This operation corrects the cavus deformity. In four to five weeks the plaster is taken off and the sutures removed.

Stage 2.—At this operation the calcaneus deformity is corrected, the foot being brought down to a right angle. The anterior ligament of the ankle-joint is divided through an open incision across the front of that joint, the tendons being held out of the way. Through a longitudinal incision over the tendo Achillis the tendon is divided and a wedge-shaped piece of bone removed from the back of the astragalus, the base backwards, the superior articular surface of the astragalus being left intact so that we can still get dorsiflexion and plantar flexion at the ankle-joint.

The foot is now brought into an over-corrected position, a sufficient portion of the tendo Achillis being removed so as to meet in this corrected position. The posterior incision is closed. In one of the cases a wide gap was left at the site of the anterior incision, and it was found necessary in order to close it to make a transverse incision above the ankle-joint, undercut its edges, and unite it vertically. Dressings are applied and the foot and leg put in plaster-of-Paris bandages. These are removed after six weeks, and the after-treatment is the same as indicated in cases of talipes equino-varus. Boots and irons are provided with a stop at the ankle to allow plantar flexion and limit dorsiflexion. A varus or valgus T-strap is added if there is any tendency to either varus or valgus respectively.

Talipes Plantaris.

This condition—"hollow foot"—is bilateral, and variously supposed to be due to wearing short boots or to a neuritis affecting the extensor group of muscles of the foot following some of the infectious fevers in childhood. The arch of the foot is higher than normal and the sole hollow, the toes are contracted, hyperextended at the metatarsophalangeal joint and flexed at the interphalangeal joint, the structures in the sole of the foot are shortened. The foot cannot be flexed to or beyond a right angle. Corns develop on the heel and ball of the foot, walking being very painful, this being the chief symptom calling for treatment. This condition can be treated on the same lines as outlined for talipes equino-varus.

Results of Operation.

By observing these cases over a number of years one finds that the paralysed limb, as a result of the operation, not only grows in length and girth, but it grows in length at a quicker rate than the sound limb. I have frequently observed cases where the affected limb has been from 1½ in. to 3 in. shorter than its fellow, that this amount of shortening decreases as time goes on, and in one case operated on four years ago, where the shortening was 1½ in., has to-day become practically nil, the gait is indistinguishable from that of a normal person, and the patient has discarded all apparatus.

Table of some Cases where Measurements have been Recorded.

Talipes equino-varus. Operation, July 18th, 1917. Amount of shortening: November, 1917, 1½ in.; December, 1918, 5/8 in.; to-day, 1/4 in.

Talipes equino-valgus. Operation, 1918. Amount of shortening: October, 1918, 1½ in.; February 25th, 1921, 3/4 in.

Talipes equino-varus. Operation, July 18th, 1919. Amount of shortening: November, 1919, 3 in.; March, 1920, 1½ in.; February, 1921, 1½ in.

Talipes calcaneo-cavus. Operation: first stage, March 19th, 1920; second stage, May 1st, 1920. Amount of shortening: 1920, 1 in.; after operation, June, 1921, 5/8 in.

Talipes equino-varus. Operation, November, 1919. Amount of shortening: June 15th, 1920, 1/2 inch; February 24th, 1921, 1/4 in.

In some cases, where the paralysis is extreme, the patient may have to continue to wear the apparatus, but even in these cases the deformity has been corrected, the gait becomes more that of a normal person and less of an eyesore to its parents and friends, and can partake more in the activities of life. A point to be noted in two cases of spastic talipes equino-varus (right-sided hemiplegia) is that the upper limb markedly improved after the foot had been operated on.

In undertaking the treatment of these cases it is necessary to have them under one's own supervision for a few years, also to have at one's command the services of a skilled masseuse, and to have provided correct boots and

irons. Messrs. C. F. Thackray, of Leeds, have provided me with efficient apparatus.

The line drawings from photographs and skiagram appended illustrate a case of talipes equino-varus before and after operation.

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PIGMENTATION OF THE VERMIFORM APPENDIX.

(A Preliminary Note.)

BY

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THE literature on the subject of pigmentation of the vermiform appendix is at present scanty. In fact, a recent search through the various available indices of medical books and periodicals proved negative. No edition of the large monographs on appendicitis is of late enough date to include this subject.

