The term "decerebrate rigidity," though convenient, is apt to suggest that the lesion producing it must be situated about the mid-brain—that the rigidity in man is the outcome of a pathological process which is the counterpart of an actual section at the colliculi in animals. These cases of mine demonstrate clearly that the level is a physiological rather than an anatomical one, and I have refrained from applying the term "decerebrate" to the rigidities in order that this significance should not be obscured.

Why generalized rigidities should make their appearance in one case and not in another of very similar gross pathology is a delicate question. It is probable that the syndrome in man depends on a very fine circulatory balance, on a relative anoxaemia sufficient to suppress cortical control but not great enough to render inactive the causal mechanism in mid- and hind-brains. Generalized rigidity may thus never have an opportunity of arising in one case; it may be but a phase in another (as Kinnier Wilson points out); whilst in a third, where the balance is maintained for a period, the rigidities may border on chronicity.

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METATARSUS VARUS.

BY

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METATARSUS VARUS is a deformity which has long been familiar clinically to orthopaedic surgeons, but it is not described even in textbooks of orthopaedic surgery, and its essential pathology has not yet, I think, been recognized.

Whitman,¹ it is true, makes passing reference to "metatarsus varus" as an incomplete form of clubfoot (congenital talipes equino-varus) or simple talipes varus, and the treatment recommended is the same as that for the varus part of congenital clubfoot.

But metatarsus varus belongs to quite a different category from clubfoot or talipes varus. It is not a contracture deformity at all. It is an anatomical deficiency deformity, and is due to congenital absence of the internal cuneiform bone. It should therefore be classed not with talipes or other congenital contractures, but with those conditions in which deformity is incidental to gross structural defect—for example, congenital absence or deficiency of the tibia, fibula, radius, ulnar, vertebrae, etc.

Clinically, the posterior part of the foot presents a normal appearance. There is no limitation of movement at the ankle or subastragaloid joints. There may even be some eversion (valgus) of the foot at the subastragaloid joint. The anterior part of the foot is sharply adducted at the tarso-metatarsal joint, but there is no inversion of the sole. The patient, therefore, stands with the sole of the foot flat on the ground, but while the posterior part of the foot is straight, the anterior part is directed inwards or forwards and inwards. The great toe is usually adducted more than the other toes (hallux varus). There is a sharp kink, which can be both seen and felt, in the inner border of the foot, just behind the base of the first metatarsal bone. The patient can walk, but his gait is awkward, and the deformity is unsightly. So far as I know, the condition is always bilateral.

It should be noted that the displacement of the anterior part of the foot is entirely abnormal—it is not even, as in talipes, an exaggeration of a normal displacement. Adduction without inversion is not a physiological movement of the foot.

Radiography shows the adduction of the anterior part of the foot, but it does not reveal the essential defect in young children, because the cuneiform bones are not ossified at the age at which these cases usually come under observation. I have not had the opportunity of examining a patient who has grown up with this deformity.

In six feet (of three patients) that I have examined at operation the internal cuneiform bone was absent and represented by a flat disc of fibro-cartilage between the base of the first metatarsal bone and the scaphoid. The tendon of the tibialis anticus divided into two distinct slips, one of which was inserted into the fibro-cartilage, and the other into the base of the first metatarsal. It was not possible at operation to make out the condition of the middle cuneiform bone. The first metatarsal was shorter than normal and strongly adducted. The scaphoid was normal. In one case the neck of the astragalus appeared to be unduly long, and the head was bent downwards.

Treatment.

From the nature of the deformity it is obvious that the treatment suitable for talipes varus or the varus part of talipes equino-varus is quite inappropriate for metatarsus varus, and the general experience of orthopaedic surgeons is that the results of such treatment are unsatisfactory. It is true that a considerable amount of apparent correction can be brought about by manipulation and fixing the foot in plaster. But the correction thus obtained is not permanent, and the deformity always relapses more or less completely.

Anatomically the inner border of the foot is too short, owing to absence of the internal cuneiform bone, while the outer border is relatively too long. Permanent correction, therefore, can only be obtained either by lengthening the inner border or by shortening the outer. Two procedures, therefore, suggest themselves: (1) Separation of the first metatarsal and scaphoid bones, and the insertion of a graft to keep them apart; and (2) excision of bone—for example, the cuboid—from the outer border of the foot.

I have tried both these operations with, in each case, immediate correction of the deformity, but they are too recent to speak yet of the ultimate results. Theoretically, to replace the missing bone by a graft, and thus to restore the inner border of the foot to its normal length, would appear to be the ideal method of correcting the deformity. But I doubt very much whether this can be attained in practice. In the first place, the results of bone grafting, excellent as they are in very many conditions, are notoriously uncertain in cases of congenital bone deficiency. In the second place, if a fixed graft is successfully inserted into the first metatarsal and the scaphoid, and perhaps also the astragalus, it is likely that the rigid foot produced will eventually prove to be a greater disability than the original deformity.

