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.

BY

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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

waist, and she developed piles. In the intervening years she was a semi-invalid, getting about very little owing to the discomfort of the haemorrhoids and the feeling of weight in her legs. She also suffered from dyspareunia and menorrhagia, but seemed to have had no further phlebitis.

When she came under the observation of Dr. W. J. Rutherford, about ten months ago, she had masses of varicose veins in both legs extending up to the thighs, varicose veins in the vulva, haemorrhoids of a severe degree, and a well marked communication on each side between the tortuous veins behind the anterior superior spine and those of the lower intercostal spaces. There was no visible communication between the epigastric systems, but this might well be hidden by the anterior abdominal fat; there was no sign of ascites, and no albuminuria. The haemorrhoids were operated upon in the Manchester Jewish Hospital, with a marked increase of the patient's comfort.

As regards the duration of life after thrombosis of the vena cava, it is interesting to look at the results of a series of experiments on dogs conducted by Béjan and Cohn.⁶ In the dog the inferior vena cava system is absolutely comparable with that of man, and ligature of that vein was performed by these investigators at different points. They reached the following conclusions: In ligature of the inferior vena cava below the level of the renal veins, whatever the points of application of the ligature, life is possible and compatible with ordinary good health. Ligature at the level of the renal veins, and comprising one of these veins in the ligature, is well tolerated. Ligature performed immediately above the renal veins, and taking in the left suprarenal body and its vessels, is also compatible with life, the circulation becoming re established; but ligature performed above the right suprarenal body and its vessels is always followed by death, the circulation apparently remaining completely obstructed.

This conclusion, Béjan and Cohn state, is confirmed by clinical observation, and is also in accord with the conclusions of all other investigators down to that time (1911). Purpura stated (1898-99) that life was possible when ligature of the inferior vena cava was performed between the junction of the iliac veins and the hepatic veins, but was more difficult to preserve if the ligature was made above the level of the renal veins; Gosset and Lecène concluded from their experiments (1904) that ligature of the inferior vena cava below the renal veins was accompanied by no pathological phenomena, not even by any oedema of the lower limbs, but ligature above the level of the renal veins was always followed by serious lesions and death. Leotta⁷ carried out similar experiments in 1907, and came to similar conclusions. The absence of albuminuria in the case described above is of importance here as being strongly suggestive that the thrombosis had stopped short of the level of the renal veins.

Shattock conducted an autopsy in the case which has been already mentioned, and found that the inferior vena cava had been converted into an impervious ribbon from the point of entry of the hepatic vein downwards, while both the renal veins were closed at their entrance into the vena cava, blood being returned from the kidneys through the veins of the capsule and by way of the lumbar veins through the azygos veins. This should be compared with the observation recorded by Morgagni.¹

It seems evident, therefore, that complete obstruction of the inferior vena cava, leads to the development of varicose veins, sometimes of very considerable extent, and to attacks in them of thrombo-phlebitis—in addition to pigmentation of the legs, varicose ulcers, and other sequelae of severe varicosity—and to other symptoms of backward pressure in the venous system of the lower part of the trunk and the legs, but is not incompatible with a prolonged and fairly comfortable life.

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DURING the last nineteen months there have been 34 cases of anthrax in New York, 20 of which have been due to the use of infected Japanese shaving brushes; 11 of the cases were fatal. A few cases were also caused by infected toothbrushes.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

COMMON CHANGES IN ERYTHROCYTES.

It is well known that in lead poisoning the red discs show abnormalities ranging from polychromasia and punctate basophilia to erythroblasts of various kinds. But the changes that follow small doses of lead in a few days have almost escaped observation. In treating occasionally internal haemorrhage and chronic diarrhoea with lead (plumbi acetatis gr. xx, ac. acetic dil. 3j, aq. (dest.) chloroformi ad 3viij; 3ss ter die), I have observed that for ten persons there were, in three to nine days, (1) about four showing polychromasia and punctate basophilia; (2) three showing in addition Cabot rings and Howell-Jolly bodies; (3) two showing all these and erythroblasts; (4) one showing no reaction.

I am not aware that attention has been directed to the frequency with which such changes follow the malarial paroxysm. My experience is limited to a few cases of benign tertian infection, but the changes were practically always present about thirty hours after the onset of pyrexia. In ordinary ambulatory cases nucleated reds are seldom seen, but in such cases polychromasia may be detected in 3 to 4 per cent. of erythrocytes, and punctate basophilia, Cabot rings, and Jolly bodies in about 1 per 1,000 of red discs. These changes are not usually observed in ordinary ailments, but I have seen them, though rarely, in influenza, typhoid fever, and scarlet fever. In each of three cases of haemolytic anaemia they were greatly increased at the paroxysms (acholuric jaundice), but never disappeared entirely. In pernicious anaemia their presence has been recognized for a long time.

The evidence suggests that these—polychromasia, punctate basophilia, Cabot rings, Jolly bodies—are signs of an erythrocytogenesis more active than is ever required in health, and therefore in this sense abnormal. In malaria such erythrocytogenesis is to be expected and is a good sign. In lead poisoning Pappenheim considered that the metal directly excited the marrow. The fact that even small doses quickly stimulate erythrocytogenesis suggested to me the use of lead in certain persistent anaemias of chlorotic character. In chlorosis one seldom meets with signs of overactive erythrocytogenesis (polychromasia, etc.), but young chlorotics promptly respond to lead. In older women with chronic or recurrent anaemia of chlorotic character I have given lead for a fortnight without observing any reaction in the film except that the reds were less anaemic, and that the increase in haemoglobin continued for weeks after stopping the drug.

In any case it is useful to recognize these changes. Ordinary stains show the coarse basophilia excited by lead, but the other changes and the finer basophilia in malaria are rarely displayed by a stain that cannot be depended on to show Schuffner's dots conspicuously. Manson's methylene blue answers well when it has been steamed (not under pressure) for half an hour on three or four successive days. It becomes intensely polychrome. It is convenient to have two small wide-mouthed bottles with glass stoppers. One contains about 20 c.cm. of pure methyl alcohol, and films are dropped in to be fixed by laying the bottle on its side for five to fifteen minutes. Into the other bottle 50 c.cm. of distilled water are poured, and four drops of 1 per cent. aqueous solution of eosin and three drops of polychrome Manson's blue added. The whole is well mixed, and the fixed film transferred with forceps to stain for half an hour to two hours or longer. Distilled water, drop tube, and clean bottles are necessary.

Ealing, W.

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A CASE OF ANEURYSM OF THE SUPERFICIAL PALMAR ARCH.

The following case of traumatic aneurysm of the superficial palmar arch is, I think, worthy of record.

A girl, aged 5 years, was admitted to the Presidency General Hospital, Calcutta, under my care on October 4th, 1921. She was suffering from a pulsating tumour on the palmar surface of her right hand. The history given was that three weeks before she sustained an injury to her right hand by a piece of glass from a burst soda-water bottle. The injury produced a linear