negative for *H pylori* admitted to flare ups of pain.[2] The fact that such symptoms probably resulted from coexistent reflux or functional bowel disease rather than recurrence of ulcer is not important in this context.

Deferring retesting of patients' *H pylori* status until symptomatic relapse has a superficial economic appeal, as the urea breath test is relatively expensive. The savings made through not retesting patients who remain asymptomatic will, however, be partly offset by the extra costs of treating patients who suffer a relapse. Even simple relapse will incur added costs (drugs, consultations, loss of work) before repeat testing is arranged, and the costs of just one complication would finance many breath tests. In a 12 month follow up study, among 66 patients with ulcer who remained positive for *H pylori* after eradication treatment two bled from an ulcer and two were admitted to hospital with abdominal pain.[3]

Excluding patients with a history of complicated ulcer and advising patients to reconsult if symptoms recur will not remove the possibility of patients presenting with severe symptoms or complications. Sonnenberg and Townsend estimated the costs of treating duodenal ulcer with alternative management strategies, including treatment to eradicate *H pylori* both with and without subsequent testing for *H pylori*.[4] When use of a post-treatment test costing up to $400 was assumed, routine verification of eradication seemed less expensive than awaiting symptomatic recurrence and resulted in patients spending less time with active ulceration.

Evidence is accumulating to support a change from Schwartz's dictum of "no acid, no ulcer" to "no *H pylori*, no ulcer." But what about "no pain, no *H pylori*"? We urge caution in the implementation of a symptom based assessment of *H pylori* status after treatment, doubting both its reliability and its cost effectiveness. It seems harsh to require some patients to suffer a recurrence of symptoms before establishing whether the treatment has been effective. The wider provision of *H pylori* testing services should be a priority; patients' wellbeing should not be risked for marginal cost savings.

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1 Phull PS, Halliday D, Price AB, Jacyna MR. Absence of dyspeptic symptoms as a test for Helicobacter pylori eradication. *BMJ* 1996;**312**:349-50. (10 February.)
2 Reilly TG, Ayres RCS, Poxon V, Walt RP. Helicobacter pylori eradication in a clinical setting: success rates and the effect on quality of life in peptic ulcer. *Aliment Pharmacol Ther* 1995;**9**:483-90.
3 Powell KU, Bell GD, Bolton GH, Burridge SM, Bowden AF, Rameh B, *et al.* Helicobacter pylori eradication in patients with peptic ulcer disease: clinical consequences and financial implications. *Q J Med* 1994; **87**:283-90.
4 Sonnenberg A, Townsend F. Costs of duodenal ulcer therapy with antibiotics. *Arch Intern Med* 1995;**155**:922-8.

Routine retesting is necessary

EDITOR,—The decision whether to retest for *Helicobacter pylori* after a course of eradication treatment in peptic ulcer disease depends on the likely outcome. If one expects that the organism will nearly always be killed by a course of such treatment and that there will be few other dyspeptic symptoms not due to ulcer, then arguing against routine retesting makes sense. Perminder S Phull and colleagues adopt just such an argument on the basis of finding a 2.5% prevalence of symptoms of reflux and no other dyspepsia in their patients from whom *H pylori* had been eradicated.[1] This low figure for continuing symptoms is, however, at odds with figures reported elsewhere and suggests that the study population may have been preselected on the basis of having "pure" duodenal ulcer disease. We found that in 140 patients with peptic ulcer whose infection was successfully treated 39% reported heartburn, 25% reported symptoms of the irritable bowel syndrome, and 22% had a further consultation with the general practitioner during a median follow up of 249 days.[2] Powell *et al* found that 12-18% of patients with peptic ulcer used H_2 receptor antagonists in each three month period after successful eradication of *H pylori*.[3]

In practice, regimens to eradicate *H pylori* achieve a success rate of 85% at most. The 15-20% of patients in whom the treatment fails are highly likely to experience recurrent symptoms and to present again, and our figures suggest that up to a third of patients in whom eradication is successful will eventually present again. In other words, around a third of all patients given eradication treatment for peptic ulcer disease can be expected to visit their doctor again with dyspepsia. Routine retesting after eradication treatment enables the clinician to provide reassurance for those in whom it has been successful if they have recurrent dyspepsia and to prescribe repeat eradication treatment in advance of clinical relapse in those in whom it has failed; in addition, routine retesting may of itself reduce reconsultation rates. Routine retesting remains our practice.

