	Post offices in deprived wards (n=16)	Post offices in other wards (n=70)	χ^2 with Yates's correction; df=1	p Value
No (%) carrying adverts	11 (68.8)	29 (41.4)	2.9	0.089
No (%) with adverts on two or more sites	5 (31.2)	15 (21.4)	0.3	0.61
No (%) with area of adverts $\geq 1\text{ m}^2$	8 (50.0)	17 (24.3)	3.0	0.08

Seventy five post offices (87.2%) sold non-post items, and 51 (59.3%) sold cigarettes. Post offices selling cigarettes were significantly less likely than those not selling cigarettes to ban smoking on the premises (11/51 v 16/33 odds ratio 0.3, 95% confidence interval 0.1 to 0.8; χ^2 test with Yates's correction = 5.5; $p=0.009$). No details on smoking bans were obtained in two shops. Post offices in deprived areas were more likely to carry tobacco advertising and to have a larger area of tobacco advertisements, although these differences fell short of statistical significance (table).

Comment

Tobacco advertising is widespread in Bristol, 30% of post offices carrying 1 m² or more of tobacco advertising. There seems to be a suggestion of more advertising in relatively deprived areas, findings which are similar to that of a study of billboard advertising in the Newcastle Metro.¹ Tobacco advertising in the United Kingdom is controlled by a voluntary agreement with the tobacco industry.¹ There has been recent agreement

to decrease the total number of external permanent advertising signs on shopfronts over the next five years, but the agreement makes no reference to the total area of advertising or the proportion of shops carrying advertising. Such a vague agreement has very little chance of being closely monitored and even less of being effective.

The Health of the Nation suggests a target for the reduction of the prevalence of cigarette smoking in men and women to 22% and 21% respectively by the year 2000.⁴ Evidence from New Zealand shows that a ban in tobacco advertising was associated with an immediate drop in both smoking prevalence and total consumption of cigarettes,³ yet despite this the British government is currently not supporting the proposed European Community Directive on a complete ban on tobacco advertising. Surely the time has come for the government to acknowledge the problems inherent in monitoring the voluntary agreement, recognise the strength of the new evidence on the effectiveness of tobacco advertising bans, and reconsider its position on this important public health issue.

We thank Miss S Lansbury, who visited and photographed all the post offices.

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Treatment of amiodarone induced thyrotoxicosis with carbimazole alone and continuation of amiodarone

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The antiarrhythmic drug amiodarone contains 37% iodine and is recognised to cause abnormal results of thyroid function tests in euthyroid patients as well as overt thyrotoxicosis or hypothyroidism in up to 10% of patients given the drug long term.¹ Withdrawal is recommended when the drug has induced thyrotoxicosis but is often inappropriate because of the underlying cardiovascular problem. The elimination half life of the drug, 90 days, determines that any response to withdrawal is slow. In addition, thyroidal iodine uptake is suppressed during treatment and after withdrawal¹ so that radioactive iodine treatment is ineffective. Thyroidectomy under local or general anaesthesia is an alternative² but may be hazardous in patients with cardiovascular disease.

Drug treatment is preferable to surgery in those with amiodarone induced thyrotoxicosis but treatment with thionamides alone has been found ineffective,^{1,3} especially in patients continuing amiodarone. Successful combination treatment with methimazole or propylthiouracil and potassium perchlorate has been reported in patients stopping amiodarone,⁴ as well as successful combination treatment with antithyroid drugs and high dose steroids,⁵ although adding potassium perchlorate or steroids has been found unhelpful by other researchers. None of these regimens are without hazard because of serious potential side effects of potassium perchlorate and prednisolone.

In view of these problems of treating amiodarone induced thyrotoxicosis we have explored other options. We describe treatment with carbimazole alone in five consecutive patients presenting with thyrotoxicosis and continuing amiodarone treatment.

Patients, methods, and results

Amiodarone induced thyrotoxicosis was diagnosed in each patient (all men; mean age 56.4 (range 31-72) years) from clinical features and circulating thyroxine, triiodothyronine, and thyrotrophin. Amiodarone (mean dose 180 (range 100-200) mg/day; mean treatment duration 27 (14-18) months) had been prescribed for resistant recurrent arrhythmias associated with ischaemic heart disease in four patients (two with supraventricular tachycardia and two ventricular tachycardia) and associated with the Wolff-Parkinson-White syndrome in one. Presenting features included weight loss (in all patients), tremor (two), increased sweating (two), fatigue (two), and worsening angina (two). No patient had a history of thyroid disease. A nodular goitre was present in one and none had ophthalmopathy other than lid lag. Concentrations of thyroxine and triiodothyronine were raised in all (free thyroxine, mean 70 (range 51.0-120) pmol/l, (normal range 9-24 pmol/l); free triiodothyronine, mean 13.0 (range 9.7->20) pmol/l, (normal range 3.9 pmol/l)) and thyrotrophin was suppressed to <0.5 mU/l. Thyroid autoantibodies were weakly positive in one patient (antimicrosomal titre 1:400, antithyroglobulin 1:80).

