couraged in general practice in the same way as they would be if they worked in a scientific department of an university.

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1 Williams WO. MD by thesis from general practice. *Br J Med Educ* 1969;3:171-5.
2 Williams WO. MD by thesis from general practice. *J R Coll Gen Pract* 1974;24:778-83.

β Blockers, lipids, and coronary atherosclerosis

SIR,—Dr Robin Northcote stated that the most consistent effect of β blockers without intrinsic sympathomimetic activity on plasma lipids is an increase in the concentration of triglycerides and a reduction in high density lipoprotein (12 March, p 731). Cardioselectivity does not appear to affect these responses.

We had the opportunity to study the effects of atenolol (a cardioselective β blocker without intrinsic sympathomimetic activity), given over 12 weeks, on the plasma lipid concentrations of 31 patients with acute myocardial infarction. There were 20 men and 11 women whose mean age was 54 (range 35-74) years. Atenolol 100 mg/day was started a mean of three days after admission because of postinfarction angina or as a secondary preventive measure. None of this group received thiazide diuretics.

We also studied the plasma lipid concentrations of 66 patients admitted with acute myocardial infarction who did not receive either β blockers or thiazide diuretics. This group consisted of 47 men and 19 women and their mean age was 58 (range 35-73) years.

Plasma lipid values were measured at admission (mean of four hours from the onset of pain) and 12 weeks after myocardial infarction. The Mann-Whitney U test and Wilcoxon matched pairs signed ranks test were used for statistical analysis.

In the β blocker group there was no appreciable difference in the plasma concentrations of cholesterol, triglycerides, or low density lipoprotein fraction between admission and 12 week values. There was, however, a significant increase in the high density lipoprotein by 12 weeks (table).

The control group showed no significant changes in the plasma cholesterol or triglyceride concentrations between admission and 12 weeks, although there was a significant reduction in the low density lipoprotein concentration and a significant increase in the high density lipoprotein at 12 weeks. We found no important difference in the concentrations of total cholesterol, triglycerides, or high density lipoprotein and low density lipoprotein fractions between the two groups at either admission or 12 weeks after myocardial infarction.

Our findings confirm other reports that atenolol does not cause significant changes in plasma total cholesterol or triglyceride concentrations.[1 2] England *et al* found that atenolol given for 12 weeks produced a fall in high density lipoprotein values,[1] whereas Eliasson *et al* found no change in this lipoprotein after 32 weeks of atenolol treatment.[2] Many of the patients studied by England *et al* received concurrent thiazide treatment, which makes the results difficult to interpret.[3] We conclude that atenolol, given for 12 weeks after myocardial infarction, caused no detrimental effect on plasma lipid concentrations.

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1 England JD, Simons LH, Gibson JC, Carton M. The effects of metoprolol and atenolol on plasma high density lipoprotein levels in man. *Clin Exp Pharmacol Physiol* 1980;7:329-33.
2 Eliasson K, Lins LE, Rossner S. Serum lipoprotein changes during atenolol treatment of essential hypertension. *Eur J Clin Pharmacol* 1981;20:335-8.
3 Northcote RJ, Todd IC, Ballantyne D. Beta-blockers and lipoproteins: a review of current knowledge. *Scott Med J* 1986;31:220-8.

Mean (and 95% confidence interval) lipid concentrations in patients receiving β blockers and controls

Lipid	β Blocker group (n=31)			Controls (n=66)		
	Admission	12 Weeks	p Value	Admission	12 Weeks	p Value
Cholesterol	6·13 (5·76-6·50)	6·29 (5·82-6·76)	NS	6·60 (6·25-6·95)	6·58 (6·29-6·87)	NS
Triglyceride	2·07 (1·48-2·66)	2·09 (1·64-2·54)	NS	1·75 (1·51-1·99)	1·98 (1·69-2·27)	NS
Low density lipoprotein	4·21 (3·84-4·58)	4·13 (3·68-4·58)	NS	4·83 (4·50-5·16)	4·51 (4·24-4·78)	<0·05
High density lipoprotein	0·99 (0·87-1·11)	1·21 (0·93-1·49)	0·001	1·01 (0·93-1·09)	1·18 (1·08-1·28)	<0·001

SIR,—Dr Robin J Northcote (12 March, p 731) quotes our study of treatment of hypertension in elderly patients[1] in support of the possibility that lowering diastolic blood pressure below 95-90 mm Hg may increase the risk of myocardial infarction.

In our trial there was no difference in the incidence of myocardial infarction (fatal or non-fatal) between the treated and control groups. There was a non-significant increase in the death rate from all cardiovascular causes with treatment in a small group of patients who had only systolic hypertension at the start of the trial—that is, systolic pressures of over 170 mm Hg and diastolic pressures of under 90 mm Hg. When we looked at mean attained diastolic pressures during the study the incidence of myocardial infarction formed a U shaped curve with higher rates at both high and low blood pressures.[2] This was present, however, in both the treatment and control groups, and the increased mortality at low pressures was therefore not an effect of treatment. We suggest that both the low pressure and the increased mortality are due to previous heart disease.

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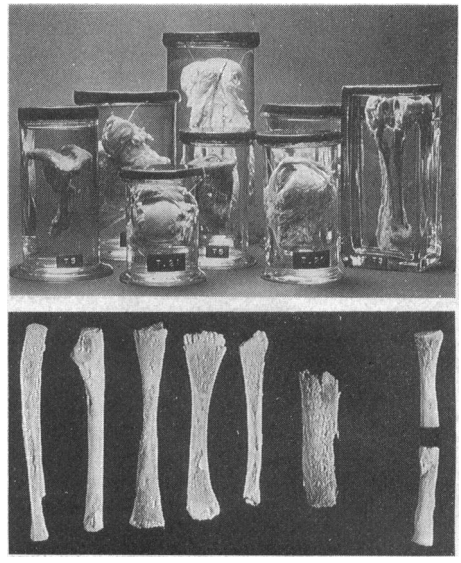
1 Coope J, Warrender TS. Randomised trial of hypertension in elderly patients in primary care. *Br Med J* 1986;293:1145-51.
2 Coope J, Warrender TS. Lowering blood pressure. *Lancet* 1987;i:1380.

Alive and well

SIR,—We are writing to you about your news item "Alas! poor Merrick" (12 March, p 794) concerning the whereabouts of certain museum specimens belonging to the collection of the Hospital for Sick Children, Great Ormond Street.



These specimens are now, and have been since 1985, in the care of the museum of pathology at the Royal Free Hospital, Hampstead. This relocation was made in an effort not only to preserve and record the collection but also to maintain the specimens until the entire collection can be returned when new accommodation is allocated. The photographs show the series of specimens presented by Barlow and Stills and part of the collection of bones known as Parrot's nodes.

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Notice of inadvertent repetitive publication

We regret that the letter by Dr S Mindel on "The full potential of ultrasound" (26 March, p 929) was substantially the same as the letter by the same author published in the *Lancet* ("The full potential of ultrasound," 30 January, p 244). We were not informed that this letter had already appeared and regret this inadvertent duplicate submission, which is against our Instructions to Authors and internationally accepted conventions.