1990). Only five people had used condoms on every occasion and eight people had not used them at all. In my clinic I have been asking girls who have had sex with strangers abroad how often they were able to persuade their partners to use condoms—or even withdrawal. I have so far reached zero out of seven. Although all the girls had asked the men to use condoms, they had refused, and the girls, all of whom had been drinking, then acquiesced after varying degrees of pressure. At least one is now positive for antibodies to HIV. I also see men who work in Africa on tours of duty lasting 6-12 months. Many have African girlfriends and are amazed when I tell them that the likelihood of the girls being positive for antibodies to HIV might be as high as 50%. Most of these men have wives and families in Britain.

As far as I am aware the only propaganda to the effect that sex abroad is dangerous is the posters at airports reminding preoccupied travellers that AIDS does not go on holiday. Warnings should be directed at business travellers as well as holiday makers, should draw attention to the risks of alcohol in reducing vigilance, and should point out that HIV infection is now more prevalent in most travel destinations than it is in Britain. Almost all our European neighbours have a greater prevalence of HIV, as do cities such as Bangkok and Rio de Janeiro, whose attraction for many visitors is readily available sex. Eastern Europe probably has a massively underestimated problem, and the position in Africa is already apocalyptic. Failure to confront these issues adequately is likely to have catastrophic consequences because although the number of cases of AIDS among those in the traditional high risk groups is tending to plateau, the number of cases in which AIDS is acquired heterosexually while travelling continues to grow exponentially.

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1 Communicable Disease Surveillance Centre. Acquired immune deficiency syndrome: United Kingdom, 1982-August 1990. *Communicable Disease Report* 1990;No 36.

The epidemiology of malpractice

SIR,—In his editorial on malpractice in the United States Dr Richard Smith expresses regret that Britain has no similar statistics.[1] We hope that the government, now that it is accepting liability for indemnity, will produce figures for this country.

The figures, however, would be quantitative and of no value to doctors except to increase alarm; to take preventive action a breakdown of medical causes is needed. We made a modest attempt at this by publishing an audit of 100 actions selected at random from the files kept in the legal department at the West Midlands Regional Health Authority.[2] In 16 cases the "adverse events" were due to minor unavoidable risks of treatment and in 39 they resulted from natural causes—not surprising as legal aid is granted by a panel of lawyers without a doctor on it. Contributory factors were failure of communication in 27 cases (16 of which might have been avoided by doctors talking to patients) and the attitude or personality of the patient in 20. After three years 73 actions had been withdrawn, many being non-runners from the start (each cost the health authority about £12 000 and the tax payer from £7500 to £40 000 because of legal aid expenses); 12 had been settled out of court; one was lost in court by the plaintiff; and 14 were pending, seven of which were due to definite negligence.

The files at the regional health authority are the best and only unselected source of what is happening at the hospital level as letters from plaintiffs' solicitors are directed to the regional solicitor and, in West Midlands region, all records are kept in one department as the solicitor is employed full time. Studies providing a medical breakdown are continuing. Allegations against doctors increased from 388 in 1986 to 475 in 1988, and certain trends were noted: requests to receive hospital records—without mention of the complaint—increased, many being because of speculative litigation; inquiries about deaths rose greatly and were sometimes precipitated by the cursory handling of the bereaved that can occur in a busy hospital; more people complained about minor procedures such as a haematoma after venepuncture; there was an appreciable decrease in actions against surgeons (surgeons may be finding more time to discuss the operation beforehand, mentioning unavoidable complications such as minor wound infection); and allegations of unwanted pregnancy decreased, probably because the Department of Health and medical defence bodies decided in 1985 that women should be told of the slight risk of failure of sterilisation and that this fact should be put on consent forms, which suggests the need to make these forms more informative in other specialties.

Allegations of malpractice in this region are successful in about 16% of actions. But these figures cannot be compared with the high ones in the United States as obvious non-runners are excluded there because American lawyers operating the contingency fee system obtain an expert medical opinion before taking on a case.

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Low level exposure to lead

SIR,—Professor W R Lee and Dr M R Moore[1] did not mention that low level exposure to lead can result in hypertension.[2,3] Europeans and North Americans have differing attitudes to the relation between lead and hypertension. Europeans tend to be uncritically dismissive and trivialise the relation, whereas North Americans uncritically embrace the association and consider that it has an appreciable impact on public health.[4] Despite these cultural differences the evidence strongly points to a causal link between low levels of lead exposure and raised blood pressure, with the physiological mediators of stress—that is, catecholamines, etc—playing a critical part.[5,6]

The views of Professor Lee and Dr Moore disagree with those expressed in the 1988 report of the Medical Research Council advisory group on lead and neuropsychological effects in children.[7] This report makes only weak and vague conclusions. It is not so bold as to dismiss a causal relation outright but does leave an overall impression that if the relation exists at all it is minor and trivial. As far as I know the Medical Research Council has not even addressed the issue of low level lead exposure and hypertension. This dismissive attitude to potential effects of low level lead exposure by a British government agency with a national overseer role is in direct contrast with the climate in the United States, where this issue seems to have been reviewed ad nauseam. Where does the balance lie?