For these reasons I think that the better practice is to remove bone from the outer side of the foot—a practice, be it noted, that is absolutely contraindicated in cases of clubfoot in young children. By removal of the cuboid the outer border of the foot is shortened and the metatarsus is abducted and brought into line with the posterior part of the foot. This, so far as my observations go, gives the best correction of the deformity with the least disturbance of function.

REFERENCE.

¹ Whitman, *Orthopaedic Surgery*, 1919.

TWO CASES OF PERNICIOUS ANAEMIA
WITH INFECTIVE FOCI IN THE
ALIMENTARY TRACT.

BY

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MUCH has been written on the etiology of pernicious anaemia, and the clinical evidence points to there being an invasion of some part of the alimentary tract by pathogenic organisms in a large percentage of cases. In the two following cases there appears to be very definite evidence of either past or present pathological changes in the alimentary tract.

CASE I.

The first case was that of A. B., aged 38, a trained nurse, who came under our care on April 8th, 1921. The history of the illness was that in July, 1920, she felt very "run down," and had to give up nursing. About a month later she noticed that

her skin and mucous membranes were pale. Amenorrhoea lasted for five months during the summer of 1920. No treatment beyond rest was adopted at this time.

In March, 1921, she felt sufficiently well to begin nursing again, but became ill again after three weeks' work. There was nothing of any importance in the family history. She had an attack of colitis nine years previously. Most of her teeth had been extracted, but she had a good artificial set with a few sound teeth of her own. No glossitis was present. She was very weak and pale; she had no other abnormal signs or symptoms, but the temperature was 100.2°.

On April 12th the result of a blood count was: Erythrocytes 1,232,000; leucocytes 1,750; haemoglobin 20 per cent.; polymorphs 53 per cent., eosinophils 1 per cent., lymphocytes 45 per cent.; colour index 0.85.

Marked poikilocytosis and anisocytosis were present, with a preponderance of microcytes. Moderate polychromatophilia and some basic stippling were observed. Two nucleated reds, both megaloblasts, were seen while counting two leucocyte films.

She was given novarsenobillon, 0.2 gram, intravenously, and the oral administration of beta naphthol, 5 grains twice daily, was commenced. Up to April 13th she had regular normal actions of the bowels, but after that date they were never open again except by enemata. The temperature rose, with morning remissions, until it was 104.4° on April 14th, falling by lysis until it reached 96.6° on April 21st. On April 14th cultures were sown from a catheter specimen of urine and a specimen of faeces. The former showed the *Staphylococcus albus* only, the latter *B. coli* only. On April 15th the patient became delirious and lay in a semi-conscious condition. On April 19th novarsenobillon, 0.1 gram, was given by intramuscular injection. On April 22nd her temperature was still subnormal, so half a pint of citrated blood from healthy and compatible donors was given intravenously. She recovered consciousness for about an hour, but her temperature again rose, and she died on April 25th, 1921.

The autopsy showed the following pathological changes. "Tabby-cat striation" was present on the columnae carnae of the heart. The liver showed fatty change, and excess of iron was present. The kidneys showed fatty degeneration, their surfaces were finely granular, and the capsules adherent.

The appendix was empty, with an apparently congenital occlusion of the lumen at its proximal end. About five feet above the ileo-caecal valve there was a sharp line of demarcation in the interior of the small intestine, but no constriction was present. Above this line the appearance was that of normal jejunum, below it normal ileum. The appearance at this place was as if an end-to-end anastomosis had been performed, but the patient had never undergone an abdominal operation. The line itself appeared to consist of a fine ring of small ulcers. Microscopic sections showed a simple ulceration of the mucous layer, the other layers being normal.

A section of the spleen showed slight fibrous change, with atrophy of the Malpighian tufts. Films of bone marrow showed some hyperplasia of the erythroblastic elements.

CASE II.

The second case was that of C. D., aged 58, a coachman. He came under our care on January 15th, 1921, with a history of diarrhoea and vomiting during the past seven weeks, accompanied by slight jaundice during the earlier part of that period. His past history was that he had two attacks of influenza about fifteen years previously, a severe attack of diarrhoea four years previously, followed by occasional milder attacks.

