TABLE I—*Fingerprint Patterns on all 10 Fingers*

Subjects	Whorls (%)	Twinned Loops (%)	Ulnar Loops (%)	Arches (%)	Radial Loops (%)
Controls (1,000)	22	5	63	6	5
Wilson's disease (22) ..	21	8	59	8	4
Wilson's carriers (13) ..	24	3	59	7	7

TABLE II—*Right Thumb Patterns*

Subjects	Whorls (%)	Twinned Loops (%)	Ulnar Loops (%)	Radial Loops + Arches (%)
Controls (1,000)	25	14	60	1
Wilson's disease (22) ..	25	15	60	0
Wilson's carriers (13) ..	46	0	54	0

ever, their findings have never been confirmed, and they have become so widely quoted both in papers and books that they have come to be unquestioningly accepted. Thus, in Professor Sheila Sherlock's book[2] it is stated in the chapter on hepatolenticular degeneration that the genetic factor is emphasized by the finding of increased "whorl" fingerprint patterns in sufferers.

Wilson's disease is an inborn error of hepatic copper metabolism and it is inherited as an autosomal recessive. We have studied the palmar dermatoglyphs of 22 consecutive patients with the disease seen at the Royal Free Hospital as well as 13 parents and offspring of these patients who are obligative heterozygotes. Our controls consisted of 500 males and 500 females who were healthy, unrelated, and came from the south and west of England. The results for fingerprint patterns are shown in Table I.

It is clear from these results that the possession of the gene for Wilson's disease in a single or double dose has no consistent effect on fingerprint patterns. To ensure that an increase in whorls was not localized to the right thumb the figures for this digit are given in Table II.

Other dermatoglyphic measurements—namely, total finger ridge count, a-b ridge count, and atd angle were also normal in our patients with Wilson's disease and their heterozygous relatives.

We conclude that dermatoglyphs will not help with the diagnosis of Wilson's disease nor will they help to detect heterozygotes, and therefore they cannot be used to aid genetic counselling. We are unable to confirm any of the positive findings of Hodges and Simon.[1]

We are indebted to Professor S. Sherlock for permission to study her patients. T.J.D. is in receipt of a grant from the South-Western Regional Hospital Board and the United Bristol Hospitals.

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1 Hodges, R. E., and Simon, J. R., *Journal of Laboratory and Clinical Medicine*, 1962, **60**. 629.
2 Sherlock, S., *Diseases of the Liver and Biliary System*. Oxford, Blackwell, 1968.

The Artist's Eye

SIR,—With regard to the prevalence of red-green colour-blindness (Dr. Ann J. Gower, 2 September, p. 586), the recent findings of the follow-up at 11 years of children in the national child development study[1] showed that 6·1% of boys and 1·1% of girls were reported by examining school medical officers using Ishihara plates to have impaired red-green colour vision. Thus in this national sample about 1 in every 16 males was affected.—I am, etc.,

CATHERINE PECKHAM
National Children's Bureau,
London

1 Davie, R., Butler, W. R., and Goldstein, H., *Birth to Seven*. London, Longmans, 1972.

SIR,—Your leader writer was of course quite right (19 August, p. 434): many investigators in many places have found an incidence of colour vision defect of between 7 and 8% in males and of 0·4 to 0·5% in females. So Dr. Ann J. Gower (2 September, p. 586), rather than finding your statement "incredible" should surely be asking herself why she seldom discovers any such defect. She does not say at what age the children are tested. The Ishihara plates, though probably the best practical test for routine examination of school children, are by no means a perfect instrument, and older children with lesser degrees of colour vision defect may escape detection—probably without detriment.