This interesting pathological condition was first discovered a few years ago by W. H. Battle,¹ and the results of a study of six of his specimens were published by Professor S. G. Shattock in 1916.² In this article the naked-eye appearances and histological characteristics of the specimens are fully described, and possible explanations of the phenomena discussed. The drawing published by Shattock shows a mottled black pigmentation of the mucosa. The irregularity of the coloration is due to a sprinkling of light areas, corresponding to the situation of the lymphatic nodules. In the specimen removed from the case described in this paper the patchy distribution of the pigment was remarkable, reminiscent of the markings on the back of a snake. On analysis Professor Shattock failed to find any traces of iron in these specimens. He was also able to show that the pigment was not derived from blood. Histologically the pigment lies in the tissue surrounding the glandular crypts and is carried in large endothelial cells, all of which are superficial to the muscularis mucosae.

The subject is at present being actively investigated by Professor Sir Arthur Keith. In a recent demonstration at the Royal College of Surgeons he showed several sections of pigmented appendices. The granules are contained in large endothelial cells lying round the bases of the crypts of Lieberkühn. In sections taken from the colon in chronic cases of intestinal stasis these cells are being attacked by large numbers of phagocytes. The actual pigment seems to be related to melanin, and contains a fatty acid which is in some way allied to adrenaline. The changes that occur in the structure of the colon in chronic intestinal stasis are profound, but one of the most interesting is certainly the pigmentation of the mucosa.

In this connexion McCarrison's³ recent work on deficiency diseases is illuminating. This observer has been able to produce experimentally, by feeding animals on a vitamin-free diet, changes in the bowel similar to those described by Keith. While no mention is made of the appendix, the mucous membrane of the caecum is said to be "usually moderately congested, ecchymotic, and often of a dark slate-grey colour." Clinical evidence is also forthcoming to show that a deficient diet, as experienced in a prisoners of war camp, may produce the same changes in the behaviour of the bowel. Guarini⁴ has studied forty cases of deficiency inanition which developed the condition of chronic intestinal stasis, x-ray examination showing visceroptosis, distension of the gut, and constipation.

These pathological changes in the bowel are associated with an alteration in the adrenaline secretion (McCarrison). Further studies on this subject are being carried out. In the meantime there appears to be sufficient evidence to justify the conclusion that the changes in the bowel associated with chronic intestinal stasis and resulting in pigmentation of the mucosa, interstitial fibrosis, and degeneration of the intrinsic neuro-motor mechanism, may

be due to, or at any rate largely associated with, avitaminosis.

The clinical history of the patient from whom the appendix here illustrated was taken is typical of that of chronic appendicitis and suggestive of intestinal stasis.

A lady, 50 years of age, was sent to me complaining of occasional right iliac pain, nausea, inability to take and digest more than a few carefully chosen articles of food, together with obstinate constipation, varying with bouts of passing loose unformed motions.

The first attack occurred thirty years ago, and for twenty years she had noticed an almost continual grumbling in the right iliac fossa with slowly increasing constipation. In 1907-8 she had to resort entirely to aperients and the motions were always loose. In 1916 chronic appendicitis was diagnosed but no operation was performed. In 1920 there was a definite attack of acute inflammation with raised temperature.

FIG. 1.—Section of appendix mucosa. Pigmented cells grouped round the bases of the glandular crypts, superficial to the muscularis mucosae.

When examined in April, 1921, the patient, a lady of a naturally dark complexion, showed a sallow skin, yellowish sclerotics, and a dirty tongue. The urine was normal and the actions contained bile. On pressure deep tenderness in the right iliac fossa was obtained, and Rovsing's sign was elicited.⁵ It was felt that extensive structural changes probably existed in the colon and short-circuiting was discussed. However, it was finally decided, at the request of the patient, to perform appendicectomy, at any rate as a preliminary measure.

The incision advocated by Jalaguier⁶ was utilized to expose the caecum and adnexa.* Several pericaecal adhesions were divided, the ileum released, and a diseased appendix was removed. The appendix as it lay *in situ* was noted to be of a slaty-grey colour, whereas the large bowel appeared normal in tint. When the appendix was slit open the mottled pigmentation was very striking.

FIG. 2.—Appendix slit open, showing mottled pigmentation.

Six months later the improvement in both the general appearance and symptoms of this patient was remarkable. She presented a clear, fresh complexion and looked years younger. The constipation was no longer her *bête noire*, and she was able to take an ordinary diet, digesting and enjoying articles of food she had been obliged to refuse for years.

SUMMARY.

1. Pigmentation of the vermiform appendix has hitherto been regarded as an uncommon condition. Attention having been drawn to it, surgeons will now probably be surprised at its frequency. The first specimens were discovered by Mr. W. H. Battle and described by Professor S. G. Shattock in 1916.

