Pneumothorax after fine needle aspiration of the breast

SIR,—We are not surprised by Mr C A Gateley and colleagues' report of pneumothorax as a complication of fine needle aspiration of the breast.[1] We note that the authors are unaware of previous reports of this complication. In fact, the incidence of this complication was reported as 0·18% in a large Italian series of 74 000 fine needle aspirations of the breast,[2] and there have been several isolated case reports.[3,4]

Seven months ago we unsuccessfully submitted to the *BMJ* a report describing this complication in a young thin woman referred to the accident and emergency department in the evening by her general practitioner. She had complained of pleuritic chest pain and dyspnoea since fine needle aspiration of a benign breast lesion at the surgical outpatient department earlier that day.

We agree with Mr Gateley and colleagues that workers other than those actually performing the procedure must be aware of the hazards, however rare. This is particularly true for general practitioners and those working in accident and emergency departments, who may be presented with this delayed complication after the outpatient clinics are closed and those working in them have long gone home.

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SIR,—Mr C A Gately and colleagues reported the interesting but rare complication of pneumothorax after fine needle aspiration of the breast as if this was new information.[1] They even state that to the best of their knowledge it has not been reported previously, but this is simply not correct. The first report (of three cases) was in 1978,[2] and a further single case was reported in 1990.[3] This lack of a review of published work so that the authors appear as the first to unveil new information is surely not acceptable when a paper is offered to any major journal.

I am glad, however, that we came to the same conclusion: that if the aspirating needle is held tangentially to the chest wall during the procedure the pleura cannot be punctured by either the clinician or the patient.

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SIR,—I have two comments to make regarding Mr C A Gateley and colleagues' short report on pneumothorax as a complication of fine needle aspiration of the breast.[1]

In their study most of the doctors who performed breast aspirations that were complicated by pneumothorax were registrars; only one pneumothorax occurred after aspiration by a consultant. It has been our practice for the past eight years to restrict fine needle aspiration to designated aspirators (two consultants and one research registrar). Of roughly 8000 aspirations that were done during this interval, over 90% were done by these designated staff. During this period three penumothoraces occurred, two after aspiration by non-designated aspirators and one after aspiration by a designated aspirator. It seems that not only are experienced aspirators more likely to obtain a diagnostic aspirate[2,3] but they are less likely to cause a pneumothorax.

I disagree with the method described for performing fine needle aspiration cytology of lesions in the upper outer quadrant of the breast. If the lesion to be aspirated is firmly fixed between the index and middle fingers of the left hand then the fingers are driven down into the intercostal spaces and the lesion is moved directly over a rib. With this method one can insert the needle through the skin directly overlying the lesion, which I am certain increases the likelihood of hitting the lesion and obtaining a representative aspirate; one may occasionally hit the rib but it is almost impossible to puncture pleura.

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Blood pressure and myocardial infarction

SIR,—A surprising aspect of the debate on the J or U shaped curve is the apparent failure to recognise that when perfusion pressure is reduced blood viscosity will increase commensurate with the reduction in the rate of blood flow.

Dr Ralph B D'Agostino and colleagues noted that "Our data shed no light on the actual mechanism of the U curve," although they seemed to recognise that a problem of blood flow was involved by suggesting that "patients with severe coronary heart disease may be vulnerable to low perfusion pressures."[1] There is ample evidence that a combination of increased blood viscosity and reduced perfusion pressure may have fatal consequences.

Letcher *et al* showed that there was a direct relation between blood viscosity and blood pressure,[2] confirming a number of early reports. From this aspect the raised blood pressure can be interpreted as a physiological response to the increased peripheral resistance offered by the hyperviscous blood. Subjects with hypertension related to viscosity treated with antihypertensive agents would be at risk of having perfusion pressure reduced to a level below that required to maintain peripheral blood flow.

Dr D'Agostino and colleagues emphasised, however, that the U curve relation was found only in subjects with myocardial infarction. Chien concluded that abnormal blood rheology played a pathophysiological role in both hypertension and myocardial infarction.[3] The U curve for blood pressure may be linked to myocardial infarction through the effect of a partial occlusion on blood flow distal to the occlusion in coronary arteries. Partial vascular occlusion reduces the rate of blood flow distal to the occlusion commensurate with the degree of occlusion, and the reduction in flow rate results in an increase in blood viscosity. Therefore the greater the reduction in patency the greater the viscosity of distal blood. In hypertensive states the rate of flow through semipatient vessels may be sufficient to maintain distal blood flow, but the therapeutic reduction of blood pressure could result in the loss of perfusion pressure sufficient to maintain distal blood flow.

