metatarsal shaft. It is found that the patient is quite able to regulate the amount of this extension suitably by withdrawing the feet slightly up into the bed. At night the strips are undone from the cross-bar and fixed again in the morning. After four or five days the patient is shown how to pull on these strips with his hands many times in the twenty-four hours, and to assist in obtaining a straight but movable new joint. The attachment to the cradle is discontinued. After ten days the stitches are removed, but not the strips; and gentle massage to the scar is begun. In fourteen days the patient is allowed to walk in soft bedroom slippers; the strips are dispensed with altogether, and exercises are started. These consist of frequently repeated flexion and extension movements of the toes, at certain stated times every day.

In a severe and long-standing case the four outer toes have all become deflected too; these must be made to lie in their normal position again. This can be done by active and passive movements, by pulling and stretching, and by inserting small pads of gauze between the second, third, fourth, and fifth toes. Then the pressure of the slipper on the fifth toe is enough to keep all these toes straight, the malposition of the big toe having been corrected.

After three weeks the patient must be fitted with walking shoes to fit the new shape of the feet, with stout soles, ample width in the tread, and straight on the inside. Patients find a flat heel less comfortable than a moderate one, especially if accustomed to wearing a high heel. In six weeks the patient can return to work, provided it does not consist in standing all day: if it does, or if both feet have been operated on, the period of convalescence must be longer.

Massage and exercises are continued throughout the whole of this period, and at night the small pads are placed between the toes. If tenderness is complained of at first when walking, it can be relieved by binding the front part of the foot firmly with one or two encircling strips of adhesive plaster.

REMARKS

The bunion—which I take to mean all the soft parts which form over the prominent bone, consisting of a bursa, fibrous tissue, and thickened skin—is left completely alone, except for the necessary incision at the operation. It has acted as a protection for years, and had better continue to do so. What is not wanted will gradually be absorbed. If it is cut away, there is a danger of the skin becoming adherent to the bone and causing a painful scar—the last thing desired.

No artery forceps are applied. If the operation is performed quickly, say five minutes from start to finish, the periosteal detachers stop all bleeding when in position, and no ligatures are required.

The bone is cut cleanly through, subperiosteally; this means that no bone dust or splinters are left in the wound, and that a cuff of periosteum remains to cover the cut end of the shaft.

Occasionally the extensor tendons of one or more of the toes may be found contracted, and are lengthened by subcutaneous tenotomy, but this is not a routine measure. If extension is not applied, the big toe shrinks back into the foot too much (the operation always shortens the foot slightly) and may become stiff from fibrous ankylosis between the phalanx and the metatarsal shaft. Surgeons have tried to prevent this by removing some of the shaft as well as the head; but this is weakening the foot unnecessarily.

CONCLUSIONS

The important points are:

1. Cut the bone correctly.
2. Leave the bunion.
3. Apply extension.
4. Give exercises and massage.
5. Order shoes that fit the newly constructed foot.

AMOEBIC HEPATITIS WITH HAEMATEMESIS

BY

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The following account of a case of amoebic hepatitis with severe haematemesis is worthy of record, as I have been unable to find a similar case in the literature.

A nurse, aged 40, who served for two years in Mesopotamia during the war, came under my care in 1925. The only sickness recorded during her war service was as follows: appendicectomy in August, 1916; pain in the lower part of the abdomen in January, 1918; sand-fly fever in August of the same year; anaemia and debility with abdominal pain and vomiting in September, 1919. She was invalided to England. Laparotomy was performed in April, 1920. The gall-bladder was excised and thirty-nine stones were removed. The right ovary was cystic. In August, 1920, she complained of pain in the epigastrium. Amoebic hepatitis was diagnosed in November, 1923. *Entamoeba histolytica* was found in the stools. The liver was two finger-breadths below the costal margin.

After the last diagnosis she received a disability pension in 1924, and came under my care in the following year. She had then a definitely enlarged liver, and suffered from chronic constipation. From 1925 to 1929 she remained at work at her hospital and reported to me once a month for treatment. During this time she only had an occasional "flare-up," which rapidly subsided under appropriate treatment. While under my care no *E. histolytica* cysts were found in the stools in the course of numerous examinations. Towards the end of 1929, as her health had been much the same for several years, her disability pension was made final, and I did not see her again until quite recently. In January, 1930, after having more or less acute pain in the liver for some days, she had a severe haematemesis, vomiting about two pints of blood, and melaena. She was still more or less constipated, and the stools were very pale in colour. As her condition had worsened since her pension was made final, her case was reopened for further adjudication, and I saw her again. The question had arisen whether the haematemesis could be regarded as a sequel of her invaliding disability.

On examination in October, 1930, I found that she had so far recovered that her condition was much the same as it was in 1929. There was no jaundice or apparent anaemia, and no enlargement or increase in liver dullness. The heart and lungs were healthy. There was some tenderness in the epigastrium on light palpation, but no mass could be felt. The ascending colon was palpable and tender. The stool examination was negative. A complete radiological examination was made of the whole intestinal tract, and the report was as follows:

"Considerable elongation of stomach. Stomach is hypotonic. Intermittent hyperperistalsis. No evidence of gastric ulcer. Duodenal cap showed persistent deformity; the second part is pulled over to right. Deformity more suggestive of adhesions than that due to an ulcer. Marked ptosis of

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flexures of colon, but apart from this no definite evidence of organic disease."

The blood count showed: red blood cells 4,500,000 per c.mm., white blood cells 8,000; polymorphonuclears 74 per cent., large mononuclears 4 per cent., lymphocytes 20 per cent., eosinophils 2 per cent.; haemoglobin 90 per cent.; colour index 1.1. No abnormal cells were seen.

