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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 homeostatic 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 For some years I have noticed a disconcerting tendency for this development to occur in treated hypertensives just as electro-

cardiographic 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 D₂ 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 re-

duction 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 D₂.

They were divided into three equal groups, one receiving 2,000 IU daily, another 10,000 IU daily, and the third 50,000 IU daily. The patients were treated for up to one year and the number of fits occurring during this time compared with the number recorded during a similar period immediately before vitamin D treatment. The results are presented in the table. A breakdown of the results into treatment groups did not reveal a dose-related effect.

Effect of Vitamin D Therapy on Fit Frequency in 21 Patients (Major fits = with convulsions; minor fits = all other types).

Type of Fit	Fit Frequency		
	Reduced	Unchanged	Increased
Major ..	6	7	8
Minor ..	11	3	7

Despite the duration of treatment and the size of the doses given to the patients no increase in the mean plasma calcium level of the 21 patients was seen (8.9 mg/100 ml before and at the end of the treatment period), which is in accord with the findings of Dr. Christiansen and his colleagues. Contrary to their experience, however, all but two of our patients showed a fall in plasma alkaline phosphatase (mean values 10.4 K.A. units before treatment and 8.3 K.A. units after).

We did not find these results sufficiently encouraging to justify proceeding to a placebo-controlled study.—We are, etc.,

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¹ Richens, A., and Rowe, D. J. F., *British Medical Journal*, 1970, 4, 73.

² Dent, C. E., et al., *British Medical Journal*, 1970, 4, 69.

Postpartum Tubal Ligation and Local Anaesthesia

SIR,—The importance of adequate anaesthetic cover in maternity hospitals has constantly been emphasized to avoid the accidents referred to in the *Report on Confidential Enquiries into Maternal Deaths*¹ and to deal with the increasing demand for epidural analgesia. Until the anaesthetic services expand to meet these requirements it is difficult to demonstrate any priority for a general anaesthetic for the routine postpartum tubal ligation which occurs at short notice.

To overcome this problem a method of performing tubal ligation under local anaesthesia within 24–48 hours of delivery has been introduced in this hospital. The patient is prepared for the possibility of a general anaesthetic, and premedication with 100 mg of pethidine intramuscularly is given 30 minutes before operation. Immediately before scrubbing up diazepam is given intravenously—10 mg at first and more added during operation if necessary. Local infiltration is carried out with 20 ml of 1% prilocaine hydrochloride and a routine Pomeroy operation performed using plain catgut.

Of 208 patients in the initial study, three required a general anaesthetic, two because they were uncooperative in spite of additional diazepam and local anaesthetic and one because of adhesions. The remainder co-operated well and there were no respiratory difficulties, though often a total of 20 mg

diazepam was required. The patients found the degree of sedation and amnesia acceptable and the postoperative sequelae were less than after general anaesthesia.

Our impressions stimulated a prospective study, and a preliminary assessment of this shows the method to be so acceptable to staff and patients that we feel it could be the method of choice even if general anaesthesia was more readily available.—We are, etc.,

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¹ Department of Health and Social Security, *Report on Confidential Enquiries into Maternal Deaths in England and Wales, 1967–1969*. London, H.M.S.O., 1972.

Malaria in the United Kingdom

SIR,—In their article on this subject Professor L. J. Bruce-Chwatt and others (29 June, p. 707) conclude that “protection from malaria infection when travelling . . . is to a large extent the responsibility of the individual concerned. The medical profession can only advise the public at large and organizations concerned with overseas travel of the risk of infection.”

I would like to know where, when, and how the “individuals concerned” (who will number over a million this year, according to the authors) are expected to get the necessary information about protection. The ordinary traveller hardly ever approaches a practitioner before he goes abroad, and, unless things have considerably improved recently, he is unlikely to get much help even if he does. (Maybe the recent issue to all practitioners by the Department of Health and Social Security of the pamphlet referred to below may have had some useful effect.)

The only people the traveller usually sees about his journey if he is going by air, as 95% do, are the agent, the airport authorities, and the representatives of the carriers, including the cabin staff. The agents and the carriers do not seem to go out of their way at the moment to offer health advice to the traveller who may be going on some glamorous package tour to the malarious tropics. International regulations regarding smallpox, yellow fever, and cholera are carried out, as they have to be, but there the matter often ends. The excellent pamphlet “Notice to Travellers—Health Protection” (Health Departments and Central Office of Information) is supposed to be issued to all travel agents. Unfortunately there is no official pressure on the agent, as there should be, to offer a copy of the pamphlet or to disclose its contents to the potential traveller.

It is time the public was properly protected from its dangerous ignorance of the sometimes serious health hazards of travel abroad by making sure that the information available reaches them. As I see it, the major responsibility for this must rest with the agent and the carrier. Of course it is ultimately “the responsibility of the individual concerned” to swallow the anti-malarial tablets that he should have been told about. The same is true of using a life jacket and swimming if the plane falls in the sea. In the aircraft the public are warned by the public address system. Why not warn them about malaria?

The medical profession should be more positive than merely offering advice. It should go out of its way to influence the press, television, and the other media which spend so much of their time cajoling the innocent (and, in my view, unprotected) public to take a holiday “in the sun,” which could easily mean exposure to potentially dangerous diseases such as malaria. To put the onus for protection against malaria on “the individual concerned” seems to me to be completely unrealistic in present circumstances.—I am, etc.,

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Vein Stripping

SIR,—Dr. D. Freedman's arguments on vein stripping (18 May, p. 387) provoke some comments.

(1) It is not fair to quote the report by Galen in 150 A.D. (or, even worse, the operative report on the dictator Marius much earlier) as an argument against vein stripping. Firstly, the operative technique was very different from modern stripping. Secondly, Dr. Freedman may have heard of something called anaesthesia and proper postoperative treatment, including analgesics, anti-inflammatory drugs, and good bandaging, which certainly make the postoperative period something very far from “mediaeval torture.”

(2) It is correct that stripping does not take care of the incompetent perforators. It should be kept in mind, however, that varicose veins and incompetent perforators are not synonymous. They are two different states with different symptoms. If both are present at the same time measures should be taken against both, which is easily done at the same operation.

(3) Stripping from the ankle to the groin is possible in one stage in about 80% of cases of even very tortuous varicose veins. If not possible further incisions along the saphenous pathway are easily done without further harm to the patient or the surgeon. It is absolutely not true that “only normal veins can be stripped.”

(4) In our series of 5,400 ambulant stripplings the risk of postoperative complications was below 2%, even very slight infections and postoperative bleeding included. We had only three more serious complications (0.06%) none of which threatened life or limb. It is true that arterial complications were reported in the literature. In Sweden, however, six such cases were reported to the Medical Board. It was found that all these operations were done by inexperienced surgeons. It is unfair to judge a surgical method by the results obtained by surgeons unfamiliar with the basic technique of the operation.

(5) The long saphenous vein may be used as a spare part for reconstructive arterial surgery in very few cases. It should be remembered (a) that there is another saphenous vein; (b) that most stripplings are performed on women and the majority of arterial reconstructions on men; (c) that many methods, such as, for example, endarterectomy, are available; and (d) that it is a doubtful medical principle to abstain from the best possible treatment for the reason that the patient in the future might develop another disease.