screening tests that do not alter on follow up is problematical. It is best decided by agreed (albeit arbitrary) criteria that define the minimum test signals consistent with HIV infection. Those recommended by the manufacturer of the Western blot strips licensed by the Food and Drug Administration are reactions with viral protein bands p24, p31, and either gp41 or gp160.[14] It may, however, be six months before a recently infected person develops all these antibodies and thus, when there is a lesser reaction on Western blot, a donor can be fully reassured only after many months' follow up without increase in reactivity. Whatever is decided, it is doubtful whether blood that continues to be reactive in the local anti-HIV screening assay can justifiably be transfused.

Recent controversy over screening for infection with HIV in other contexts has tended to concentrate on these false positive reactions. Opponents of large scale testing programmes—for example, of antenatal patients or hospital inpatients—have invoked assay non-specificity as a reason not to attempt surveillance in populations with low prevalence of HIV infection.[12 14-16] There may be good reasons to hesitate before embarking on such initiatives, but the success of the British screening programme for blood donors shows that false positivity need not be one of them. Even on a single specimen a high degree of specificity can be achieved by applying several assays of different methods,[17] and this approach, which has worked well in confirmatory testing in the transfusion service, would be easy to apply in seroepidemiological studies. When the prevalence of HIV infection is low so few truly infected individuals would have an uncorroborated reaction by a single assay method that such reactions could be ignored.

A more proper concern, but one which has attracted less attention, is false negative results. These may arise because antibody has not yet appeared, because the antibody that is present is not detected, or because there has been a technical or clerical error. Errors may easily happen through fatigue or, when prevalence is very low, through boredom, and no system can be devised that will protect against all the ways that they can arise. If, however, laboratories were to test twice specimens from patients known to have suggestive illness or increased risk and doctors were never to accept unquestioningly an unexpected negative result some errors would be avoided.

Only by such attention to detail can the fullest advantage be gained from assays for antibodies to HIV. Properly used, these are excellent tests, and they will gradually win the good reputation they deserve. Moreover, the lessons learnt in developing, evaluating, and applying them will eventually raise standards in many other areas of diagnostic serology.

PHILIP P MORTIMER

Director,
Public Health Laboratory Service
Virus Reference Laboratory,
London NW9 5HT

1 Hodgkin P. HIV infection: the challenge to general practitioners. *Br Med J* 1988;**296**:516-7.
2 Hagen MD, Klemens BM, Pauker SG. Does the risk to the surgeon outweigh the risk to the patient? *JAMA* 1988;**259**:1357-9.
3 Esteban JI, Shih JWK, Tai CC, Bodner AJ, Kay JWD, Alter HJ. Importance of Western blot analysis in predicting infectivity of anti HTLV III/LAV positive blood. *Lancet* 1985;ii:1083-6.
4 Allain JP, Laurian Y, Paul D, Senn D. AIDS-Haemophilia French Study Group. Serological markers in the early stages of human immunodeficiency virus infection in haemophiliacs. *Lancet* 1986;ii:1233-6.
5 Ranki A, Valle S-L, Krohn M, *et al.* Long latency precedes overt seroconversion in sexually transmitted human immunodeficiency virus infection. *Lancet* 1987;ii:589-3.
6 Albert J, Gaines H, Sönnerborg A, *et al.* Isolation of human immunodeficiency virus (HIV) from plasma during primary HIV infection. *J Med Virol* 1987;**23**:67-73.
7 Gaines H, von Sydow M, Parry JV, *et al.* Detection of immunoglobulin M antibody in primary human immunodeficiency virus infection. *AIDS* 1988;**2**:11-5.
8 Lange J, Paul D, Huisman HG, *et al.* Persistent HIV antigenaemia and decline of HIV core antibodies associated with transition to AIDS. *Br Med J* 1986;**293**:1459-62.
9 Von Briesen H, Becker WB, Henco K, Helm EB, *et al.* Isolation, frequency and growth properties of HIV variants: multiple simultaneous variants in a patient demonstrated by molecular cloning. *J Med Virol* 1987;**23**:51-66.
10 Mortimer PP, Parry JV, Mortimer JY. Which anti HTLV III/LAV assays for screening and confirmatory testing? *Lancet* 1985;ii:873-8.
11 Reesink HW, Lelie PN, Huisman H. Evaluation of six enzyme immunoassays for antibody against human immunodeficiency virus. *Lancet* 1986;ii:483-6.
12 Lelie PN, Reesink HW, Huisman H. Evaluation for three confirmatory assays for antibodies to human immunodeficiency virus. *Vox Sang* 1988;**54**:84-91.
13 Hickman M, Mortimer JY, Rawlinson VI. Donor screening for HIV: how many false negatives? *Lancet* (in press).
14 Update: serologic testing for antibody to human immunodeficiency virus. *MMWR* 1985;**36**: 833-40, 52.
15 Meyer KB, Pauker SG. Screening for HIV: can we afford the false positive rate? *N Engl J Med* 1987;**317**:238-41.
16 Hudson CN, Howie PW, Beard RW. HIV testing on all pregnant women. *Lancet* 1988;i:239.
17 Glynn AA, Mortimer PP. HIV infection: the challenge to general practitioners. *Br Med J* 1988;**296**:714.