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2 Reilly TG, Ayres RCS, Poxon V, Walt RP. Helicobacter pylori eradication in a clinical setting: success rates and the effect on quality of life in peptic ulcer. *Aliment Pharmacol Ther* 1995;**9**:483-90.
3 Powell KU, Bell GD, Bolton GH, Burridge SM, Bowden AF, Rameh B, *et al.* Helicobacter pylori eradication in patients with peptic ulcer disease: clinical consequences and financial implications. *Q J Med* 1994;**87**:283-90.

Screening for diabetic retinopathy

EDITOR,—J M Mason and colleagues state that the performance of high street optometrists in the Department of Health's study of screening for diabetic retinopathy was poorer than that of general practitioners and that this is surprising.[1] A tabulation that I have done of the results from that and other studies shows that the rate of detection of sight threatening diabetic retinopathy by high street optometrists and general practitioners when they use direct ophthalmoscopy alone is similar: the rate for general practitioners was 52% in one study and 55% in another, and that for optometrists 48%—that is, both groups miss about half of the cases.[2] As other studies in the tabulation show that even ophthalmologists, when allowed only direct ophthalmoscopy, have detection rates of only 64% and 65%, the main problem is shown to be not with the screeners but with the method used—direct ophthalmoscopy.

Mason and colleagues refer to recent work showing that specialist optometrists detect 71% of cases of sight threatening diabetic retinopathy with ophthalmoscopy, with this figure rising to 100% when photography is added. They erroneously reference a paper by Gatling *et al* as the source of these data. In fact, the data were collected in my department.[3] The optometrist, who had a (relatively good) detection rate when using ophthalmoscopy of 71%, was highly experienced, specialised in diabetic retinopathy, and had been screening large numbers of diabetic patients in the hospital diabetic clinic for many years. This cannot be extrapolated to the mass of high street optometrists using ophthalmoscopy alone.

Mason and colleagues are also concerned about the cost of adding photography, but is it that great? Once the patient is in front of the screener and has had his or her visual acuity measured and pupils dilated, taking photographs results in a minimal additional cost. In my department we estimate that our camera has undertaken of the order of 10 000 eye screenings in the past five years, and it is still going strong. The cost of the camera is well under £1 per patient and falling all the time. We use medical photographers of medical technical officer grade 2 at a cost of less than £1 per patient screened, but this cost is obviated if the ophthalmoscopist does the photography.[4] Polaroid photographs are about £1 an eye, and the instant digitised images that will probably characterise the photography of the future not only seem to provide higher detection rates[5] but remove the cost of the Polaroid photographs.

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1 Mason JM, Drummond MF, Woodward EG. Screening for diabetic retinopathy. *BMJ* 1996;**312**: 441. (17 February.)
2 Ryder B. Screening for diabetic retinopathy. *BMJ* 1995;**311**:1231. (4 November.)
3 Ryder REJ, Close CF, Gray MD, Souten H, Gibson JM, Taylor KG. Fail-safe diabetic retinopathy detection and categorisation by experienced ophthalmic opticians combining dilated retinal photography with ophthalmoscopy. *Diabetic Medicine* 1994;**11** (suppl 2):S44.
4 Jacob J, Stead J, Sykes J, Taylor D, Tooke JE. A report on the use of technician ophthalmoscopy combined with the use of the Canon non-mydriatic camera in screening for diabetic retinopathy in the community. *Diabetic Medicine* 1995; **12**:419-25.
5 Ryder REJ, Kong N, Bates AS, Sim J, Welch J, Carey SD, *et al.* Instant electronic imaging systems superior to Polaroid at detecting sight-threatening diabetic retinopathy. *Diabetic Medicine* 1996;**13**(suppl 3):S41.

The melanoma epidemic

Excess exposure to ultraviolet light is established as major risk factor

EDITOR,—Jonathan L Rees confuses histological nomenclature of early malignant melanoma and the relation between the risk of melanoma and exposure to the sun.[1] We need to separate the steadily increasing incidence of melanoma in all countries over the past 40 years from reported short term dramatic increases in localised areas. The short term increases are usually associated with increased awareness resulting in attention being drawn to melanomas that may have started to develop 10-20 years previously. The long term increase, however, is not an artefact and is causally related to exposure to the sun.[2]

The fact that pathologists now discuss the exact criteria for in situ melanoma, the radial growth phase, and early invasion is good news, since 10 years ago they were diagnosing thick tumours with a poor prognosis. What cannot be known is the natural course of early melanomas or those in the radial growth phase had they not been excised. A proportion would probably have progressed to the vertical growth phase with full capacity for metastatic spread.

