herbal and other aperients and enemata, and I suspect they often spend long periods straining in an effort to overcome supposed constipation. Could the suggestion that the squatting posture protects leg veins be tested by observing the prevalence of venous disorders in communities which have adopted low-residue diets but retained the traditional squatting position (such as some affluent Asian communities)?—I am, etc.,

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¹ Cleave, T. L., *On the Causation of Varicose Veins*. Bristol, 1960.

SIR,—With regard to the letter by Mr. R. S. Lawson (9 September, p. 645), may I suggest that all his three points are answered in our joint work?¹

(1) It is known that colonic stasis in many people is centred in the rectum itself (in fact, Hurst introduced the term dyschezia for just this form of constipation), and as a result haemorrhoids are only too likely to occur independently of varicose veins.

(2) It is generally recognized that constipation is considerably commoner in women, and as regards pregnancy (which evolutionarily speaking should never be a factor anyway) it is in the earlier stages that varicose veins and haemorrhoids chiefly become prominent, long before the fetus could exert any pressure on the iliac veins but when constipation is often most pronounced.

(3) That hereditary traits in the veins may decide the distribution of varicosities is not denied for a moment but whether any varicosities occur at all is dependent on something much deeper, which is compatible with the epidemiology set out by Mr. D. P. Burkitt (3 June, p. 556) and is based on the deprivation of dietary fibre.—I am, etc.,

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¹ Cleave, T. L., Campbell, G. D., and Painter, N. S., *Diabetes, Coronary Thrombosis, and the Saccharine Disease*, 2nd edn. Bristol, Wright, 1969.

Ampicillin and Mononucleosis

SIR,—There has been considerable interest in your columns re the question of rashes in infectious mononucleosis. Dr. I. J. Nazareth¹ stated that the great majority of patients suffering from infectious mononucleosis develop a sensitivity rash when given ampicillin. He found that when graduated doses of ampicillin were administered to such patients six months after their illness no rashes developed.

We saw a woman aged 27 suffering from infectious mononucleosis who was admitted to hospital on account of marked purpura requiring fresh blood transfusion and prednisone 60 mg a day, the platelet count being less than 5,000/mm³. No rash developed when a urinary tract infection was treated with ampicillin 2 g daily for 10 days from the day of admission.

A full case report of the haemorrhagic manifestations will appear in the *New Zealand Medical Journal*. Prednisone administration may have suppressed the development of a rash as a reaction to ampicillin in a

patient with infectious mononucleosis.—We are, etc.,

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¹ Nazareth, I. J., *British Medical Journal*, 1971, 3, 48.

Dissecting Aneurysm and Autoimmune Thyroiditis

SIR,—An association between dissecting aneurysm and post-thyroidectomy myxoedema has been described previously¹ but its association with autoimmune thyroiditis has not been recorded in the recent English literature. Therefore we present the following case report.

A 75-year-old woman was admitted to hospital with a three-day history of pain across the scapulae radiating to both arms, which had subsided gradually. There was a 12-month history of increasing tiredness, lethargy, and slowness. Her voice had become deeper and she had noted intolerance of cold, unsteadiness on walking, and paraesthesiae of the fingers and toes. Hypertension (B.P. 210/120 mm Hg) had been recorded 20 years ago during successful treatment for carcinoma of the bladder. No hypotensive therapy was given.

The patient had signs of hypothyroidism—deep voice; pale, dry, cold skin; and delayed supinator and ankle reflexes. The pulse rate was 80/min, regular, and the blood pressure 190/120 mm Hg. There were no signs of congestive heart failure and the peripheral pulses were palpable and symmetrical. Investigations confirmed the diagnosis of myxoedema and tests for thyroid antibodies were positive. Chest x-ray examination showed a dissecting aortic aneurysm and a left pleural effusion. After admission her general condition gradually deteriorated and she died from bronchopneumonia after two months. Postmortem examination showed a dissecting aneurysm of the aorta extending for 20 cm from the left subclavian artery to 10 cm proximal to the coeliac artery. Histologically the aorta showed severe extensive, ulcerative, calcific atheroma of the intima. The dissection had occurred in the outer third of the media and in the descending aorta had extended at one point to the adventitia, the site of leakage into the left pleural cavity. The thyroid gland showed typical features of an autoimmune (Hashimoto's) thyroiditis histologically.