100 years of contact lenses

Leonardo da Vinci described the optics of contact lenses over 500 years ago. Centuries later German glass blowing technology finally achieved the necessary precision, and Fick, a Zürich ophthalmologist, fitted his first patient 100 years ago. A recent conference in London celebrated the centenary and reviewed the possibilities and problems of modern lenses.

The early lenses were scleral. These large lenses compress the limbal blood vessels and obstruct the exchange of tears, reducing the oxygen diffusion essential for normal corneal metabolism. They are used occasionally to fit eyes whose irregular contours preclude corneal lenses.

Rigid small diameter corneal lenses were developed in the 1940s. The exchange of tears and hence oxygen transmission is encouraged by the considerable lens movement that occurs with every blink. More recently gas permeable materials have been introduced. These allow oxygen to diffuse directly through to the cornea, and some of the latest are sufficiently permeable to allow extended wear.

Soft contact lenses originated in Czechoslovakia in the 1950s. They extend just on to the limbal conjunctiva and are immobile. Corneal oxygenation relies on their gas permeability, not on exchange of tear fluid. Worn either daily or continuously for up to three months, patients like soft lenses because they are comfortable. In contrast, rigid lenses are uncomfortable on the first use and tolerance builds up only over weeks. Soft lenses are optically inferior to rigid lenses: corrected visual acuity may not be so good, and astigmatism is not so readily corrected.

Most patients are fitted with lenses for cosmetic or social reasons. A minority wear them, often on the recommendation of an ophthalmologist, as the best means to correct their eyesight. These patients include high myopes and those with aphakia and keratoconus. Therapeutic soft lenses are used occasionally to treat conditions such as bullous keratopathy or recurrent corneal erosions.

Contact lenses are generally considered safe; but there are few data on their use in the community, and the incidence of complications can only be estimated. Hospital based studies show more complications with soft contact lenses, especially when worn continuously, than with rigid types. One study of elderly patients with aphakia showed that serious complications occurred 10 times more often with soft lenses worn continuously than with hard lenses removed daily.[1] In two years of follow up the most serious complication, suppurative

keratitis, occurred in 3% of the group wearing soft lenses continuously but in none of those removing their lenses daily. In a group of patients of all ages with aphakia followed over four years serious complications occurred in 55% of those wearing soft lenses continuously compared with 9% of those removing their lenses daily.[2] A case-control study of patients wearing contact lenses who presented to a casualty department showed twice the rate of complications in those wearing soft lenses compared with those wearing rigid lenses.[3]

Soft contact lenses reduce the natural flushing action of the tears and cause retention of micro-organisms and dead epithelial cells, which may predispose to suppurative keratitis. Other contributing factors include poor lens hygiene and lens induced damage to the epithelium facilitating the entry of organisms. Many patients do not adhere to the recommended cleaning and disinfecting routine, and often the responsible organism can be cultured from the contact lens case. A soft lens, acting as a bandage, may mask early symptoms of keratitis and cause the patient to present late, whereas discomfort is exacerbated by a rigid lens causing it to be promptly removed.

One hundred years ago contact lenses were used only when medically indicated. Today most lenses are fitted by optometrists as an alternative to spectacles. Soft lenses, particularly those worn continuously should be prescribed with caution. Instruction of the patient should emphasise the importance of lens hygiene and the need for early expert advice should problems occur.

B L HALLIDAY

Lecturer in Clinical Ophthalmology,
Moorfields Eye Hospital,
London EC1V 2PD

1 Graham CM, Dart JKG, Buckley RJ. Extended wear hydrogel and daily wear hard contact lenses for aphakia. Success and complications in a longitudinal study. *Ophthalmology* 1986;**93**:1489-94.
2 Graham CM, Dart JKG, Wilson-Holt NW, Buckley RJ. Prospects for contact lens wear in aphakia. *Eye* 1986;2:48-55.
3 Franks WA, Adams GGW, Minassian D. Contact lens related disease in an ophthalmic casualty department and the increased risk of soft lens wear. *Br Med J* (in press).