Rees's arguments against exposure to the sun being a factor in the aetiology of melanoma are not original. It is well recognised that primary melanoma may occur on a covered site and that a high total lifetime exposure to the sun does not equate with an increased risk of melanoma. One of us and a colleague, however, have shown clearly that, per unit area of epidermis, the male ear (a site that has considerable exposure to the sun) has the highest incidence of melanoma of any part of the body.[3] In addition, patients with melanoma have a significant excess of solar elas-

tosis, actinic keratoses, and non-melanoma skin cancers, which are all objective indicators of excess exposure to the sun.[3]

Studies of migrants to Australia clearly implicate exposure to the sun in early childhood as a major risk factor for the development of melanoma.[4] British studies have identified large numbers of benign melanocytic naevi as the main independent risk factor for melanoma.[5] These observations have led to studies aimed at determining the main risk factors for developing naevi, one of which is exposure to the sun in early childhood.

Thus the case for excess exposure to ultraviolet radiation as a major risk factor for cutaneous malignant melanoma is established. More work is needed to define the relevant wavelengths of the spectrum and the exact quantity of ultraviolet radiation experienced by individuals of different phenotypes that lead to individual high risk. The pathogenesis of sunburn is also unclear, yet no one doubts the causal role of exposure to the sun.[2] Aetiological studies of melanoma over the long term should not be confused with current studies aimed at improving the nomenclature of early invasive malignant melanoma.

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1 Rees JL. The melanoma epidemic: reality and artefact. *BMJ* 1996;312:137-8. (20 January.)
2 Solar and ultraviolet radiation. *IARC Monogr Eval Carcinog Risks Hum* 1992;No 55.
3 Green A, Williams G. UV and skin cancer: epidemiological data from Australia and New Zealand. In: Young AR, Bjorn LO, Moan J, Nultsch W, eds. *Environmental UV photobiology*. London: Plenum, 1993:233-54.
4 Khlat M, Vail A, Parkin M, Green A. Mortality from melanoma in migrants to Australia: variation by age at arrival and duration of stay. *Am J Epidemiol* 1992;135:1103-13.
5 MacKie RM, Freudenberger T, Aitchison T. Personal risk factor chart for cutaneous melanoma. *Lancet* 1989; ii:487-90.

Author's reply

EDITOR,—I am saddened but not entirely surprised by the comments from Rona M MacKie and colleagues, which if nothing else provide an example of what I referred to in my final paragraph when I said that "the antithesis of science is not art but politics"[1]—in this case the politics of the prevention of skin cancer. I would, however, like to correct them on several points and disagree with them on others.

Firstly, I did not argue, as they suggest, against exposure to the sun being a factor in the aetiology of melanoma; quite the contrary. What is at issue is whether the secular trends in the incidence of melanoma are solely attributable to changes in exposure to sunshine. If they have objective evidence of these changes in individual exposure linked to rates then they should publish it rather than accept the usual chat of the glossy magazines and talk of package holidays, and so on. Their quasi-religious certitude on this issue contrasts with the more scholarly precision of a recent report from the National Radiological Protection Board (of which I note MacKie is a member).[2]

Secondly, I did not dispute that, after normalisation for surface area, melanoma is commonest on certain sites that are exposed to the sun. What I said was that most melanomas occur on sites that are only intermittently exposed and that the body site distribution of melanoma differs from that of squamous malignancy, in which the relation with exposure to the sun is more straightforward. Rather than bask complacently we would do better to worry about the biology underlying these differences. The authors' comments about nomenclature miss the point entirely. Once the material is in formalin, nature's own bioassay has been denied. You can have consensus over policy and nomenclature until you are blue in the face: you need experiments, and until some experiments are done the predicament that those of us in clinics find ourselves in will continue.

Finally, do the authors really believe that the causal pathway between sunshine and melanoma is as sufficient or as rich as that between ultraviolet radiation and sunburn? Of course, if you do not distinguish visible from ultraviolet radiation, etc, you would have to argue that not only would we have to block out the ultraviolet but we would also have to continue to stay in the dark.

