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CORRESPONDENCE

Correspondents are asked to be brief

Hypertension and Myocardial Infarction

SIR,—In a recent leading article (5 January, p. 1) you included hypertension among the main factors that "predispose" to myocardial infarction. Like Dr. T. Lovegrove (4 May, p. 279) I find it surprising that the validity of this almost universal assumption together with its inbuilt therapeutic implication, should have so long remained largely uncontested.

The evidence against it is formidable. Gross coronary disease is commonly found in normotensive subjects, while in some severe hypertensives the coronaries are unaffected.[1] Moreover, abnormal coronary angiograms are seldom associated with antecedent hypertension. The claim that hypertension contributes substantially to coronary degeneration appears to rest largely on its well-known correlation with angina. But it could equally be expected to provoke angina from a heart already damaged by coronary disease. It seems that we have been beguiled by the demonstrable causative link between hypertension and the consequent development of arterial lesions at other sites and in different forms.

Not only has hypotensive therapy failed to restrain the onset of coronary disease; it is even possible that it may somewhat have increased the risk of infarction, particularly when attempts have been made to restore the pressure to so-called normality. Coronary atheroma, whatever its origins, is surely more likely to lead to infarction if the diastolic filling pressure should fall below the capacity of any homoeostatic mechanism to sustain an adequate coronary circulation. Thus it is not unreasonable to suppose that an occasional excess fall of pressure in a patient on hypotensive drugs might precipitate infarction—for example, during sleep. Whatever its other effects, in such circumstances the original hypertension might have come to work as a saving influence.

There is some clinical support for this hypothesis. In a small study of young male hypertensives I was surprised to find that the incidence of infarction in a treated group was slightly greater than that for roughly comparable controls.[2] Similar observations made by Dr. E. D. Freis and his associates revealed that "nonfatal myocardial infarction occurred in five of the treated patients as opposed to two of the control group."[3,4] For some years I have noticed a disconcerting tendency for this development to occur in treated hypertensives just as electrocardiographic appearances of left ventricular strain have receded.

The immense overall benefits of modern hypotensive therapy have been proved beyond dispute. Nevertheless, whether or not hypertension may sometimes be a factor in the aetiology of coronary artery disease, we may have to recognize that these benefits are attainable only at the price of a somewhat increased incidence of myocardial infarction, especially if the pressure be lowered prematurely or the reduction pressed too far in a misguided attempt to restore "normality."

—I am, etc.,

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1 Harrison, C. V., and Wood, P., *British Heart Journal*, 1949, **11**, 205.
2 Stewart, I. McD. G., *Lancet*, 1971, **1**, 355.
3 Veterans Administration Co-operative Study Group on Antihypertensive Agents, *Journal of the American Medical Association*, 1970, **213**, 1143.
4 Stewart, I. McD. G., *Lancet*, 1971, **1**, 1073.

"Anticonvulsant Action" of Vitamin D in Epileptic Patients

SIR,—Dr. C. Christiansen and others (4 May, p. 258) report a reduction in the number of fits in epileptic patients treated with vitamin D_2 in doses of 4,000-16,000 IU daily compared with placebo. Four years ago we reported the results of a study on calcium metabolism in residential patients in the National Hospital-Chalfont Centre for Epilepsy.[1] We naturally wondered at this time whether correction of the plasma calcium level in patients who were hypocalcaemic might be accompanied by a reduction in fit frequency. We thought this would be unlikely because the plasma calcium levels in our patients were not low enough to have precipitated fits. The lowest value we encountered was 7·3 mg/100 ml in a 16-year-old boy whose case history was reported by Dent *et al.*[2] In this case the fit frequency was unaltered by vitamin D treatment despite a return of the plasma calcium to normal.

Following the publication of these reports we treated 21 patients with vitamin D_2.