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1 Lee WR, Moore MR. Low level exposure to lead. *BMJ* 1990;301:504-6. (15 September.)
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AUTHOR'S REPLY,—Dr Dan S Sharp draws attention to hypertension as another outcome of low level exposure to lead. One of us (Dr M R Moore) was a member of the Glasgow group that first identified an association between low level exposure to lead and hypertension.[1] The British regional heart study, however, has shown that lead is a relatively minor contributor to hypertension.[2]

We do not agree that our views "disagree with those expressed in the 1988 report of the Medical Research Council advisory group on lead and neuropsychological effects in children." Rather, we reviewed the evidence that has been published since, pointing out the reasons why opinion seems to be moving toward accepting a causal relation, albeit small. If there is a relation then it could have profound implications. The cost of progressive abatement of lead exposure would rise alarmingly, though the perceived benefit may be relatively small compared with benefits from attention to other remediable causes of hypertension.

In the United States there is a strong impetus to proceed with lead abatement because of the association with hypertension, and there is at least as much attention given to lead as to the other possible causes. Are they trying to achieve the right result in the wrong sequence?

Dr Moore is now two years into an investigation to evaluate the effects of lead exposure, and we hope that in the future we shall be able to take a more balanced outlook than that currently taken by some protagonists.

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Three types of erythromelalgia

SIR,—Dr Andrew J Carmichael and colleagues question our proposed classification for erythromelalgia.[1,2]

The symptoms of painful red feet and ankles with sparing of the toes that they describe are not characteristic of erythromelalgia associated with thrombocythaemia. Moreover, true erythromelalgia causally related to thrombocythaemia is ruled out by the absence of microvascular thrombotic lesions in skin biopsy specimens and by the ineffectiveness of aspirin. Therefore the unclassifiable condition in these two patients resembles secondary erythermalgia.[2]

Dr Carmichael and colleagues also challenge the association of the conditions such as thromboangiitis obliterans, atherosclerosis, and other vascular diseases with secondary erythermalgia because a "warm skin is an integral part of the erythromelalgia triad." From a semantic point of view the authors' remark is justified. The link has, however, repeatedly been reported,[3-5] and we therefore considered it justified to rank these

erythromelalgia-like conditions under secondary erythermalgia. As yet, none the less, we have not observed this association.

The suggestion that the relation between connective tissue disorders and secondary erythermalgia is purely coincidental is highly speculative and certainly not based on scientific evidence. Dr Carmichael and colleagues state that "All is not red or white in the classification of erythromelalgia"; true enough in every clinical classification. Our objective was to clarify the existing information. We agree that the arrangement in primary and secondary erythermalgia is made merely on clinical grounds in the absence of clear cut laboratory indicators. In general, however, patients with burning pain and red swollen extremeties can be assigned with our classification.

Finally, the threat of therapeutic nihilism is limited because using our classification does not interfere with common and sound therapeutic practice.

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Tanning with ultraviolet A sunbeds

SIR,—Dr B L Diffey and others argue against using ultraviolet A sunbeds.[1] This is quite logical as the evidence suggests that exposure to ultraviolet light is harmful. The public, however, are unlikely to heed such advice unless the risks are perceived as overwhelming, and current behaviour indicates that sunbathing is viewed more like alcohol than smoking: enjoyable in moderation and dangerous only in excess.

Two questions therefore need to be asked. Firstly, Is it possible to "cheat" by obtaining a tan with ultraviolet A and not run the risk of skin cancer? And, secondly, Can using a sunbed before an annual holiday in the Mediterranean "protect" against the risk of melanoma? The answer to the first question is probably no. In humans the action spectrum for tanning exactly parallels that for erythema,[2] and in hairless albino mice the action spectrum for erythema is a good approximation of the spectrum for carcinogenesis induced by ultraviolet.[3] Experimental data also show that the induction of squamous cell carcinoma is a function of total dose.[4] In terms of non-melanoma skin cancer the idea of a safe tan is therefore a delusion.