His appearance was cachectic and his mucous membranes were very pale. His teeth were dirty, but seemed sound otherwise. Emphysema of the lungs almost obliterated the cardiac dullness. Some scabbing was present in the right nostril, and a small polypus was found at the anterior end of the middle turbinal bone. This gave rise to a clear mucous discharge which was sterile. A blood count was taken on January 17th, with the following result: Erythrocytes 1,240,000; leucocytes 4,400; haemoglobin 35 per cent.; polymorphs 20 per cent., eosinophils 0, lymphocytes 80 per cent.; colour index 1.4. Marked poikilocytosis, extreme anisocytosis, and an abundance of macrocytes were observed. Seventeen nucleated reds, including five megaloblasts, were seen. Most of the reds showed nuclear remains. The patient was given haemoglobin and malt, 1 drachm thrice daily, also beta naphthol 5 grains twice daily. He started weekly intravenous doses of novarsenobillon 0.2 gram. The following pathological tests were made: Wassermann, negative; faecal examination, excess of fatty crystals; faecal culture, coliform bacilli and Gram-positive diplococci; urinary culture, a few sarcinae.

A blood count on February 3rd showed some improvement: Erythrocytes 1,528,000; haemoglobin 46 per cent. On February 16th the count was worse than the original one. This was attributed to a somewhat severe reaction after each dose of novarsenobillon. The dose was reduced to 0.1 gram, which produced no untoward symptoms, and has been gradually increased to 0.2 gram, which is being given at the present time. By April 8th he was again improving: erythrocytes 1,776,000. On April 4th an x-ray examination showed that the fangs of the lower lateral incisors were rather bulbous. These were extracted under a general anaesthetic with special antiseptic precautions, and yielded a pure growth of streptococci. A course of autogenous vaccine was given until June 13th, followed by a course of stock streptococcal vaccine. On June 28th the count was as follows: Erythrocytes 3,216,000; leucocytes 5,600; haemoglobin 65 per cent.; polymorphs 22 per cent.,

eosinophils 2 per cent., lymphocytes 76 per cent.; colour index 1.0. Fairly marked poikilocytosis and polychromatophilia. Six normoblasts, no megaloblasts.

The patient now enjoys good health, having the novarsenobillon and vaccine weekly. Time alone will show whether this is genuine recovery or a remission.

The points which we wish to emphasize about these two cases are the history of colitis in the first case and of diarrhoea in the second. In the first case a definite ring of ulcers was found in the intestine, and in the second an infective focus in the teeth. Novarsenobillon seems to be a valuable drug in this disease; the value of the autogenous vaccine cannot be estimated as it was given concurrently with other treatment. Evidence of alimentary derangement was also present in a fatal case of pernicious anaemia following dysentery which we treated in 1920. There was a history of alimentary derangement in a case of aplastic anaemia and a case of pernicious anaemia which Dr. Rivaz Hunt has kindly allowed us to quote. The latter appears to be enjoying normal health under treatment with novarsenobillon.

EIGHTY-NINTH ANNUAL MEETING

OF THE

British Medical Association.

Held at Newcastle-on-Tyne, July, 1921.

SECTION OF PHYSIOLOGY, PHARMACOLOGY, THERAPEUTICS, AND DIETETICS.

H. H. DALE, C.B.E., M.D., F.R.S., President.

BEFORE proceeding to the business of the Section, Dr. H. H. DALE and Professor A. V. HILL gave two eloquent appreciations of the work and character, both as a physiologist and as a man, of the late Professor J. A. Menzies, Vice-President of the Section, who died only a few days before the opening of the Annual Meeting of the Association. It was decided that a resolution expressing the "deep sense of the loss medical science has sustained by the death of Professor James Acworth Menzies, Professor of Physiology in this University, and Vice-President of this Section," should be conveyed to the University, and that in addition a message of respectful sympathy should be sent to Professor Menzies's widow.

THE USE OF THE HOT WIRE FOR INVESTIGATING THE TIME RELATIONS OF THE PULSE AND THE CHARACTERISTICS OF VOLUNTARY CONTRACTION IN MAN.

BY

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[Abstract of Communication.]

A VERY fine platinum wire (of diameter about one-hundredth of a millimetre) is mounted in a tube which is connected to a tambour or funnel placed upon the subject's artery. The wire is heated to just below redness by an electric current. The rise of temperature in the wire raises its electrical resistance, which, however, is lowered each time a pulse of air comes along the tube and cools the wire. The pulses of air caused by the movements of the artery, therefore, cause corresponding changes in the resistance of the wire, and a continuous record of the latter is therefore also a continuous record of the movements of the artery. The electrical resistance of the wire may be recorded in one of three ways:

1. By a Wheatstone's bridge arrangement and a string galvanometer. The hot wire is placed in the fourth arm of the bridge and the other resistances are so adjusted that no current passes through the string galvanometer, and therefore the string is not deflected. When the resistance of the hot wire changes, the balance is upset and