Treating the discharging ear in general practice

The pattern of disease producing otorrhoea has changed substantially over the past 40 years. In a large cohort study in Newcastle upon Tyne in 1947 6% of all infants had ear discharge.[1] Most cases were probably caused by acute otitis media, which is seen almost 100 times more often in general practice than in hospital. A large study in 1957 noted that ear discharge occurred in 29% of episodes of acute otitis media in children,[2] but more recent studies in general practice have found discharge in between 1·5%[3] and 2·5%[4] of episodes. This decreasing incidence of ear discharge might be caused by widespread and early use of antibiotics. But the role of antibiotics in treating acute otitis media needs further evaluation because of evidence that the cause of the disease is little altered by their use.

The causes of discharging ear are very different in general practice and hospital practice. In general practice acute conditions such as acute otitis externa and acute otitis media are the main cause, whereas in hospital practice chronic otitis externa, discharging mastoid cavities, and active chronic suppurative otitis media are more often the cause. The second national morbidity study showed that a general practitioner will see 35 episodes of acute otitis media for every 10 episodes of otitis externa and two of chronic suppurative otitis media.[5] Otitis externa, or inflammation of the external ear canal, is associated with otorrhoea in about 90% of cases[6] and occurs most often in the summer and autumn. Hawke found that exposure to water, use of cotton applicators, and previous use of eardrops predisposed to both acute and chronic otitis externa, and hearing aid moulds predisposed to chronic otitis externa.[7] In acute otitis externa the discharge is usually mucoid and the tympanic membrane unaffected, whereas in chronic otitis externa the discharge is usually purulent and may be associated with changes in the tympanic membrane.

One of the most important aspects of managing the discharging ear is distinguishing between "safe" and "dangerous" causes, which may be made difficult by the opaque discharge. For this reason aural toilet (cleaning the meatus using a head mirror, speculum, and cotton tipped probe) and follow up or referral may be necessary so that good views of the attic region may be obtained and "dangerous" causes, such as cholesteatoma, excluded. On page 1649 a survey of general practitioners' approaches to treating otorrhoea with a tympanic membrane perforation suggests that they are too conservative because of undue concern about the ototoxicity when using ear drops containing aminoglycosides. Most cases of otorrhoea seen in general practice, will not, however, be associated with a perforated tympanic membrane, and whether or not aural toilet is used probably matters more than which preparation is used. And how many general practitioners or practice nurses are capable of conducting aural toilet?

Aural toilet is seldom necessary for young children with otorrhoea after acute otitis media because the discharge resolves rapidly and the meatus may be damaged if the toilet is not properly performed. But aural toilet remains a most important aspect of managing otitis externa with moderate or considerable discharge not least because it allows topical treatment to be more effective.

Topical treatments with steroid and antiseptic drops may be all that is necessary in milder cases of otitis externa. The patient should avoid getting water in the ear by using cotton wool and soft paraffin to keep it dry and by avoiding self cleaning. More severe otitis externa may be managed by aural toilet every 24 to 48 hours and by packing the meatus with ribbon gauze soaked with steroid ointment. Management of the most severe cases is usually performed in hosptial using microscopes and suction apparatus.

JOHN BAIN
Professor of Primary Medical Care
IAN WILLIAMSON
Lecturer in Primary Medical Care

University of Southampton
Aldermoor Health Centre
Southampton
SO1 6ST

1 Spence J, Walton WS, Miller FJN, Court SDM. *A thousand families*. Oxford: Oxford University Press, 1954.
2 MRC Working Party for Research in General Practice. Acute otitis media in general practice. *Lancet* 1957;ii:510-4.
3 Bain J, Murphy E, Ross F. Acute otitis media: clinical course among children who received a short course of high dose antibiotic. *Br Med J* 1985;**291**:1243-6.
4 Ross K, Croft P, Collins M. Incidence of acute otitus media in infants in a general practice. *J R Coll Gen Pract* 1988;**38**:70-2.
5 Office of Population Censuses and Surveys. *Morbidity statistics from general practice. Second national study, 1970-1*. London: HMSO, 1974. (Studies on Medical and Population Subjects, No 26.)
6 Hicks SC. Otitis externa are we giving adequate care? *J R Coll Gen Pract* 1983;**33**:581-3.
7 Hawke M, Wong J, Krajden S. Clinical and microbiological features of otitis externa. *J Otolaryngol* 1984;**13**:289-95.