COMMENTARY

The interesting point about this case is that the clinical, radiological, and microscopical examinations appear to exclude all the commoner conditions giving rise to a haematemesis of such severity. The patient stated that she had never suffered from any gastric symptoms in her life, nor did the radiological examination show any evidence of gastric or duodenal ulcer, nor had she been treated for either at any time. There is certainly a possibility that the deformity of the duodenal cap was due to an ulcer as well as to adhesions. The past clinical history undoubtedly points to the latter cause. At the same time it seems incredible, if an ulcer did exist, that it would give rise to no clinical symptoms save a sudden and severe haematemesis, which did not recur and for which no special treatment was given.

There had never been any evidence of cirrhotic changes in the liver. Thrombosis of the portal vein can be eliminated, as the other usual sequels—namely, enlargement of the spleen and ascites—were entirely absent, and the haemorrhage had not recurred. The normal blood count, together with a normal spleen, excluded early splenic anaemia and leukaemia. What, then, was the cause of the haematemesis? We appear to be left with a gastrostaxis occurring by diapedesis. Might this not be the result of a very sluggish liver of long duration giving rise to a more or less constant hyperaemia of the gastric mucosa? However sceptical one may be in regarding the haematemesis as a sequel of the amoebic hepatitis, it is difficult to exclude the contingency occurring in such an obstinate and long-standing case.

It must be borne in mind that the patient is a nurse on duty in a fair-sized hospital, where she has always had constant and immediate medical attention from a highly competent staff of medical practitioners, so that any gastric disturbances would have been promptly treated.

I have to thank the Director-General of Medical Services, Ministry of Pensions, for permission to record this case.

CONGENITAL ANEURYSM OF THE BRAIN

BY

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The problems of differential diagnosis are nowhere more strikingly illustrated than in aneurysms of the base of the brain. It is now well recognized that besides those instances in which the condition is associated with infective endocarditis or cerebral arterio-sclerosis, there is a group of cases in which the aneurysm is congenital, or depends on a congenital weakness in the arterial wall at certain points. To this congenital weakness of the arterial wall the case reported owes its interest.

CASE NOTES

History.—The patient, a married woman aged 33, was quite well until a month ago, when she had a slight attack of vomiting and complained of a vague headache. Since then she had not felt really well. On September 8th she fainted twice and vomited without warning. During the following week she complained of low occipital headache and pain in the back, and felt "feverish." On September 16th she walked to her doctor's house at 8 p.m. While in the surgery she suddenly collapsed and became comatose. The doctor applied artificial respiration and made arrangements for her admission to hospital.

Condition on Admission.—The patient was deeply comatose. The face and body were pale and cold. The muscles were flaccid and all reflexes absent. The pupils were widely dilated. Examination of the discs showed nothing abnormal. The systolic blood pressure was 110, and the heart rate 70, irregular, but not fibrillating. The urine contained no sugar, but a trace of albumin. No incontinence. Lumbar puncture obtained blood.

Progress.—Artificial respiration was maintained until 2.30 a.m., when intratracheal air was given. Fibrillation was noted at 3 a.m. Death occurred at 3.30 a.m.

Necropsy.—A complete post-mortem examination was made with the following findings. The convolutions of the cerebrum were flattened, and there was a large amount of blood clot in the pia-arachnoid at the base of the brain. The haemorrhage extended along the sulci over the right side of the cerebrum. The fourth ventricle contained blood clot, the lateral ventricles blood-stained cerebro-spinal fluid. Over the junction of the right anterior cerebral and anterior communicating artery was a pale fibrinous clot, about the size of a pea, evidently the remains of an older haemorrhage. The two frontal lobes were adherent to each other in this area. On lifting off this clot there was found a saccular aneurysm measuring 1 by 0.5 by 0.5 cm. occupying the space between the two anterior cerebrals and springing from the junction of the above arteries. The aneurysm showed a white fibrous-looking wall, thinned on the lower side, where it was ruptured. The other blood vessels of the brain showed no abnormality. The pericardium was adherent to the posterior aspect of the left ventricle by some old cord-like adhesions over an area of 2 cm. square. No other abnormality was found in the body. Microscopical examination of the liver and kidneys revealed no sign of disease. The Wassermann reaction of the blood obtained at the post-mortem examination was negative.

COMMENTARY

On reviewing the case in the light of the necropsy, it is evident that the attack on September 8th was due to a leakage from the aneurysm causing a small subarachnoid haemorrhage, which became arrested before causing material damage to the brain or cranial nerves. The subsequent attack was due to a sudden large haemorrhage causing a rapid and fatal compression of the medulla. No evidence of the presence of an aneurysm was detectable before rupture.

The clinical symptoms of a congenital aneurysm may be conveniently divided into symptoms prior to rupture and those following rupture.

Before rupture, localizing symptoms causing pressure on neighbouring structures may be recognized. In the region of the anterior half of the circle of Willis the second, third, fourth, fifth, or sixth nerve may be irritated. Thus a case has been reported where an intractable and apparently idiopathic trigeminal neuralgia was the only symptom. Again, an irregularity of the pupil on one side might be the only tell-tale sign. In the posterior half of the circle of Willis, pressure on the brain stem, in addition to paralyses of the last four cranial nerves, might give rise to paraplegia, cerebellar symptoms, dysarthria, dysphagia, or contralateral hemianaesthesia.

After rupture, leakage of blood into the subarachnoid space at the base of the brain gives rise to clinical signs upon which a diagnosis may be made with reasonable certainty, although meningitis or influenza may be closely simulated. Depending upon the rate of the haemorrhage, the intensity of the symptoms varies. Loss of consciousness passes on to deep coma with a severe haemorrhage, while with a small one, giddiness, headache, and vomiting may be the only

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