work much is done to promote change and publicise the problem. One result has been the national survey of blindness and low vision in The Gambia[3] —a whole country was surveyed, producing the best hard facts on blindness in west Africa. Other activities include the promotion of local courses for an ophthalmic nursing diploma in Sierra Leone and Ghana.

By increasing awareness, both at home and abroad, pressure may be maintained on governments to develop services that will relieve this vast burden of suffering, and in this respect Dr Potter should be congratulated.

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Mesothelioma and exposure to asbestos

SIR,—Mr Peter Pockley's report on white asbestos and mesothelioma[1] contains several statements that are either misleading or controversial. Dr James Leigh and his colleagues in their as yet unpublished paper are quoted as stating that the risk of developing mesothelioma is related to the degree of exposure to asbestos. This is hardly a novel observation, having been made by Whitwell *et al* almost two decades ago.[2] It was subsequently reaffirmed by Doll and Peto.[3] Of greater concern, however, is his statement of the relative risk of mesothelioma being produced by crocidolite, chrysotile, and amosite. All the published evidence indicates that mesothelioma is rare in subjects exposed to chrysotile alone, and indeed McDonald and McDonald in their study of almost 8000 deaths in miners in Quebec found only about 30 deaths from mesothelioma, of which seven occurred in miners exposed to crocidolite.[4] Thus most of the evidence presently available suggests that chrysotile does not induce mesothelioma unless contaminated by the amphibole tremolite. This has also been ably discussed by Gibbs.[5]

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Tuberculosis and HIV infection

SIR,—Dr David Inwald found a low prevalence of infection with HIV-I in patients with tuberculosis in Zululand and concludes that preventive measures may avert the expected increase in HIV-I infection.[1] Unfortunately, the epidemic is now progressing relentlessly, as in many other sub-Saharan African countries. Current estimates predict that by the end of this year between 317 000 and 446 000 black South Africans will be positive for HIV-I,[2] a level higher than that in Uganda, which is thought to be the worst hit country in Africa. Natal and Kwazulu have the highest seroprevalence and fastest rate of spread.

HIV-I infection among the black community in Zululand is of relatively recent onset. Zulus attending sexually transmitted diseases clinics were first identified as a risk group in 1987-8.[3] Subsequently the countrywide picture of HIV-I infection seems to have changed from a type I pattern predominantly affecting white homosexuals to a type II pattern similar to that in other parts of sub-Saharan Africa.

Opportunistic infections with *Mycobacterium tuberculosis* are unlikely in patients with HIV-I infection acquired recently. The comparison made by Dr Inwald of HIV-I seropositivity rates in patients with tuberculosis in the Ivory Coast and southern Africa will therefore not reflect the recent escalation of HIV-I infection in South Africa. The considerable morbidity from tuberculosis among black people in South Africa[4] and the high prevalence of sexually transmitted diseases in rural Zululand,[5] however, suggest that in this population tuberculosis and HIV-I infection will be a major problem.

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Endoscopic and histological findings in subjects with dyspepsia

SIR—,The paper by Dr Roar Johnsen and colleagues presented further evidence that there was poor correlation between the macroscopic appearances of the upper gastrointestinal tract, microscopic histological findings, and the symptoms of dyspepsia.[1] They misquoted a paper of mine from 1985[2] as claiming that corpus (sic) and antral gastritis could cause symptoms. This is a pity because exactly the opposite was my hypothesis and the conclusions from that paper were entirely in agreement with theirs: "no relationship was found between the length of history, severity of pain and histological abnormalities."[2]

In the past 20 years an increasing number of tests have come on to the market in gastroenterology and we are under an obligation to analyse them for their usefulness. Armed with the latest from the Mayo Clinic in the late 1960s, I and others tackled hiatus hernia as a disease. It became clear that there were three basic abnormalities to account for the symptoms of gastro-oesophageal reflux: an anatomical hiatus hernia, oesophagitis, and reflux. The challenge was to find out a correlation between symptoms and each variable. Try as hard as we could, we could not find much correlation between the investigations and the symptoms. The same approach was applied to the symptoms of duodenal ulcer disease[3] and gall bladder dyspepsia.[4] The symptoms of an ulcer had anyway previously been shown to be non-specific,[3,5] and the radiological appearances of a duodenal ulcer did not correlate with symptoms either.[6]

Research to show that the epigastric pain of duodenal ulceration could be reproduced by acid in the lower oesophagus but not by acid in the duodenum or stomach[7] did not seem to worry anybody, yet it fitted into the overall hypothesis that the results of investigations in relation to symptoms might have some different explanation and should be treated with caution. Another paper showed that it was extremely difficult to measure why some surgeons advised surgery for a duodenal ulcer and that it was probably a random phenomenon.[8] These results were not negative or destructive, although they could be interpreted as such by opponents. They were positive because they showed that the "symptoms ruled OK."

In the mid-1950s and '60s two specific investigations became popular: tests of gastric acid secretion and oesophageal motility. Their clinical use was analysed critically by, respectively, Baron[9] and myself[10]—two people who had spent many years on the subject but could stand back and discuss honestly and intellectually their true value. I believe it is now time that endoscopists critically analysed the accumulating data about endoscopic appearances and the histology of the upper gastrointestinal tract in a similar fashion for the benefit of their colleagues and patients as well as those who pay the bills.

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Postviral fatigue syndrome

SIR,—In his letter Dr Anthony Knudsen comments[1] on the recent paper by Dr J W Gow and colleagues on the postviral fatigue syndrome.[2] Dr Knudsen refers to the fact that the aetiology of the syndrome has not been established and to the dearth of definitive pathological findings. Though he does not directly express an opinion, he mentions "the view held by some that the condition is stress related and of psychological origin."

The body of opinion that holds that the postviral fatigue syndrome has a physical, organic origin seems often to be criticised because it cannot produce "the evidence." Yet these critics seem quite sanguine about putting forward the hypothesis that the syndrome is of psychological or psychiatric origin without a hint of an opinion regarding the basis of this hypothesis, far less evidence to support it.

It is disheartening to see patients referred to a psychiatric clinic on the premise that if the test results are negative the inevitable conclusion must be that "it's all in the mind." This not uncommon equation suggests a view of psychiatric diagnosis and practice that is not reassuring. Diagnosis in psychiatry, as in other specialties, is made on positive grounds and with careful consideration of the clinical presentation. When a patient is "relegated" to a psychiatric clinic, having failed to receive treatment in a general hospital, it is difficult for both the patient and the psychiatrist. Some patients with the syndrome are appropriately seen at psychiatric clinics for various problems secondary to their illness, not the least of which is their experience of not being believed.