of the specialized tissue. He felt that for inhibition to occur it should be shown that the vagus nerve innervated the entire ventricular specialized network, and that acetylcholine should be shown to suppress spontaneous activity of ventricular Purkinje fibres. It would seem that these two conditions also require to be met with regard to verapamil and the human heart before the claims of Drs. Livesley and Oram can be justified.—I am, etc.,

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¹ Vassalle, M., *American Journal of Cardiology*, 1971, 28, 245.

Epidemic Ataxia in Western Nigeria

SIR,—Dr. B. O. Osuntokun (3 June, p. 589) states, "I have found no evidence of viral infection as an aetiological factor [of epidemic ataxia]." It is true that efforts to isolate a virus have so far been unsuccessful, and Dr. Osuntokun is undoubtedly referring to the work of the Virus Research Laboratory of the Faculty of Medicine, University of Ibadan, although he did not say so. In collaboration with the Wesley Guild Hospital, Ilesha, Western Nigeria, we have been investigating the possible viral aetiology of this condition, and the first report is at the moment being prepared for publication. Experience dictates that we should not close our minds to the possibility of viral aetiology, as it is a known fact that in the developing countries, where laboratory facilities are relatively limited, a negative result does not permit one to pronounce a categorical verdict. A "no find" may well be due to a "no extensive" or "no exhaustive" examination. A classical example is the case of dengue viruses in Africa. Physicians and indeed scientists have for a long time concluded that the continent of Africa was a dengue-free zone. But in 1970 from our laboratory dengue viruses were isolated and fully documented.¹

In clinical virology, while there is the danger to look upon viruses as a diagnostic dump heap, there is also the equally dangerous risk of returning a negative verdict too early in the operation. An open mind will help open prospects for future closer examination.—I am, etc.,

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¹ Carey, D. E., Causey, O. R., Reddy, S., and Cooke, A. R., *Lancet*, 1971, 1, 105.

Dressler's Syndrome

SIR,—Dr. M. S. T. A. Lawrence and Professor Ralph Wright (11 March, p. 665) report a case of Dressler's syndrome complicated by tamponade unassociated with haemorrhage into the pericardial space. I would like to report a further case.

A 62-year-old man originally presented in May 1971 with an acute myocardial infarction. At follow-up two months after the first attack he was complaining of tiredness, dyspnoea, and shoulder and back pain. His chest x-ray showed an enlarged cardiac shadow with no sign of pulmonary con-

gestion. He was treated with diuretics and practolol without symptomatic or clinical benefit.

In November 1971 he was first seen at this hospital. At this time his jugular veins were filled to his ears, liver edge palpated 5 cm below the costal margin, 15 mm Hg arterial paradox, but no sign of fluid in his lungs. His chest x-ray showed a large globular heart shadow but no pulmonary oedema. He was admitted for further investigations, which, apart from Hb 12.3 g/100 ml and E.S.R. of 43 mm, were unremarkable.

The diagnosis seemed clear: post-myocardial infarction (Dressler's) syndrome, complicated by tamponade. An attempt was made to aspirate the pericardial fluid but was unsuccessful. He was started on prednisolone 60 mg a day with a brisk improvement in his condition. His jugular venous pressure fell to normal, and his cardiac shadow returned to a normal size and shape. Steroids were reduced to a maintenance dose of 10 mg a day. He has now been well for six months, apart from one episode of pericardial pain and a friction rub occurring a month after discharge, which responded to a brief increase in his steroid dosage. His steroids have now been tailed off.

This man seems a typical example of a postmyocardial infarction syndrome presenting two months after the initial infarct with tiredness, anaemia, a raised E.S.R., and signs of pericardial disease. He had physical signs of cardiac tamponade with x-ray confirmation. It seems unlikely that this could have been due to haemorrhage as he was not on anticoagulants and because he responded so well to steroid therapy. It seems likely that this situation can be complicated by pericardial effusion, severe enough to give tamponade and which responds very well to steroid therapy.

I would like to thank Dr. Turner for his permission to report his case.

