distribution of blood pigment in pernicious anæmia, cannot be produced experimentally otherwise than by special agents, and especially not by any simple exaggerated blood destruction such as follows transfusion; beyond these points I do not consider that my results go. Therefore in deducing from the foregoing facts that the blood destruction in pernicious anæmia is probably due to the action of some special poison; and inasmuch as it occurs in the portal area, that this poison reaches the blood from the gastro-intestinal canal, one goes further than the facts appear to justify. If these deductions be taken alone apart from their context—the context in this case being the pathological and physiological data above described—they are naturally open to such a criticism.

I do not consider, however, that it is right to consider these deductions apart from their context. The whole view as to pernicious anæmia being a special form of anæmia is based on facts which establish that an excessive blood destruction, initiated within the portal area, is the characteristic feature of this disease; and that this destruction differs from that in health, from other forms of excessive blood destruction, and notably from anything met with in the anæmia of wasting and malignant disease where no increased destruction at all occurs.

DIFFERENCE BETWEEN PERNICIOUS ANÆMIA AND CHLOROSIS: REGARDED BOTH AS VARIETIES OF INTESTINAL ANÆMIA.

I think that the above point is one of some importance in considering the true relationship of pernicious anæmia to other forms of anæmia. For if attention be confined exclusively to the final deduction that pernicious anæmia is the result of the absorption of poisons from the intestinal tract, the disease might erroneously be grouped in the same category as chlorosis and other forms of what might for convenience be termed "intestinal" anæmias, which, if we accept Sir Andrew Clark's view, are also the result of absorption of poisonous substances from the intestine. Both varieties of anæmia—pernicious anæmia and chlorosis or the anæmia of adolescence associated with constipation—would be referred to the absorption of poisons from the intestinal canal.

And yet no greater contrast could exist between two diseases than, in my opinion, exists between pernicious anæmia and chlorosis—chlorosis being the result of defective blood formation (that is, hæmogenic), pernicious anæmia being the result of excessive blood destruction (that is, hæmolytic).

I express no opinion as to the correctness or otherwise of the above view of chlorosis being an intestinal anæmia due to the absorption of deleterious substances. I would merely point out that the view that chlorosis is an intestinal anæmia is in my opinion a purely clinical view. It is devoid of the slightest pathological basis. The blood change is a fact; the constipation may or may not exist. But even if both co-exist, the relationship between the two is as dark as ever. There is no evidence whatever to connect the deficient formation of hæmoglobin with the absorption of poisonous products from the intestine.

In the case of pernicious anæmia, on the other hand, the matter stands as I think very differently. The facts denoting excessive destruction are, I think, indubitable, and are easily demonstrable, whether regard be had to the blood pigment in liver and kidney, to the increased excretion of urinary pigment increased excretion of iron in the urine, to occasional presence of blood-pigment granules in urine. Hardly less conclusive, in my opinion, are the experimental data which appear to indicate: (1) That this destruction occurs in the portal circulation, especially within the spleen; and (2) that it is not simply an exaggeration of that which occurs in health, but resembles rather that produced by special poisons.

To deduce, then, from these data that the blood destruction in pernicious anæmia has special characters of its own, and, inasmuch as it occurs within the portal area, that the agent or agents that affect the blood injuriously reach it from the intestine—such a conclusion, I venture to think, is not so much a hypothesis—in the sense that the view regarding chlorosis is a hypothesis—as a legitimate interpretation of the data, both pathological and physiological, which we have as yet to hand. So far as the available data go, the position I would venture to take up is that the facts in favour of per-

niciouæmansia being a special variety of anæmia are more numerous and more firmly based than those in favour of it being merely an extreme form of ordinary anæmia.

It still remains to be ascertained what the nature of the special intestinal factor is underlying the disease. I have shown in a former paper that it is not any excess of ordinary putrefactive processes. The alternative appears to be the view I then expressed, that the special factor underlying that form of anæmia termed pernicious, "is the presence, under certain favourable conditions, of organisms of specific nature within the gastro-intestinal tract."² It is, in short, a specific form of gastro-intestinal infection.

Moreover, the view that there is something special in pernicious anæmia seems to me not only based on physiological and pathological data, but to accord most fully with the clinical facts. For it serves most simply, in my opinion, to account for the comparative rarity of the disease, a fact, as it appears to me, difficult to explain on the view that it is only an extreme condition, the product of many morbid conditions.

Cancer, elsewhere than in the gastro-intestinal tract, never causes the distinctive features of this variety of anæmia. Moreover, it is only exceptionally that cancer, even in that tract, does so.

Moreover, as I pointed out in my first work, and as Dr. Hale White has very fully shown from a careful study of all the *post-mortem* records of Guy's Hospital, it is mostly in connection with the gastro-intestinal tract that lesions and morbid conditions are found, whether malignant disease, or inflammatory affections, or worms, etc.

