

### Cardiac Lesions in Reiter's Disease

SIR,—I was very interested to read the article by Drs. G. W. Csonka, J. W. Litchfield, J. K. Oates, and R. R. Willcox (January 28, p. 243) on the involvement of the heart, particularly the aortic valves, in Reiter's disease. May I add to this report? The development of Reiter's disease into a chronic stage with symptoms resembling those of ankylosing spondylitis has been known for some time. In one of the two cases which I have observed myself, an endocarditis developed in the eleventh year of the disease, leading to aortic valvular insufficiency.<sup>1</sup>

The history of the disease was characteristic: 52-year-old man; 1942, mild dysentery, two to three weeks later monoarthritis in the right knee and purulent conjunctivitis; 1943, oligoarthritis of the joints of the knees, feet, and toes; 1944, iritis; 1949, again arthritis of the knees, and backache; 1953, state of heart: systolic murmur, roentgenologically normal cardiac outline, E.C.G. regular.

On examination in 1956: Ankylosing spondylitis with stiffness of the dorsal and lumbar spine and partial stiffness of the cervical spine. X-ray investigation showed bilateral sacro-iliitis and syndesmophytes between the lumbar vertebral bodies. Only slight pain on movement in the knee and toe joints, which were without swelling or stiffness. Heart: systolic apical murmur, and diastolic aortic murmur. E.C.G., left-preponderance, ST lowered, T flattened. Blood-pressure 140/70. No signs of decompensation. In both eyes there was iritis with synechiae; right eye: secondary glaucoma and almost blind. Balanitis circinata and beginning induratio penis plastica. No urethra discharge, no gonococci after prostatic massage. E.S.R. 24/51. Anti-streptolysin titre normal. Streptococcal agglutination test negative.

This case is interesting because (1) the progressive balanitis circinata accords with the persistency of a chronic Reiter's disease; (2) clinically and roentgenologically the spine showed the characteristic signs of classical ankylosing spondylitis; (3) there was severe iritis in both eyes with secondary glaucoma on one side; and finally (4) because in the eleventh year of the disease characteristic insufficiency of the aortic valve was evident. The close relationship between Reiter's disease and ankylosing spondylitis obviously is evident not only in the development of ankylosing spondylitis out of Reiter's disease, in the tendency of both diseases to affect the calcaneus and to produce iritis, but also in the cardiac complication of the aortic insufficiency in some cases.—I am, etc.,

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#### REFERENCE

- <sup>1</sup> Gamp, A., *Münch. med. Wschr.*, 1956, **98**, 334.

### Oral Vitamin B<sub>12</sub>

SIR,—We appreciate the careful studies reported by Dr. N. K. Shinton (June 3, p. 1579) on the behaviour of the serum-vitamin-B<sub>12</sub> levels on oral vitamin-B<sub>12</sub> peptide therapy, as compared with vitamin B<sub>12</sub> alone. We also recognize that the significance—if any—of these levels on this form of therapy is a matter of some importance to all who are interested in the development of a safe and reliable oral method of treating pernicious anaemia. It is essential, therefore, to dissociate speculation from fact, and to avoid conclusions based merely on inference, in the study of this problem.

We agree that, in the untreated case of pernicious anaemia, serum-vitamin-B<sub>12</sub> levels are usually low; furthermore, in patients receiving intrinsic-factor

therapy, a fall in the serum-vitamin-B<sub>12</sub> levels often precedes a relapse. We seriously doubt, however, whether this entitles Dr. Shinton to assume that low serum-levels reflect "in all circumstances" a corresponding deficiency in tissue stores, or conversely—as is implied—that normal levels reflect tissue saturation. However, let us analyse Dr. Shinton's own data derived from the patients receiving crystalline vitamin B<sub>12</sub> by mouth in Table II (p. 1580) in the light of this assumption.

Taking the body reserve of vitamin B<sub>12</sub> in health to be approximately 4,000 µg.,<sup>1</sup> and a mean normal serum level of 400 µg./ml.,<sup>2</sup> then for patients receiving 100 µg. of crystalline vitamin B<sub>12</sub> daily the mean absorption is 33% of the dose taken, for patients receiving 200 µg. daily it is 43%, and for those receiving 500 µg. it is 34%. Thus it would appear that Dr. Shinton's patients have absorbed several times more vitamin B<sub>12</sub> from the gut than does the average normal individual—as estimated either by faecal excretion<sup>3</sup> or hepatic uptake<sup>4</sup> of the radioactive vitamin. As it is well established that the pernicious-anaemia subject absorbs, on average, only 1% of a 100 µg. dose,<sup>5</sup> we are bound to conclude from Dr. Shinton's own experimental data that his basic assumption that serum levels reflect the degree of tissue concentration is not correct. To our minds, a far more likely explanation of the behaviour of the serum levels on crystalline vitamin-B<sub>12</sub> therapy is that the vitamin when given in that form binds itself to the serum to a disproportionately high degree, as compared with the tissues.

Regarding vitamin-B<sub>12</sub>-peptide therapy, the only published evidence of which we are aware, concerning tissue concentration, has clearly shown that in the gastrectomized patient administration of the vitamin as a peptide complex virtually restores its absorption by the liver to normal.<sup>6</sup> In view of this, and as the liver contains about 50%, and the serum less than 0.03%, of the total body content of vitamin B<sub>12</sub>, it is a matter of some surprise to us how much importance is still placed on the serum vitamin-B<sub>12</sub> estimations alone by various observers, on this form of therapy. Indeed, to our minds, the relatively high degree of liver absorption renders the behaviour of serum vitamin-B<sub>12</sub> levels of theoretical importance only, and of no practical significance in the oral treatment of pernicious anaemia with the vitamin-B<sub>12</sub>-peptide complexes.—We are, etc.,

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#### REFERENCES

- <sup>1</sup> Grasbeck, R., Nyberg, W., and Reizenstein, P. G., *Proc. Soc. exp. Biol. (N.Y.)*, 1958, **97**, 780.  
<sup>2</sup> Shinton, N. K., *Brit. med. J.*, 1961, **1**, 1579.  
<sup>3</sup> Mollin, D. L., Baker, S. J., and Doniach, I., *Brit. J. Haemat.*, 1955, **1**, 278.  
<sup>4</sup> Glass, G. B. J., Boyd, L. J., and Stephanson, L., *Proc. Soc. exp. Biol. (N.Y.)*, 1954, **86**, 522.  
<sup>5</sup> Mooney, F. S., and Heathcote, J. G., *Brit. med. J.*, 1961, **1**, 232.  
<sup>6</sup> Milhaud, G., *Nature (Lond.)*, 1961, **189**, 33.

### Tuberculosis and Re-employment

SIR,—Your leading article on tuberculosis and re-employment (April 29, p. 1221) makes depressing reading. It would appear that the approach in civil life to the adequately treated case of pulmonary tuberculosis is completely out of tune with modern knowledge and the enlightened era in which we live. In the Army, on the other hand, tuberculosis is regarded as a disease which may not only be cured but the ex-patient is normally considered fit to return to duty, in the first place in a restricted medical category, should he so elect, but ultimately to regain the medical fitness