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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### 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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## 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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## 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-