potential artefacts described by Quinn and Allen, which, as they say, relate to changes in the interpretation of the World Health Organisation's rule 3 and changes in policy regarding inquiries to certifiers for further information. Overall, during 1979-94 the average annual European age standardised mortality from breast cancer in Scotland (39·0/100 000) was similar to that in England and Wales (39·7). If we assume that the data are of comparable quality then the higher incidence and similar mortality in Scotland imply that survival prospects are more favourable there than in England and Wales. In terms of relative survival from breast cancer, however, there were no substantial differences between Scotland and the English registries in the Eurocare study.[2] Although the European age standardised mortality for 55-69 year olds has been lower and more variable in Scotland, it has followed a similar trend over time to that observed in England and Wales. The recent fall in mortality observed in England and Wales seems, however, to have been less dramatic in Scotland (fig 1).

We agree that it is too soon for any recent changes in mortality to be attributed directly to the national breast screening programme. Preliminary analysis of the Scottish Cancer Therapy Network's recently completed national audit of breast cancer shows that, between 1987 and 1993, surgery to the axilla has increased, the quality of pathological reporting has improved, and the proportion of patients receiving systemic adjuvant treatment has increased (papers in preparation). While there are no grounds for complacency, this lends support to the view that treatment factors may indeed have some impact on mortality.

We thank Mike Quinn and Liz Allen for supplying the data from their paper.

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Several factors must have a role in improved figures

EDITOR,—Mike Quinn and Elizabeth Allen's suggestion that better treatment in breast cancer has led to a fall in mortality from 1990 onwards is interesting.[1] As the authors imply, the widespread use of tamoxifen can be only part of the explanation.

A more direct way of looking at the outcome of changes in treatment is to analyse trends in survival. Survival rates among 34 107 women with breast cancer diagnosed between 1968 and 1987 have been calculated from data from the Scottish cancer registries.[2] Figure 1 shows the five year relative survival rates for women aged between 35 and 74 with breast cancer diagnosed during four quinquennial periods before the introduction of the United Kingdom's national breast screening programme. Survival increased monotonically from 1968-72 to 1983-7 in each age group. The increase in five year relative survival between

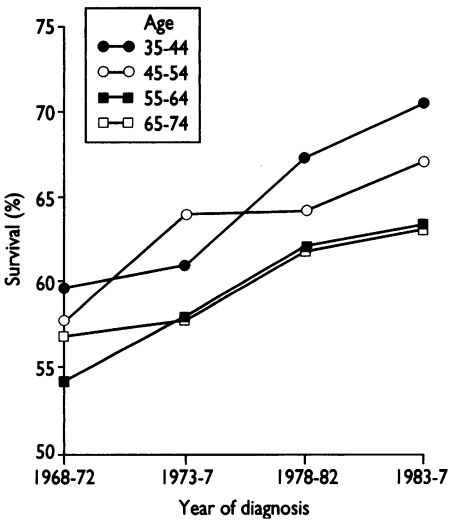

Fig 1—*Five year relative survival rates by year of diagnosis*

1968-72 and 1983-7 was larger in the age groups under 55 (11% and 9% for women aged 35-44 and 45-54 respectively) than in the older age groups (9% and 6% for women aged 55-64 and 65-74 respectively).

While use of tamoxifen since the early 1980s could partly explain recent improvements in survival in postmenopausal women, it could not account for the large increase in survival in the youngest age group, which is unlikely to have been prescribed tamoxifen.[3] This implies that treatment factors other than the use of tamoxifen must have a role. These are likely to include more effective and widespread use of chemotherapy and radiotherapy, the emergence of multidisciplinary breast cancer teams, and increasing specialisation of breast cancer surgeons.

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Women are often followed up too soon after treatment for cervical intraepithelial neoplasia

EDITOR,—Mark Emberton is right in saying that there is no uniformity of opinion among clinicians regarding the indications for and frequency of follow up after a procedure.[1] The individualistic approach to follow up continues even when national consensus has existed for some time.

It has been recommended that the initial follow up (whether by cervical smear testing alone or with colposcopy) of women who are treated for cervical intraepithelial neoplasia should be delayed until six months after the treatment.[2] Healing during this time often produces artefacts that may be mistaken for residual cervical intraepithelial neoplasia,[3] and this may result in further unnecessary treatment and increase the woman's anxiety. We looked at the interval between treatment of cervical intraepithelial neoplasia and the next intended follow up visit during a six week survey of colposcopy at all 19 hospitals in the former North West region in 1994.[4] During this period 246 women were treated at first colposcopy for suspected cervical intraepithelial neoplasia. The interval between treatment and follow up was six months (that is, in agreement with the national guidelines) in 93 of 241 cases. The remaining 148 women were asked to attend for follow up within six months of the treatment; this practice could be regarded as unnecessary and likely to overdiagnose residual cervical intraepithelial neoplasia.