The association of autoimmune thyroiditis with dissecting aneurysm and hypertension in this patient may have been coincidental. Hypertension in association with dissecting aneurysm is well established² and other studies have suggested that hypertension is more common in hypothyroidism than in the general population.³ Kountz and Hempelmann¹ reported four patients who died of dissecting aneurysm in association with post-thyroidectomy myxoedema. At necropsy medio-necrosis of the aorta was found. Its severity was related to the duration of hypothyroidism. On this basis they postulated that hypothyroidism led to advanced degeneration of the blood vessels, but since severe hypertension was present in three of these patients the association of dissecting aneurysm with hypothyroidism was considered by Burchell⁴ to be coincidental.

In our patient the dissection had occurred in a severely atherosclerotic aorta with no medial degeneration—the most common aortic lesion preceding dissecting aneurysm. An association between autoimmune

thyroiditis, hyperlipidaemia, and an increased incidence of atherosclerosis of the coronary arteries has been well documented.^{5,6} We would suggest that a combination of hypertension and severe atherosclerosis exacerbated by autoimmune hypothyroidism led to a dissecting aneurysm in this patient.—We are, etc.,

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- 1 Kountz, W. B., and Hempelmann, L. H., *American Heart Journal*, 1940, 20, 599.
- 2 Hirst, A. E., Johns, V. J., and Kime, S. W., *Medicine*, 1958, 37, 217.
- 3 Fuller, H., Spittell, J. A., McConahey, W. M., and Schirger, A., *Postgraduate Medicine*, 1966, 40, 425.
- 4 Burchell, H. B., *Circulation*, 1955, 12, 1068.
- 5 Bastenie, P. A., Vanhaelst, L., and Neve, P., *Lancet*, 1967, 2, 1221.
- 6 Fowler, P. B. S., Swale, J., and Andrews, H., *Lancet*, 1970, 2, 488.

Lead Poisoning

SIR,—I would like to make some comments on Dr. M. K. Williams's letter (2 September, p. 586) about acceptable blood-lead levels. Since the recommendation in 1968¹ fresh evidence has been presented which indicates that there is no threshold below which lead does not interfere with metabolism. A recent Scandinavian study² has shown that there is a significant negative correlation between blood-lead levels and α -aminolaevulinic acid dehydrase activity in persons with no industrial exposure, indicating that biochemical alterations are being induced by the current levels of lead in the environment. In none of the persons studied was the blood lead greater than 20 μ g/100 ml.

Any recommendation concerning acceptable blood levels of any toxic agent can be made only in the light of the data available at the time. It does not seem unreasonable to suppose that recommendations may subsequently be altered as more information comes to hand. The paper by Dr. A. D. Beattie and his colleagues (27 May, p. 488), to which Dr. Williams refers, is surely just one more indication that the acceptable upper limit of blood-lead levels should be revised and set at a lower limit.—I am etc.,

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- 1 Lanc, R. E. et al., *British Medical Journal*, 1968, 4, 501.
- 2 Hernberg, S., and Nikkanen, J., *Lancet*, 1970, 1, 63.

Atypical Pseudocholinesterase in Leprosy

SIR,—Drs. Molly Thomas and C. K. Job (12 August, p. 390) have drawn attention to a most interesting distribution of atypical plasma cholinesterase among leprosy patients in Karigiri and in villages near Vellore, South India. Their observations are valuable to geneticists and clinicians alike.

They do not comment on the fact that the distributions of apparent homozygotes and heterozygotes do not fit the distributions expected on the basis of the Hardy-Weinberg equilibrium in either the lepromatous or tuberculoid patient groups. Furthermore, reference to data on expected dibucaine numbers (D.N.) in various genotypes¹ shows that the distributions cannot be made to fit, even