The ascending limb of the U curve of Dr D'Agostino and colleagues can be interpreted as a demonstration of the anticipated response to antihypertensive treatment of subjects with partly occluded coronary arteries, raised blood viscosity, and hypertension. Those subjects contributing to the lowest point of the curve (75-79 mm Hg) would have either the most viscous blood or the least patent vessels. With the progressive reduction in diastolic pressure, increasing numbers of subjects would have perfusion pressures that were inadequate to overcome the combined effects of high blood viscosity and poorly patent coronary vessels.

In practical terms this concept provides a basis for treating at least some patients with hypertension (for example, those with increased blood viscosity) as haemorheological problems. Treatment would be based on agents such as fish oil rich in ω3 fatty acids or oxpentifylline, both of which have been shown to have haemorheological benefits. The use of a vegetarian diet to lower blood pressure by the group in Perth, Australia,[4,5] was based on information that showed that vegetarians have low blood pressure, low blood viscosity, and a low incidence of coronary heart disease.

If the mortality associated with lowering blood pressure in subjects with myocardial infarction is to be reduced it seems essential to measure blood viscosity before deciding on the most appropriate means of reducing blood pressure.

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Improving survival after large bowel cancer

SIR,—Mr T G Allen-Mersh's editorial on surgical resection of hepatic metastases from colorectal cancer raises several important points.[1] The unexpected finding of hepatic metastasis at operation for apparently resectable primary disease should no longer be regarded as an open and shut case. Mr Allen-Mersh comments, however, that survival after resection of lesions detected by intraoperative ultrasonography "may be better" than that after resection of larger tumours detectable by conventional means. Even if this had been shown by an appropriate trial any difference is more likely to arise from lead time bias, estimated to be of the order of 16 months.[1,2] A 4 mm deposit (at the limit of detection of this technique) contains 10^8 cells which have had one to two years to metastasise, so it is likely that no increase in cures will be seen.

The rationale behind improving cure rates by gaining regional control of metastases is not new. Halstead and his successors explored increasingly radical chest and lymph node resections to prevent systemic metastasis from breast cancer. It is now

accepted that most systemic metastases are haematogenous de novo and that simpler means of locoregional control are equally successful.[3] The most effective adjuvant treatment is likely to be systemic agents such as fluorouracil and levamisole,[4] although a significant survival advantage associated with postoperative infusion of fluorouracil into the portal vein has been found, reaching a 60% reduction in the odds of death.[5] Other groups have repeated this study and confirmed the reduced mortality but found it to be of smaller magnitude; many of these studies, however, were unable to show any reduction in the incidence of hepatic metastases—presumably the improved survival arose from systemic effects of the fluorouracil.[6] The benefits of portal vein infusion are being reassessed in the axis trial of the United Kingdom Coordinating Committee for Cancer Research; the flexibility of the trial's design could perhaps accommodate a desire by the surgeon to resect small lesions before portal vein infusion.[7]

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Adenoma screening and colorectal cancer

SIR,—The central theme of the editorial by Drs Allyson M Pollock and Philip Quirke is that the value of polypectomy as a means of preventing colorectal cancer is unproved and by implication perhaps not worth while.[1] From their armchair viewpoint, however, the authors contribute little other than "knocking copy."

They challenge the "inevitability of the adenoma-carcinoma sequence," but no one has ever claimed this. Morson emphasised that only a small proportion of colorectal adenomas progress to carcinoma.[2] About 30% of the population have an adenoma by the age of 60, but the lifetime risk of colorectal cancer is only about 3%, which suggests that only 10% with adenomas develop cancer in their lifetime.

A minimum of five genetic changes are required for the formation of a colorectal cancer; fewer changes are required for the development of an adenoma.[3] Larger adenomas seem to have more genetic abnormalities, which fits with the clinical observation of their greater likelihood of containing cancer. Most adenomas are small, and it is indeed problematic to predict which could become malignant. From the observation by Muto *et al* that 40% of villous adenomas already had a focus of malignancy by the time of excision,[4] however, Drs Pollock and Quirke incorrectly conclude that the remaining 60% would never progress to cancer. They also state that "only 46% of polyps more than 2 cm will contain an invasive focus"; this figure, based on Muto *et al*'s surgical series,[4] is probably an exaggeration, but the incidence is still exceedingly high.