Consultant gynaecologists in the region have been made aware of our finding, and we hope to see a greater concordance with the recommendation on follow up in a future survey.

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Prevalence of hepatitis C virus infection in Hashimoto's thyroiditis

EDITOR,—Duclos-Vallée *et al* have reported that infection with hepatitis C virus may have a role in triggering Hashimoto's thyroiditis.[1] Using enzyme linked immunosorbent assay (ELISA), they detected antibodies to hepatitis C virus in 12 of 50 patients, and a second generation recombinant immunoblot assay confirmed the finding in five of the 12 cases.

As we did not see such a high prevalence of hepatitis C in our outpatients we retested 118 consecutive patients with Hashimoto's thyroiditis for possible infection with hepatitis C virus. Hashimoto's thyroiditis was diagnosed on the basis of serum thyroid hormone concentrations below the normal range (total thyroxine $<64{\cdot}4$ nmol/l, total triiodothyronine $<1{\cdot}39$ nmol/l), an increased thyroid stimulating hormone concentration (>4 IU/l), and high concentrations of antibody to thyroid microsomes (>150 IU/ml) and to thyroglobulin (>250 IU/ml)). Blood samples were tested with an ELISA, and samples yielding positive results were retested with the polymerase chain reaction.[2]

Additionally, 59 patients in whom both the ELISA and the polymerase chain reaction gave positive results for hepatitis C virus were tested for Hashimoto's thyroiditis (using the above criteria).

Hepatitis C virus was detected by both the ELISA and the polymerase chain reaction in 3% of the patients with Hashimoto's thyroiditis (table 1); this prevalence was slightly higher than that in healthy people in the area (0·21%).[3] The prevalence of hepatitis A virus (53%) and hepatitis B virus (4%) did not exceed the prevalences in the area (hepatitis A virus, 20-87% depending on age[4]; hepatitis B virus, 1-5%[5]).

Among the 59 patients positive for hepatitis C virus according to the polymerase chain reaction, increased concentrations of antibodies to thyroid

Table 1—*Thyroid antibody concentrations (expressed as means (SD)) and prevalence of hepatitis antibodies in patients with Hashimoto's thyroiditis and with hepatitis C*

	Hashimoto's thyroiditis (n=118)	Hepatitis C (n=59)
Antibodies to:		
Thyroid microsomes (IU/l)	3196 (5875)	22 (50)
Thyroglobulin (IU/l)	1763 (5928)	52 (124)
No (%) of patients positive for:		
Hepatitis A virus	62 (53)	Not done
Hepatitis B virus	5 (4)	Not done
Hepatitis C virus	3 (3)	59 (100)

microsomes were seen in two patients and increased concentrations of antibodies to thyroglobulin in one. Table 1 shows the mean concentrations in both groups of patients. Thyroid hormone concentrations were below the normal range in only one patient positive for hepatitis C virus, and in this patient thyroid autoantibodies were not detectable.

We conclude that in patients with Hashimoto's thyroiditis the prevalence of hepatitis C virus is only slightly increased and the prevalence of hepatitis A and B viruses is not increased. Raised thyroid antibody concentrations, as seen in some patients with hepatitis C, may reflect a non-specific immune reaction or susceptibility to other autoimmune diseases.

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Method of election to BMA craft committees should be changed

EDITOR,—Douglas Carnall asks why doctors do not get involved in medical politics.[1] Two possible reasons that he does not mention are, firstly, the damage that many junior doctors believe that active involvement in medical politics can do to their careers and, secondly, a prevailing public ethos over the past 15 years that self interested behaviour is laudable but collective action is not. Carnall mentions the recent accusations that some craft committees are unrepresentative and out of touch.[2-4] I suggest that all crafts should take note of the unease that has been expressed about the way in which the General Medical Services Committee is elected.

A sizeable proportion of members of all craft committees are elected at the BMA's annual representative meeting. This effectively allows well known political figures to be re-elected without having to return to the "grass roots" to obtain legitimacy. Craft committees would be more representative and more in touch if doctors sat on them only after election by the membership as a whole.

It is, of course, always necessary to find a compromise between ensuring the efficacy and efficiency of the political process and being over democratic. I suggest, however, that the current practice of allowing election to craft committees from the annual representative meeting is counter productive if it allows cliques continually to re-elect themselves without needing to refer back to the wishes of the wider membership. Imagine the furore that would occur if a quarter of members of parliament were elected by members of parliament themselves rather than by the general public.