2. The condition may be regarded as secondary to changes in the big bowel associated with chronic intestinal stasis (Keith).

3. The pigment cells contain melanin and a fatty acid substance probably allied chemically to adrenaline.

4. While the exact significance of the pigmentation is not yet clear, it appears constantly in cases of chronic intestinal stasis along with the degenerative changes in Auerbach's plexus (associated with a defective motor mechanism), diminished activity of the intestinal mucosal cells, and lack of the usual protective mechanism against bacterial infection (McCarrison).

5. It is probable that all these changes are closely related to avitaminosis, since all the pathological conditions already mentioned have been produced experimentally in animals by appropriate diets (McCarrison).

6. From the practical point of view the importance of this conception cannot be over-estimated. Chronic intestinal stasis, chronic appendicitis, and many morbid conditions of the caecum may be prevented by the administra-

* The operation is a variation of Battle's, described in 1895. The skin is incised along the linea semilunaris, the anterior sheath 3/4 inch internal to this and after displacement of the rectus the posterior layers are divided transversely *entre deux arcades nerveux*.

tion of a diet containing sufficient natural foodstuffs which are especially rich in vitamin B.

These notes have been written at the suggestion of Professor Sir Arthur Keith, to whom I am indebted for much valuable information and who kindly prepared the sections described. I wish also to thank Lord Dawson for the opportunity of examining the patient clinically and subsequently obtaining the specimen at operation.

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THROMBOSIS OF THE INFERIOR VENA CAVA:

WITH SPECIAL REFERENCE TO PROGNOSIS.

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THROMBOTIC obstruction of the inferior vena cava is a condition which has long been known in medicine. In Alexander's¹ translation of Morgagni's *Seats and Causes of Disease* there is a very clear description of the condition, derived from a letter from Stancari, and Morgagni also gives a detailed reference to a description by Haller.² It may follow an infection (such as typhoid fever or puerperal sepsis), traumatism, or malignant tumour of the kidneys, suprarenal bodies, or liver. In 1911 Pleasants³ collected records of 314 cases of obstruction of the inferior vena cava; of these, 171 were due primarily to thrombo-phlebitis, usually associated with inflammatory foci elsewhere; in 88 cases the obstruction was due to malignant growths; the cause of the remainder was indefinite.

As regards the all-important point of the eventual prognosis of the condition and the average duration of life in those cases unconnected with malignant disease very little appears to be known, and little or no information is given, with few exceptions, in the reports of cases which have been published. This lack of knowledge was emphasized in a discussion on the subject of thrombosis of the inferior vena cava last year in the Section of Medicine of the Royal Society of Medicine, introduced by Dr. F. Parkes Weber.⁴

In 1903 Parkes Weber examined a man, aged 28½, for life insurance, and he was accepted, with ten years added; on May 8th, 1917, he was still alive and well and about to pay the last premium of the policy. Parkes Weber also quotes Shattock's⁵ case of a medical colleague who developed thrombosis of the inferior vena cava in 1884, and who lived for twenty-five years afterwards, death occurring in 1909 from tonsillitis and septicaemia; during the last six years of his life, however, he had been subject to attacks of thrombo-phlebitis in his enlarged saphenous veins. In 1907 Parkes Weber saw a man, aged 26, whose inferior vena cava was thrombosed seven years previously as a sequel of typhoid fever; the thrombosis was associated with oedema of the legs and varicose ulcers, and he had had three attacks of thrombosis. In 1912 the condition of this man was much the same.

I have recently had under my care a patient who developed thrombosis of the inferior vena cava fifteen years ago, from puerperal sepsis, and whose condition is of some interest in regard to the ultimate prognosis of such cases. The patient was sent to me by Dr. W. J. Rutherford, to whom I am also indebted for the historical notes upon this rare condition.

A married woman, aged 41, came under treatment because of very severe thrombosed and ulcerated internal haemorrhoids. Her history was that fifteen years ago she had a very difficult childbirth, involving two days' labour and an instrumental delivery; following this, puerperal sepsis supervened, with white swelling of the left leg and, a week or two later, of the right.

She was completely bedridden for three months, and partially so for nine months. Before her confinement she had been quite healthy, with no sign of varicose veins or haemorrhoids. The swelling and oedema of the lower extremities subsided very slowly. She stated that the superficial veins in the front of the abdomen became greatly distended to the level of her