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1 Advisory Group on Non-Ionising Radiation. *Board statement on effects of ultraviolet radiation on human health and health effects from ultraviolet radiation*. Chilton: National Radiation Protection Board, 1995. (Documents of the NRPB 6(2).)
2 Carey P, ed. *The Faber book of science*. London: Faber and Faber, 1995.

Figures have risen by over 150% over 10 years

EDITOR,—For the past 20 years I have kept accurate records of every malignancy that occurs in the practice in which I work (an urban practice with an average of 14 000 patients). Analysis of the figures for the past 10 years shows a 150% increase in the number of skin cancers, in particular basal cell carcinomas and melanomas (table 1). The survey covers the period from January 1986 until December 1995 and is complete.

Table 1—*Numbers of cases of various skin cancers in one general practice, 1986-95*

	1986-7	1988-9	1990-1	1992-3	1994-5
Basal cell carcinoma	31	29	30	46	71
Melanoma	2	2	2	5	7
Intraepithelial carcinoma	11	8	4	11	18
Squamous cell carcinoma	8	0	5	2	8

My data come direct from the general practice population as opposed to statistics on hospital outpatients. The increase shown here is alarming.

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HIV antibody test should be a routine investigation for undiagnosed pneumonia

EDITOR,—The General Medical Council's booklets *Duties of a Doctor* set out the basic essentials of good medical practice for three topics—confidentiality, advertising, and HIV infection and AIDS.[1-3] Having a separate booklet about HIV infection perpetuates the myth that this is different from all other conditions.

A 37 year old man was referred with a three week history of cough, fever, and weight loss. A chest *x* ray film was normal one week into the illness. He had basal crepitations and bilateral shadowing in the lower zone, thought to be consistent with resolving pneumonia. One week later, because the chest *x* ray film had not improved and no specific diagnosis was evident from the results of routine blood tests, sputum examination, or serological tests, bronchoscopy was arranged for four days' time. He was questioned twice about "risk factors," and none was elicited. He became ill three days after this consultation, discouraged his wife and friends from seeking medical help, and died within 24 hours. Necropsy showed *Pneumocystis carinii* pneumonia complicated by *Staphylococcus aureus* infection.

I contend that the clinicians should have felt able to order an HIV antibody test as part of routine investigations for undiagnosed pneumonia without feeling restricted by the General Medical Council's guidelines. While questioning failed to elicit risk factors, this must be a most insensitive way of trying to exclude exposure to HIV. In this case, knowledge that the patient was positive for HIV would have influenced his management and almost certainly altered his prognosis. It is as acceptable to counsel a patient after an HIV test as it is to tell someone that he or she has legionellosis, leukaemia, or syphilis. Performing any test to exclude a suspected diagnosis without mentioning all the possible differential diagnoses cannot breach the trust between patients and doctors. The level of discussion about investigations depends on the patient's understanding.

The guidance on confidential and sensitive treatment of all patients should be sufficient to deal with HIV infection and AIDS. The General Medical Council's booklet highlights only one specific distinction from other conditions: "the serious social and financial consequences which may ensue for the patient from the mere fact of having been tested for this condition." These consequences are unimportant compared with the value of excluding AIDS in a sick patient for whom specific treatment may be life saving. A doctor is not obliged to list in detail all the diseases that a patient does not have and therefore should not be required to tell a patient that he or she had an HIV antibody test.

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1 General Medical Council. *Duties of a doctor*. London: GMC, 1995.
2 Court C. GMC launches new "blue book." *BMJ* 1995;311:972. (14 October.)
3 Adshead G. Only doing my duty? *BMJ* 1995;311:965-6. (14 October.)

Torture should not be trivialised

EDITOR,—What a depressing example of blinkered self pity Alison Martin's short article on the subject of "A patient who changed my practice" is.[1] No one would defend for a moment excessive hours worked in inhumane conditions to the detriment of both doctor and patient, but to compare a hard weekend on call with deliberate, politically motivated torture shows a breathtaking lack of perspective. In the first, the doctor is doing worthwhile work, which he or she has chosen, for agreed remuneration. If the conditions become intolerable the doctor is at liberty to resign, as Martin did. To equate this with incarceration and interrogation by hostile and dangerous men, with the threat of violence to oneself or one's family ever present, demeans and trivialises the experience of the victim.

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1 Martin A. Torture *BMJ* 1996;312:415. (17 February.)