We do not know how to identify those adenomas that will grow, but the ease of endoscopic removal makes it unlikely that their natural course can be observed ethically as the polyp-cancer relation is widely accepted. It is, unfortunately, simplistic to call for an "urgent . . . randomised controlled trial of polypectomy" as Reasbeck has calculated that, even in high risk subjects, matched groups of 7000 patients would be needed to show a reduction in mortality from cancer.[5] Studying patients with an average risk would avoid the ethical problems of not subjecting the control group to colonoscopy, but the numbers in each group would need to be increased at least threefold (to allow for decreased compliance as well as a lower yield).

A society like ours must surely wish to identify those at risk of such a common and potentially preventable cancer, and (except for surgery in certain cases) polypectomy is currently our only weapon for reducing that risk. Epidemiologists may wish to debate available data, but surely the commonly held view that polyps present a golden opportunity to prevent cancer should remain the basis for surveillance. To denigrate colonic polypectomy in blanket fashion in a general medical journal is unreasonably nihilistic and clinically misleading.

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Pyoderma gangrenosum

SIR,—We were interested in the recent picture report of severe ulceration of the scalp.[1] Diabetes mellitus occasionally predisposes to severe skin infections, but the diagnosis of the ulceration in this case is unclear from the details given. The history of a rapidly expanding ulcer after minor trauma suggests pyoderma gangrenosum, for which paraproteinaemia is a known predisposing factor.[2] The scalp is an unusual though well recognised site for pyoderma gangrenosum, and on the basis of the dramatic photograph we suggest that this was the diagnosis. Although the prognosis in such a severe case is likely to be poor, high dose systemic corticosteroids may be of dramatic benefit.[3,4]

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Surgeons and hepatitis B

SIR,—Dr David Snashall and colleagues,[1] commenting on Mr Stuart Kennedy's personal view,[2] ask why the doctors had not been immunised. The regrettable answer must be that despite the considerable amount of information available about high infectivity of hepatitis B surgeons as a group have not appreciated the risks to which they are exposed and the protection that may be obtained by immunisation.

There has been much discussion recently within the surgical royal colleges and the surgical specialty associations about transmission of HIV during surgical procedures. With the aim of determining attitudes about testing patients and doctors for HIV the Federation of Surgical Specialty Associations—representing the 10 major specialty associations—recently sent a questionnaire to all surgeons. The opportunity was taken to ask also whether the respondent has been immunised against hepatitis B and whether he or she thinks that such immunisation should be compulsory. The fact that the question has been asked may encourage those not immunised to seek this protection. In addition, the answers should give some indication of opinion about the whole question of the risk of infection from surgical practice.

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Taxation of alcoholic beverages

SIR,—In the *Observer* colour magazine of 28 July 1991 Dr John Collee wrote about the excessive drinking of alcohol. His article included the following paragraph:

A lead article by Luisa Dillner in the British Medical Journal points out that Britain will next year be committed to harmonising its import duty with the rest of the European Community. This will result in a fall in the price of most alcoholic drinks with, we anticipate, a dramatic rise in consumption.

This would seem to be a quotation from the *BMJ* leader on alcohol abuse earlier this year[1]:

The most immediate threat to the level of alcohol consumption will come from the European Community. Britain is committed to the harmonisation of duty within the single market next year, which will mean a fall in the retail prices of most alcoholic beverages in this country. The Institute for Fiscal Studies estimates that this will result in a 46% increase in the volume of alcohol drunk in each household.

This statement needs to be corrected. Although the original 1987 proposal of the commission might have posed a threat by setting a single rate of duty for each product group, the proposal was amended in December 1989 to provide for a minimum rate of duty to take effect on 1 January 1993 and a common target rate to be achieved over a longer period.[2]

This more flexible approach strikes a better balance between the member states' interest in determining tax revenues and health policy, and the European Community's interest in securing sufficient convergence of rates to abolish fiscal frontiers by 31 December 1992. In particular, the principle of a minimum rate leaves member states free to set the duty on alcoholic beverages at levels which reflect their health concerns.

A significant step in this direction was taken on 24 June 1991 when the economics and finance ministers reached political agreement on minimum rates of excise duty for beer and wine. Those for fortified wines (sherry, port, vermouth, etc) and for spirits will be set later this year.