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.,

K. M. WADDELL

Kagando Hospital,
Uganda

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.,

T. L. CLEAVE

Fareham, Hants

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.,

T. P. CASEY
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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.,

H. A. WALDRON

Edgbaston,
Birmingham

- 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

Subjects		Dibucaine Numbers			Gene Frequency		χ^2	P
		>70	40-70	<40	E ₁ ^u	E ₁ ^a		
Normal	O	328	14	1	0.9767	0.0233	3.62	>0.1
	E	327.20	15.61	0.19				
Lepromatous .. .	O	65	130	17	0.6132	0.3868	18.17	<0.001
	E	79.72	100.56	31.72				
Tuberculoid .. .	O	76	100	2	0.7079	0.2921	22.87	<0.001
	E	89.20	73.61	15.19				

if assumptions are made that there are abnormally high frequencies of the fluoride or silent genes present in these groups. Assuming that only the usual (E₁^u) and atypical (E₁^a) genes are present, the observed (O) and expected (E) distributions of phenotypes by D.N. number are indicated in my Table.

In both patient groups there appears to be selection of marked degree, apparently in favour of heterozygotes or against homozygotes. The abnormally high frequency of the E₁^a gene suggests that the selection is largely against the atypical homozygotes. On this basis the real frequency of this gene in leprosy patients may be much higher than the data suggest.

One can merely speculate on the nature of the selection involved but analysis of the data in respect of age of the patients and severity of their disease seems warranted.—I am, etc.,

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¹ Pantuck, E. J., in *Advances in Anesthesiology: Muscle Relaxants*, eds. L. C. Mark and E. M. Popper, p. 63. New York, Hoeber, 1967.

Fluorides and Dental Decay

SIR,—With reference to your leading article on fluorides and the prevention of dental decay (19 August, p. 431), while there is some doubt about the long-term effects of compulsory fluoridation of all water and some evidence that it can be harmful in certain cases it would be wrong to put this policy into effect.

As you point out, there are local applications which can be used at individual choice, and it is well known that nothing will prevent dental decay fully so long as children are allowed (and encouraged) to consume vast quantities of white sugar, lollies, and ice creams, especially between meals. Fluoride also destroys catalase. People should be allowed freedom of choice and action and not have compulsory fluoridation of water thrust upon them.—I am, etc.,

E. STEWART

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Surrey

Gastrin and Gastrointestinal Disease

SIR,—In your leading article on gastrin and gastrointestinal disease (9 September, p. 604) you observe that it may well be that hiatus hernia, oesophageal reflux, and reflux oesophagitis, so long regarded as a surgical condition, may have an endocrinological basis, and I write to support this view.

In a Hunterian lecture I put forward the thesis that disorders of secretion and motility of the oesophagus and stomach are likely causes of some cases of incompetence of the oesophagogastric sphincter and hiatus hernia.

It is unlikely that all cases of hiatus hernia are so caused, for if so the failure rate following operative repair would surely be very great. In fact, though all reported series of cases show a failure rate in experienced hands it is very small, though ever persistent despite increasingly elaborate techniques of construction of valve mechanisms, fixation, and repair.

One of the principal points of my thesis is that some cases of hiatus hernia are anatomical hernia and nothing more. These will respond to a simple surgical repair. Others are due to a motility disorder of the oesophagus and stomach—probably of the foregut. These will not respond to a surgical repair directed to the hiatus and oesophago-gastric junction as if they formed an isolated and independent structure.

A second important point is that hiatus hernia has always been considered due to abdominal pressure pushing the stomach through a lax hiatus. It may well be due to traction by the longitudinal oesophageal muscle pulling the stomach up, especially in cases due to secretomotor disorder. While we can now measure the effects of gastrin and of many drugs on the circular muscle of the oesophagus we cannot measure the activity of the longitudinal muscle, which forms by far the larger proportion of the oesophageal muscle coat. An excessive longitudinal pull will exert a distracting effect upon the lower sphincter and accentuate any tendency to incompetence. A technique for measuring longitudinal muscle activity would be most welcome.—I am, etc.,

KENNETH MULLARD

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¹ Mullard, K. S., *Annals of the Royal College of Surgeons of England*, 1972, 50, 73.

Continuous Intra-gastric Feeding in Infants

SIR,—We were interested in the paper by Dr. H. B. Valman and others (2 September p. 547), as we have been using continuous intra-gastric feeding for the last two years. However, we have restricted the technique to babies under 1,500 g weight in whom it has proved difficult to maintain an adequate intake with frequent aliquots of feed by nasogastric tube.

We have tended to stop continuous feeding at a point when the babies were able to tolerate adequate volumes of feed in two- or three-hourly aliquots via a nasogastric tube, with a view to moving on to bottle-feeding when tolerated. It is not clear to us how long the authors maintained continuous intra-gastric feeding and whether this was continued past the time when the babies might have been expected to begin feeding from the bottle. The data given in Tables II and III suggest that some of the babies might have progressed to taking satisfactory amounts of feed by bottle within a relatively

short time. Surely some "light-for-dates" babies who are approaching 2 kg in weight will relatively early accept adequate volumes of feed from the bottle. In such babies we consider it important to encourage sucking and to allow the mother to feed and to handle the baby as soon as possible.

We have found that continuous intra-gastric feeding has been most helpful in babies of very low birth weight, particularly those less than 1,250 g. To achieve an adequate fluid and calorie intake in these babies (with a more modest aim of 40 ml/kg body weight of feed on the first day) has proved difficult via nasogastric tube even with very small aliquots hourly. Often with such babies we have found the problem of increasing volumes of gastric residue and a risk of regurgitation. We have found continuous intra-gastric feeding to be more successful, as judged by checking gastric residues. In very-low-birth-weight babies this regimen may require very low rates of between 1 and 2 ml feed per hour. We have found that the ease and accuracy of this technique have been considerably increased by the use of a constant-infusion pump, the one which we use being the Ho'ter pump.

The use of drip feeding from a conventional chamber requires considerable nursing supervision, especially at low rates of flow, and at the low rates described may be impossible. The unit in which we work, like that of the authors, is staffed mainly by pupil midwives and the optimum use of nursing time is important.—We are, etc.,

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Renal Dialysis and Transplantation

SIR,—The report just published¹ on dialysis and transplantation in the treatment of chronic renal failure gives a clear account of the influence of treatment on mortality. That nearly two out of three patients dependent on a machine can expect to be alive after three years is worth hailing as a real milestone in the advance of therapeutics.

Nevertheless it seems regrettable that the report is restricted to a survey of mortality with no evaluation of morbidity. There is now an established literature on the symptoms associated with the stress of living dependent on a machine. Such stress can produce not only an impaired capacity to work but significant disturbance in mood and in the harmony of family life. For example, it is not uncommon for patients to develop an apparent inability to co-operate in dietary and fluid restriction, resulting in hyperkalaemia. There is often good reason to suspect that many of those who die do so for reasons related to an impaired desire to live. Yet many patients enjoy living with a remarkable lack of distress.

It is not our purpose in drawing attention to the extensive morbidity to discourage implementing facilities for treating all who need it. However, those responsible for implementing these recommendations should be aware that staff to assist distressed patients will be required as an essential contribution to an effective unit. A social worker must be in contact with all patients and their relatives to ensure that every aspect of morbidity is