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Coronary heart disease and atrial fibrillation

Studies have not shown a causal relation

EDITOR,—Gregory Y H Lip and colleagues are not the first to state that coronary heart disease is top of the list of common causes of atrial fibrillation.[1] This "fact" continues to be quoted in journals, textbooks, and medical schools, yet the evidence for a causal link between chronic atrial fibrillation and coronary disease is weak, if such a link exists at all.

A distinction has to be made between conditions that happen to be associated and those that are causal. The data from the Framingham study depicted by the authors show hypertensive heart disease to be the commonest condition associated with the development of atrial fibrillation, occurring in 45·7% of male and 51·2% of female patients. Since the condition was common in the control population (men 28·3%, women 36·7%), however, its predictive value was not great, the relative risk of atrial fibrillation being 2·1 and 1·9 in men and women respectively. Cardiac failure and rheumatic heart disease had a high predictive value (relative risks in men 7·5 and 8·3 respectively and in women 5·7 and 15·3 respectively) but were less commonly associated (14·3% and 10·2% respectively in men, 14·3% and 26·5% respectively in women).

With regard to the supposed causal relation with ischaemic heart disease, when the influence of coexisting cardiac failure and rheumatic and hypertensive heart disease was excluded from analysis of Framingham data the independent effect of coronary disease on the development of chronic atrial fibrillation was not significant in either sex.[2] Even the relation with transient atrial fibrillation was found only in men. Onundarson *et al* studied over 9000 subjects and found no significant relation between the two conditions.[2] The coronary artery surgery study investigated 18343 patients with angiographically proved coronary disease and found atrial fibrillation to be present in only 0·6%,[3] a figure comparable with that found in studies in other populations.[4] Haddad *et al* noted only one case of atrial fibrillation in 496 patients with angiographically proved coronary disease.[5]

Surely if a link existed it would have been shown in these studies. I challenge Lip and colleagues to give hard evidence to justify placing coronary disease at the head of a list of causes of atrial fibrillation.

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Author's reply

EDITOR,—Nigel Wheeldon discusses the association between atrial fibrillation and coronary disease, but much depends on the criteria used for diagnosis. Many epidemiological studies have depended on electrocardiographic criteria or the clinical history. Studies depending solely on ST-T wave abnormalities may encounter difficulties in interpretation, especially if patients are taking digoxin or have left ventricular hypertrophy.

The frequency of coronary disease or other aetiological causes of atrial fibrillation depends on the population studied. In surveys of patients admitted to hospital acutely with atrial fibrillation, ischaemic heart disease was the commonest aetiological factor, followed by hypertension and valve disease.[1,2] The studies cited by Wheeldon in angiographically proved coronary disease could be considered to have been carried out in highly selected hospital based populations. Furthermore, atrial fibrillation may occur in patients with myocardial infarction or heart failure; when the latter is not due to hypertension, valve disease, or idiopathic cardiomyopathy, coronary disease is usually assumed to be the cause, especially in elderly patients.

In contrast to hospital based studies, population studies such as the Framingham[3] and Manitoba studies[4] reported that hypertension was the aetiological factor most commonly associated with atrial fibrillation, with coronary disease coming second. In these follow up studies data were analysed for causality (that is, the risk of atrial fibrillation developing). In the Framingham study, even after multivariate analysis, hypertension and heart failure remained important risk factors for atrial fibrillation.[3] In the Manitoba study, however, ischaemic heart disease was associated with one of the highest relative risks for atrial fibrillation (3·62 for myocardial infarction, 2·84 for angina) with a multivariate Cox model, compared with a lower relative risk from hypertension (1·42).[4]

The population study by Onundarson *et al* cited by Wheeldon studied only relatively young subjects (aged 32-64), in whom the prevalence of atrial fibrillation and ischaemic heart disease is usually low. Age is an important consideration: the prevalence of atrial fibrillation increases with age, and it is often presumed that the likeliest underlying cause is occult coronary disease. Unfortunately, there have been few population based studies in Britain, and published studies have been criticised as having been performed in unrepresentative populations.[5]

Colleagues and I recently completed a community based survey of atrial fibrillation in two general practices in Birmingham; the commonest aetiological factors were hypertension (in 37%), ischaemic heart disease (29%), and valve disease (26%). Interestingly, however, only a third of patients with atrial fibrillation had presented to hospital, which suggests that hospital based populations may greatly misrepresent the true