The rôle of these various conditions in causing the disease is only a favouring one, and not the chief one. If without the intervention of some special factor they could cause this variety of anæmia, then pernicious anæmia would not be the comparatively rare disease it is. Both clinical and pathological facts appear to me, therefore, to point to something special as the occasion of the destruction which marks this form of anæmia, and the gastro-intestinal tract as the seat of origin of the disease.

Dr. GIBSON observed that he had personally received much benefit from the work of Dr. Hunter, more particularly in regard to the question of treatment by intestinal antiseptics. With reference to the pathological side of the disease he would only remark that in the *post-mortem* examinations he had seen there had often been few, if any, hæmorrhages. The last case under his own care, for example, was one in which by means of treatment the patient had temporarily recovered; his hæmocytes had risen from 500,000 or 600,000 to over 5,000,000; and he had been discharged from hospital in a condition which seemed to be one of health. He returned, however, in a few weeks to tell that he had begun to feel unwell, and within a week he died. After death there was not a trace of hæmorrhage, not even in the retina. The speaker's own opinion was strongly in favour of the views of Dr. Hunter.

A CASE OF PULMONARY HYPERTROPHIC OSTEO-ARTHRORPATHY.¹

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THE case which I am bringing before you to-day illustrates a condition at first confounded with acromegaly, but subsequently shown by Marie to be quite distinct. It is of interest from its comparative rarity, as only seven other cases have been described as such in English medical papers, and two of these are doubtful, while a third is an Australian case. Taking the whole literature of the subject, I have been able to discover altogether thirty-one cases which have been recognised by their reporters and which appear to be typical, and twenty-eight more which may belong to the same group but are either disputed, insufficiently reported, otherwise described, or not quite typical.

J. M., umbrella maker, aged 34, came under my care on April 1st, 1895, as an out-patient of the North London Consumption Hospital. His

² *Trans. Med. Soc.*, vol. xiii, 1890.

¹ Read in the Section of Pathology at the Annual Meeting of the British Medical Association, held in London July-August, 1895.

family history presented nothing remarkable. Two of his fingers (right index and left medius) were damaged in early life by accidents; many years ago he thinks he had a touch of rheumatism in the joints of his arms and legs, but was not laid up with it, and had no other definite illness, remaining in good health until early in 1894. He has apparently been temperate, and has not had syphilis. His present illness began insidiously with cough, general weakness, and pain in the back, which finally obliged him to give up work in October, 1894. Up to this time he could use small tools without difficulty, but six or eight weeks before Christmas his hands rapidly swelled and assumed their present condition. He cannot say when his feet began to swell. There were occasionally slight pains in his hands and feet, but no headache of any consequence and no affection of sight nor any noticeable change in his features.

On admission, an ill-nourished, somewhat anæmic man, with large hands and feet, contracted chest, and high dorsal angular curvature; height 5 ft. 5 in.; weight 8 st. 6½ lbs.; upper arms thin with ill-developed muscles; forearms thickened in their lower fourth in both directions, the enlargement beginning suddenly and chiefly or entirely involving the bones, not materially disguising the bony landmarks, without œdema, skin thickening, marked pain, or tenderness; hands enlarged, with thick, flat palms and long, thickened, spindle-shaped fingers; some bony thickening of the carpus, but the main alteration is in the heads

of the metacarpal bones and the fingers; the finger pads are swollen, especially in thumbs and middle fingers; the nails large and convex in both directions, deeply implanted, and raised at their roots, thin, dotted, and longitudinally striated, quick in their growth, like parrot beaks on side view; the skin over the hands is neither thickened, red, nor adherent; it is thin and tightly stretched over the enlarged finger bones; movements of hands clumsy; the fists cannot be completely clenched. Movements in shoulder-joints a trifle limited, with creaking to be felt but no effusion; outer ends of clavicles, acromion processes, and spines of scapulae obviously thickened; the elbow-joint has some synovial thickening, and cannot be completely extended; wrist-joints also a little thickened; proximal interphalangeal joints are enlarged, that of the left medius being hyperextended and ankylosed from old injury; terminal phalanges of some of the fingers hyperextended. In the lower extremities there are similar but less marked changes, somewhat masked by œdema and varicose veins. The enlargement here affects the soft parts to a greater extent, and is associated with a little more tenderness of bones. Calf muscles badly developed; knee-joints present bony enlargement and a little synovial thickening without effusion; there is a little thickening of iliac crests; movements somewhat limited in knees, ankles, and feet; there is probably a little fluid in the ankle-joints; skin of lower limbs nowhere red, adherent, nor thickened.

