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“Operation Cataract”

SIR,—The fact that 17% of patients have waited for cataract surgery for over a year¹ is disheartening given the ages of patients affected. I am sure that the lists would be considerably reduced were outpatient cataract surgery the norm rather than the exception. In the eye unit here during the past full year, 1987-8, we carried out 1158 cataract procedures, which were spread among four surgeons. Of this number over half (660) were done as outpatient procedures. Among the remainder only a few patients were admitted on medical grounds. The largest number were admitted because of sociological factors such as living alone or having a long distance to travel—not uncommon of course in Canada. Virtually all these patients were operated on under local anaesthetic with neuroleptic agents, and the complication rate in outpatients compared with inpatients did not differ greatly.

At present we have waiting lists of around six months, although, of course, this varies somewhat from surgeon to surgeon.

The hospital is now mooring acquiring an old nurses' home to use for overnight stay for patients having outpatient surgery; it would be staffed by a single registered nurse. This would be at some cost to the patient, but it would be minimal; as a number of patients have already elected to stay at their own expense at local hotels and motels I think that it will be well accepted.

Most of the inpatients operated on during the past five years have returned home the following morning, and again this has not led to any great increase in complications. The idea of patients remaining longer than one day, much less being operated on under general anaesthetic, is by and large anathema in North America and certainly helps to keep the waiting list down and the bed utilisation up.

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In spectacular retreat

SIR,—In Bernard Dixon's opinion piece leprosy is in spectacular retreat.¹ I suggest that he may be misleading general medical readers and the general public with his optimism.

The disease is hardly beating a retreat in the face of modern pharmacology if only 10% of the world's known patients with active leprosy are receiving

the proper multidrug treatment recommended by the World Health Organisation since 1982.

We should be cautious before assuming that the polymerase chain reaction for detecting *Mycobacterium leprae* DNA is the final answer to the problem of detecting leprosy bacilli in people. Hartskeerl *et al* detected single numbers of bacilli in suspensions of purified leprosy bacilli from armadillos,² but when they tested liver tissue from armadillos the sensitivity dropped to 1000 bacilli. Good histopathological techniques can detect bacilli in biopsy specimens from patients with leprosy at densities of 500 per ml of tissue. The ability of the polymerase chain reaction to identify *M leprae* DNA in histological sections of biopsy specimens that contain few or apparently no bacilli—that is, paucibacillary—is currently being investigated.

I agree that the polymerase chain reaction will be a useful epidemiological tool, but for individual cases we need controlled investigation to be certain that a positive signal from the polymerase chain reaction indicates infection with *M leprae*. False positive results are known in other polymerase chain reaction systems, and in countries where leprosy is endemic technical stringency may not always be optimal. Paucibacillary leprosy is often difficult to diagnose even with combined clinical and histological findings. It may transpire that detection of *M leprae* DNA by the polymerase chain reaction is comparatively insensitive and thus contributes little to the management of individual cases, or that it is so sensitive that positive signals are detected in people who seem clinically normal or have lesions inconsistent with leprosy. I doubt that this new technology will supersede classic methods of diagnosis; we await statistical evaluation of its sensitivity and specificity in a clinical context.

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Sweeping away superstition

SIR,—Textbooks on leprosy¹⁻³ and even general medical textbooks⁴⁻⁶ no longer consider skin to skin transmission important in the transmission of leprosy. Instead there is ample evidence that the main exit of *Mycobacterium leprae* is in the nasal discharge⁷ and that the portal of entry is probably the upper respiratory tract.

The scientific evidence against skin to skin transmission comes from microbiological, histological, and experimental studies. Bacteria in general cannot penetrate intact skin, and *M leprae* has no distinctive features that would enable it to do so⁸; histological studies have not shown *M leprae* on intact skin,⁹ and experimental transmission of leprosy even to immunologically compromised nude mice does not occur when bacilli are smeared on to intact or abraded skin.¹⁰ A necessary condition for skin transmission seems to be direct inoculation—for example, into the foot pads of mice¹¹ or, rarely, after tattooing in humans.¹²

Dr Coleman's advice to Princess Diana of wearing gloves to prevent transmission of leprosy may have been well meant,¹³ but it cannot be regarded as a balanced scientific opinion derived from critical and informed assessment of evidence, particularly when he invokes an authority that cited no evidence for its statement.¹⁴

Perhaps we could also reiterate the widely held feeling that the word “leper” should not be used. To describe patients with leprosy as lepers only

perpetuates the stigmatisation associated with this disease.

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Detection of deep venous thrombosis

SIR,—In response to the letter by Mr A F da Silva and colleagues¹ the point that we wished to emphasise in our article was the significance of a negative C reactive protein concentration.

It is well recognised that there may be many reasons for a raised C reactive protein concentration,^{2,3} and for this reason a positive result is too insensitive to predict deep venous thrombosis after surgery. Even if daily monitoring is done post-operatively there may be insults other than thrombosis responsible for an unexpected increase. Contrast phlebography will still be necessary to confirm the diagnosis. In our experience, however, if the C reactive protein concentration is <10 mg/l then deep venous thrombosis is not a concern. It is the negative predictive value of the C reactive protein concentration that is significant.

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SIR,—The observations of Dr E A Thomas and others on the use of liquid crystal thermography in detecting deep venous thrombosis¹ are at variance with those of previous investigators^{2,3} and with ours in a similar investigation just completed. As Dr Thomas and others did not describe exclusion criteria for the use of thermography presumably none was applied, so that many patients with exothermic lesions of the legs—for example, local sepsis or trauma or chronic deep venous insufficiency⁴—were included in their study, giving a high number of false positive thermograms.

Of more concern are the false negative thermo-