After amiodarone induced thyrotoxicosis had been diagnosed, treatment with carbimazole was started (starting dose 20-60 mg/day, mean dose 37 mg/day). Patients were biochemically euthyroid (had normal concentrations of free thyroxine and free triiodothyronine) after 3-16 (mean 8.6) weeks of carbimazole treatment and were clinically euthyroid after a mean of 14 weeks' treatment. Carbimazole doses were titrated

against free thyroxine, free triiodothyronine, and thyrotrophin, and treatment was continued for 12-15 months. One patient became hypothyroid while taking carbimazole and remained hypothyroid after drug withdrawal. He remained well on long term thyroxine replacement. Three further patients remained well and euthyroid 6-18 months after withdrawal of carbimazole. One patient's thyrotoxicosis relapsed 12 months after carbimazole was stopped; he was treated for a further 15 months and was euthyroid without carbimazole nine months later.

Comment

Amiodarone induced thyrotoxicosis was successfully treated in five patients with carbimazole alone and continuing amiodarone. The present report contrasts with previous descriptions of the ineffectiveness of carbimazole treatment in amiodarone induced thyrotoxicosis.^{1,3} The reason for this discrepancy is not clear, but it may reflect the iodine replete state of British patients, which contrasts with that of those in Tuscany and other iodine deficient areas, where carbimazole has been found to be unhelpful.³ In addition, there was evidence for pre-existing thyroid disease (goitre and

positive thyroid antibodies) in only one patient, whereas other series from outside the United Kingdom included many patients with pre-existing thyroid disorders.⁴ We suggest that in patients presenting with amiodarone induced thyrotoxicosis, especially in areas not deficient in iodine, carbimazole alone should be the preferred treatment. Amiodarone may be continued in such patients, and long term remission of thyrotoxicosis may still result.

We are grateful to the Birmingham cardiologists for permission to report their patients.

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Long term effects of tamoxifen on blood lipid values in breast cancer

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Tamoxifen is now established as adjuvant therapy in patients with operable breast cancer.¹ As large numbers of women take it for many years its other metabolic effects are important.² A recent trial^{3,4} has shown a lower incidence of fatal myocardial infarction in women receiving adjuvant tamoxifen. We present data on blood lipid values in a subset of patients from this trial.

Patients, methods, and results

The 44 patients studied were postmenopausal (mean age 59 years, range 45-74), had unilateral invasive breast cancer, and were included in the Scottish adjuvant tamoxifen trial¹ between 1980 and 1984 and followed up till 1990. Blood samples were taken preoperatively and at roughly annual intervals thereafter. Serum was stored at -70°C. Twenty four

patients in the treatment group received tamoxifen 20 mg daily for five years after mastectomy or until first relapse. At five years 18 were randomised again—eight to stop tamoxifen ("tamoxifen stop" group) and 10 to continue ("tamoxifen continue" group). Twenty patients in the control group were operated on but did not receive tamoxifen.

In 1991 the samples were slowly thawed and the total cholesterol and high density lipoprotein concentrations measured. These determinations were carried out by using an enzymatic method on a COBAS Bio analyser. The precipitant used in high density lipoprotein cholesterol analysis was magnesium chloride phosphotungstic acid. Two sample comparisons were made with Mann-Whitney non-parametric tests. Measurements made six months or less before any recurrence (local, distant, or contralateral) have been excluded. The mean pre-operative serum cholesterol concentration in the treatment group was 6.72 mmol/l (standard error (SE) 0.22) and in the control group 7.03 mmol/l (SE 0.21) (NS).

For each patient the preoperative concentration was taken as baseline and changes from this were calculated. The mean change in cholesterol and high density lipoprotein concentrations was then calculated for each group of patients for each year of follow up. For each patient in the tamoxifen stop and tamoxifen continue groups a new baseline was calculated, being the mean value of cholesterol and high density lipoprotein for years 1-5; and subsequent changes in lipid values were compared with this. The mean changes in serum cholesterol concentration are shown in the table. No significant differences in high density lipoprotein values were observed between either the treatment and control groups or the tamoxifen stop and tamoxifen continue groups (not tabulated).

Comment

This study shows that tamoxifen consistently lowers the serum cholesterol concentration and that this effect ends with cessation of treatment. No significant change in serum high density lipoprotein value was observed but, given the small numbers of patients studied, such an effect cannot entirely be excluded. The particular strengths of this study are that it comprises patients who were treated within a randomised clinical trial¹

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Mean changes in serum cholesterol concentration in each year of follow up for treatment group compared with control group and for tamoxifen stop group compared with tamoxifen continue group

		Year:				
		1	2	3	4	5
Treatment group	Mean change (mmol/l)	-0.52	-0.96	-0.96	-0.88	-1.02
	SE	0.34	0.21	0.25	0.22	0.24
	No of patients	21	15	16	17	19
Control group	Mean change (mmol/l)	-0.06	+0.13	+0.01	-0.01	+0.27
	SE	0.15	0.15	0.25	0.20	0.21
	No of patients	18	12	14	15	13
p Value		0.013	0.003	0.022	0.009	0.001

		Year:		
		6	7	8
Tamoxifen stop group	Mean change (mmol/l)	+0.29	+0.78	+0.74
	SE	0.34	0.38	0.28
	No of patients	7	6	5
Tamoxifen continue group	Mean change (mmol/l)	-0.10	+0.17	-0.39
	SE	0.17	0.20	0.22
	No of patients	9	8	5
p Value		0.169	0.220	0.037