—I am, etc.,

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Food and Cyanide

SIR,—I would like to comment on two specific aspects raised in your leading article (27 May, p. 481) which have been of particular interest to me since our original studies on the aetiology of tobacco amblyopia¹ and retrobulbar neuritis in pernicious anaemia.²

Firstly, it is certainly important to remember that, besides being present in tobacco smoke and alcohol, cyanide is widely distributed in the plant kingdom. In this connexion, I contended that retrobulbar neuritis in patients with vitamin B₁₂ deficiency who are non-smokers is the result of exposure to cyanide from some other source than tobacco.³ Abnormally raised plasma-cyanocobalamin levels have been found in some patients with the so-called "West Indian amblyopia"⁴ and in Nigerian patients with tropical amblyopia and ataxic neuropathy.⁵ The ophthalmic findings, as reported, are consistent with the strict criteria we specified and adopted in the diagnosis of tobacco-amblyopia visual fields,¹ and add further support to the hypothesis that exposure to cyanide from the dietary source

of cassava, or other roots or vegetables containing cyanogenic glycosides, contributes to the pathogenesis of tropical amblyopia.

If the indiscriminate dumping of industrial cyanide waste continues unchecked, with the inherent risk of pollution of soil and water supplies, there may well come the time when more widespread chronic cyanide neurotoxicity occurs from a dietary source in persons with a genetic or acquired error of cyanide or vitamin B₁₂ metabolism.

Secondly, I consider that your statement that "although it has been suggested that vitamin B₁₂ has a primary role in the detoxification of cyanide, this is not finally proved" is open to misinterpretation. It is certainly well recognized that cyanide is also converted to thiocyanate by an independent pathway via the enzyme rhodanese, which is present in vivo in high concentration in the liver.⁶ Clinical studies from many centres have shown conclusively that hydroxocobalamin, but not cyanocobalamin, is a powerful cyanide antagonist. Because confusion is likely to persist in the profession over the differences between various forms of vitamin B₁₂ commercially available, we have drawn attention to the superiority of hydroxocobalamin, and the possible adverse effect of cyanocobalamin in neuro-ophthalmological disorders, and submitted a case for the complete withdrawal of cyanocobalamin in favour of hydroxocobalamin for therapeutic use.⁷ In our view, the only indication for the continued availability of cyanocobalamin is its use in the performance of the Schilling test of vitamin B₁₂ absorption.

Looking to the future, it is possible that other cyanide antagonists may become available for our therapeutic use with the obvious advantage of oral administration. In such circumstances, I would strongly urge that all patients with proved tobacco amblyopia and patients with optic atrophy, myelopathy, or neuropathy of obscure origin should be very carefully screened for evidence of vitamin B₁₂ deficiency before embarking on any therapy other than hydroxocobalamin.—I am, etc.,

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- 1 Heaton, J. M., McCormick, A. J. A., and Freeman, A. G., *Lancet*, 1958, 2, 286.
- 2 Freeman, A. G., and Heaton, J. M., *Lancet*, 1961, 1, 908.
- 3 Freeman, A. G., *Lancet*, 1959, 2, 963.
- 4 Wilson, J., Linnell, J. C., and Matthews, D. M., *Lancet*, 1971, 1, 259.
- 5 Osuntokun, B. O., and Osuntokun, O., *American Journal of Ophthalmology*, 1971, 72, 708.
- 6 Smith, A. D. M., *Lancet*, 1961, 1, 1001.
- 7 Foulds, W. S., Freeman, A. G., Phillips, C. I., and Wilson, J., *Lancet*, 1970, 1, 35.

Neuromuscular Involvement in Pituitary Gigantism

SIR,—Dr. P. D. Lewis (27 May, p. 499) describes two cases of pituitary gigantism with neuromuscular involvement. I would like to report a case of similar neuromuscular involvement in a man aged 69 with acromegaly of at least eight years duration.

This man first presented in a Dublin hospital in 1964 with left foot drop; acromegaly was noted for the first time. The acromegaly has not apparently progressed since then, and clinical indices of activity have been negative over the past two years. His feet, however, have got steadily worse.