A detailed study of the impact of colour vision defect upon education, carried out in Hampshire,[1] revealed incidentally that there was very considerable observer variation in the use of the Ishihara test. In view of the extensive use made of colour in teaching, particularly in the infant schools, it is important that significant degrees of colour vision defect should be detected at an early age so that these children are not at an educational disadvantage. A colour vision test applicable to 5-year-olds is now in use in Hampshire schools.—I am, etc.,

LIONEL BACON
Winchester

1 Bacon, L. J., *Medical Officer*, 1971, **125**, 199.

SIR,—I was interested to read the letter from Dr. Ann J. Gower concerning colour-blindness in school children (2 September, p. 586). It is a pity that she does not quote any figures.

A few years ago I formed a similar impression of an unusually low prevalence of this condition in the schools where I performed routine medical examinations. When I came to check this I found that out of 308 boys in two schools there were 22 cases of red-green colour-blindness as shown by the Ishihara plates. This is a prevalence of just over 7%, or 1 in 14, not very different from the figure of 1 in 12 to which Dr. Gower takes such exception.

I did incidentally find a higher prevalence in grammar school boys (8 out of 85 or 1 in 10·6) than in boys at a secondary modern school where there were 14 cases out of 223 boys examined, a prevalence of 1 in 15·9. I do not know the significance of this, but it might explain the small number of cases found by Dr. Gower. I believe that a higher prevalence of refractive errors has also been found in boys of grammar school standard.—I am, etc.,

R. H. G. CHARLES
London W.5

Abortion Deaths

SIR,—I serve as one of the regional assessors on maternal deaths, and I must dissociate myself from Sir George Godber's preface to the *Report on Confidential Enquiries into Maternal Deaths in England and Wales 1967-69* (Reports on Health and Social Subjects No. 1).

Sir George writes: "In 1969 there was a substantial reduction in the number of deaths due to abortion." In chapter 5 on Abortion (page 47) the following figures of deaths from abortion reveal clearly a drop in deaths from illegal abortions but a rise in deaths from legal abortions. The total remains approximately the same.

	Deaths from Abortion		
	Illegal Abortions	*Legal Abortions*	*Total*
1967	28	1	29
1968	29	5	34
1969	17	12	29

As the total number of abortions, legal and illegal, rises towards 200,000 per annum in England and Wales it seems likely that the *total* number of deaths from this operation (quite apart from subsequent serious complications) will keep pace.—I am, etc.,

HUGH CAMERON MCLAREN
Birmingham Maternity Hospital,
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Aetiology of Varicosity

SIR,—Mr. Denis P. Burkitt's data (3 June, p. 556) on the geographical distribution of venous disorders are very striking, and it is interesting to see the outcome of all the forms we fill in for him. However, I find the postulated mechanisms much less convincing. He implies throughout that rural Africans are happily free from constipation. This is certainly not their own opinion, though their definition of constipation differs from ours and would be something like less than two bowel actions a day. They frequently complain of it, and a loaded colon is commonly palpable in the African abdomen (at least, it is around here). Even though their stool transits twice as fast as that of people in the U.K., if its volume is, as Mr. Burkitt states, more than four times as great, the amount in the colon at any one time could be at least as great as that of people in the U.K. They might not have faecal arrest but the hypothetical pressure on pelvic veins would be no less. This would invalidate Cleave's explanation.[1]

Similarly, the alternative explanation that Africans do not strain at stool is an unproved assumption. They are avid users of

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. Nazareth[1] 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/mm^3. 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
J. R. D. MATTHEWS

Auckland Hospital,
Auckland, New Zealand

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 previously[1] 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 established[2] and other studies have suggested that hypertension is more common in hypothyroidism than in the general population.[3] Kountz and Hempelmann[1] 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 Burchell[4] 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 sugest that a combination of hypertension and severe atherosclerosis exacerbated by autoimmune hypothyroidism led to a dissecting aneurysm in this patient. —We are, etc.,

ANGELA M. HILTON
R. S. WHITTAKER

Departments of Medicine and Pathology,
University Hospital of South Manchester,
Manchester

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 1968[1] fresh evidence has been presented which indicates that there is no threshold below which lead does not interfere with metabolism. A recent Scandinavian study[2] 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 μg/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 Lane, 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 genotypes[1] shows that the distributions cannot be made to fit, even