The answer to the second point is more difficult. The incidence of cutaneous malignant melanoma is increasing, and this has been attributed to changing recreational habits.[5] Epidemiological data indicate that intensity of exposure to ultraviolet is more important than cumulative dose.[6] Skin type is a risk factor, but so is a history of sunburn in childhood.[7] Apart from lentigo meligna melanoma, cutaneous malignant melanoma is the only skin cancer induced by ultraviolet for which indoor workers are more at risk than outdoor workers. The relative risk of cutaneous malignant melanoma is lower in those who are exposed all year round than in those who are exposed for short periods, even though total doses may be equivalent.[8] These data suggest that tanning is protective and that people planning holidays in areas of high ultraviolet intensity might reduce their melanoma risk by promoting a tan before departure. An ultraviolet A sunbed is arguably a better method of tan promotion than sunlight as ultraviolet dose can be predicted more accurately and burning can be avoided. Unfortunately, ultraviolet A tanning offers little protection against erythema induced by ultraviolet B.[9] Tolerance to ultraviolet radiation is not dependent just on melanogenesis. Increased thickness of the epidermis and stratum corneum is also important, and this seems to be mainly caused by ultraviolet B.[10]

In summary, the use of sunbeds cannot be justified on either count. Gentle tan promotion may be protective for those contemplating holidays abroad, but those with skin types I or II would be better advised to avoid exposure to ultraviolet altogether.

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The new new general practice

SIR,—Those thousands of us who came home from the war in the late 1940s to approve, join, and establish the NHS must defend ourselves from the patronising inaccuracies of the article by Drs Anna Livingstone and David Widgery in the anniversary issue.[1]

Country general practice in Somerset, with our long established cottage hospitals, added a range of consultant outpatient clinics and "cold" surgery in our operating theatre within months in 1948 and, by 1953, our own maternity unit with resident staff, and soon a geriatric unit as well, sufficient for our needs. The domiciliary consultant service was prompt and rewarding to patient and practitioner alike. Certainly we practised from our homes, and our lives were an integral and vital part of the service we gave. We knew patients by their names in a relaxed atmosphere and in complete confidentiality. Our local medical societies held lectures by well known experts speaking on their own original work, and of course vocational training began 20 years before 1980, with away courses all over the country. And the trainee system was well established.

The 1966 charter arose from general practitioners' pressure, and two thirds of us placed our resignation papers in the hands of the BMA to strengthen its position. The minister responded within hours and many reforms came in, which we wanted. We were not hampered by suspension of the Rent and Rates Scheme if we attempted to resign then, for we owed them nothing.

To suggest that we had no antibiotics available before 1948 is ridiculous. The growing range of sulphonamides was available to us from 1939 onwards, and families of "panel patients" who were not covered for the cost of them were often supplied from the practice if they could not otherwise afford drugs. Penicillin was available in 1949.

Our mentally ill and mentally handicapped patients were cared for in hospital when necessary, and were not on the streets or in prison or under the underground trains. And waiting lists were a fraction of what they are now because Nye Bevan called them "a blasphemy."

The first 25 years of the NHS were years of achievement of which we were proud. And the constant and justified complaint by the public, which is now sadly so common, had not come to pass—ask the older patients.

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1 Livingstone A, Widgery D. The new new general practice: the changing philosophies of primary care. *BMJ* 1990;**301**:708-10. (3 October.)

Sodium-lithium countertransport activity in red blood cells

SIR,—We would like to comment on the paper by Dr James D Walker and colleagues.[1] The authors found no significant difference in mean blood pressure between the two groups of parents. Previous reports from the authors' own[2] and other[3] laboratories have shown significantly higher blood pressure in parents of patients with diabetic nephropathy. This family history of hypertension has been held responsible for the raised sodium-lithium countertransport observed in diabetes. This latest study disagrees with this hypothesis and its genetic implications, but this is not commented on in the discussion.

The authors calculate a mid-parental value for sodium-lithium countertransport and use this in postulating modes of inheritance. But they give no justification for this calculation and no precedent in other biological measurements. They state that an increased maximum velocity was observed. We and others have shown that the standard assay for countertransport is performed at a non-saturating external sodium concentration so maximum velocity is not measured.[4,5]

Pearson correlation coefficients are dubious with such outliers as in their first figure. In addition, our statistical tables indicate that an r value of 0·37 (n=40) has borderline significance, with a p value around 0·05 not <0·001.

It is known that hyperlipidaemia can lead to raised sodium-lithium countertransport, yet this seemed to be excluded by history alone. The authors argue that previous studies have shown that 80-90% of variance in sodium-lithium countertransport in the general population is explained by genetic transmission.[6,7] One study[6] showed 80% variability was in pedigrees selected because of cardiovascular disease and is unlikely to be applicable to the general population, and the study in the general population found that only 50% of variability was attributable to genetic factors.[7]

Finally, they state that raised sodium-lithium countertransport in hypertensive patients is associated with increased risk of renal and cardiovascular complications. The studies quoted in fact showed the incidence of raised countertransport in