A small kyphotic projection is seen about the fourth dorsal vertebral spine, apparently due to quiescent caries. The chest is flattened anteriorly, immobile and cylindrical, the third and fifth ribs being thickened. Resonance is altered in quality nearly everywhere, suggesting emphysema with fibrosis. Signs of consolidation and, perhaps, cavity in right apex. Dulness on light percussion at bases laterally without

marked change in breath or voice sounds, or vocal fremitus. Some consolidation posteriorly around the kyphosis, and near left anterior axillary fold. A few bronchial sounds here and there, but no crepitation or friction. Breath short on exertion, respiration 20. There had been occasional night sweats from time to time since autumn, and a little hæmoptysis on one occasion. Cough is troublesome, and keeps him awake at night. Expectoration copious, muco-purulent, not offensive. Heart impulse in fourth and fifth spaces visible but rather feeble; pulse, 120 to 140. Spleen a little large; liver apparently normal; thyroid small; no albuminuria or glycosuria; reflexes of both kinds lively; no ataxy, anæsthesia, or paralysis; no enlargement of tongue, larynx, ears, hips, nose, or genitals.

Progress of the Case.—During observation crepitations have appeared in the patch near left anterior axillary fold, and also above liver on right side, and a month ago some pleural effusion took place, for which he was admitted into the hospital as an in-patient. Towards the end of May there was again slight hæmoptysis, and effusion appeared first in one knee-joint, then in the other. In all other respects he has improved under treatment, which has consisted mainly in cod-liver oil, tonic acid mixtures, a little creasote, and iodine externally.

From the foregoing condensed report it will be seen that the case is one of osteo-arthropathy, not of acromegaly. Apart from the history of antecedent lung trouble, the distribution of the bony changes is characteristic. The lower jaw is unaffected, the bony enlargement affects the lower parts of forearms and the metacarpo-phalangeal more than the carpo-metacarpal part of the hands, while the finger ends and nails are highly characteristic. There is little or no enlargement of soft parts, whether in the limbs or elsewhere, if we except the œdematous and quasi-elephantoid enlargement of the lower limbs, and there are no symptoms pointing to enlargement of the hypophysis cerebri. Mr. Lawford kindly examined his fundi and visual fields with negative results. Marie was of opinion that the kyphosis of acromegaly was upper dorsal, that of osteo-arthropathy lower dorsal or dorso-lumbar; but as in this instance the kyphosis is most likely due to caries, the exception is less important, even if the rule be reliable. The case throws no fresh light on the nature of the disease. Most of the reported cases have followed pleural effusion, especially if purulent; after this the chief causes have been chronic bronchitis and pulmonary tuberculosis; and Thorburn, in an interesting paper published in 1893, was disposed to regard the condition as a tuberculous osteo-arthritis. A review of the published cases, however, shows that we cannot take such a view without special pleading. Several cases appear to have been associated with distinctly non-tuberculous lung troubles, and it is exceedingly improbable that the bone and joint condition is a local tuberculosis, as it presents none of the characteristic features of tuberculous change. Two or three of the published cases seem to have been dependent upon syphilis, and one was cured by antisyphilitic treatment. There is, I think, no doubt that the disease is closely related to the clubbed fingers of chronic heart and lung disease. The majority of cases have been associated with conditions interfering with aëration of the blood. We may, I think, provisionally assume with Marie that the bone and joint changes are caused by a ferment or toxin, but there is evidence to show that, contrary to Marie's original statement, this is not always pulmonary in origin, and it is quite possible that it may be produced by more than one micro-organism, if indeed it be not an auto-intoxication due to perverted action of some part of the human body itself. It is worth while remembering that ill-nourished tissues are always prone to inflammation, and herein may lie in part the explanation of this form of osteo-arthritis. As regards the clumsy name, it would, I think, be an advantage to replace it with that proposed by Arnold—secondary hyperplastic osteitis, or simply to call it hyperplastic osteo-arthritis.

THE twenty-first London course of lectures and demonstrations for sanitary officers and students at the Sanitary Institute will commence on February 17th. The course comprises eighteen lectures and sixteen demonstrations. The lecturers are Professor Wynter Blyth, Dr. Herbert Manley, Dr. Arthur Newsholme, Dr. J. F. J. Sykes, Professor Bostock Hill, Professor W. H. Corfield, Dr. Joseph Priestley, Dr. Alfred Hill, Dr. L. W. D. Mair, Mr. Wallace Peggs, Professor Roger Smith, Mr. J. Wright Clarke, Dr. George Reid, Mr. W. C. Tyndal, Professor Henry Robinson, and Mr. Charles Mason. The fee for the course is £2 2s. Further particulars can be obtained from the Secretary of the Institute.