

syphilis to predict its devastating effect throughout Europe in subsequent centuries. We must look to continuing surveillance to help resolve the unanswered questions.

Evidence of only limited second generation transmission in the United Kingdom has led some to criticise public education campaigns.¹⁰ They have argued that too little attention has been paid to the considerable geographical variation in the prevalence of HIV infection and the importance of other major risk behaviours of sexual partners. Should prevention campaigns be directed solely at those at highest risk or is an approach also aimed at the general population the right one? Limiting our efforts to those at highest risk may be short-sighted. Our concept of high risk has evolved as the epidemic has spread—for example, in the early 1980s risk was restricted to gay men who reported sexual intercourse with partners in the United States.

The world does not divide neatly into those at high risk and those at low risk. Epidemics of sexually transmitted diseases are determined by rates of change of sexual partners and patterns of sexual mixing as well as by the transmission efficiencies of organisms.¹¹ British data suggest considerable heterogeneity in the number of sexual partners people have in a lifetime, and people may change their risk category as they move from periods when they change partners rapidly to periods when they have a more settled sexual lifestyle.¹² As those with high risk lifestyles may mix with those with low risk lifestyles both parties need to be informed of the risks of sexually transmitted infection. Prospective sexual partners do not come with a log book of their past partners and experiences, and many heterosexual people will not be aware of their partners' risks.¹² Perhaps the most important lesson

to be learnt from the accounts of second generation heterosexual transmission in the United Kingdom is that many of those infected did not perceive themselves as at risk.¹²

Uncertainty is a difficult message to put across. It neither sells newspapers nor makes good television. Scientists must be honest about where uncertainty lies. But the public should accept that scientists have a duty to warn. We should be prepared to act on this potential threat and invest in self protection in the face of it. Only then will the historians of the next millenium be able to congratulate us on "preventing the epidemic that never happened."

ANNE M JOHNSON

Senior Lecturer in Epidemiology,
Academic Department of Genitourinary Medicine,
University College and Middlesex School of Medicine,
London W1N 8AA

- 1 Evans BG, Noone A, Mortimer JY, Gilbert VZ, Gill ON, Nicoll A, *et al*. Heterosexually acquired HIV-1 infection in England, Wales and Northern Ireland. *Communicable Disease Report* 1992;2:R49-55.
- 2 Gilbert VL, Evans BG, Noone A, Mortimer JY, Gill ON. Second generation heterosexual transmission of HIV-1 infection. *Communicable Disease Report* 1992;2:R55-59.
- 3 Centers for Disease Control. *HIV/AIDS surveillance US AIDS cases reported through December 1991*. Washington, DC: US Department of Health and Human Sciences, 1992.
- 4 European Centre for Epidemiological Monitoring of AIDS. *AIDS surveillance in Europe to 31st December 1991; quarterly report No 32*. Paris: European Centre for Epidemiological Monitoring of AIDS, 1992.
- 5 Willocks L, Hamilton B, Povey S, Brettell R. HIV seroprevalence and antenatal clinics. *Lancet* 1992;339:622.
- 6 Moss A. AIDS and intravenous drug users: the real heterosexual epidemic. *BMJ* 1987;294:389-90.
- 7 Ades AE, Parker S, Bury T, Holland SJ, Davison CF, Cubitt D, *et al*. Prevalence of maternal HIV-1 infection in Thames regions: results from anonymous unlinked neonatal testing. *Lancet* 1991;337:1562-4.
- 8 Department of Health and Welsh Office Working Group. *Short term prediction of HIV infection and AIDS in England and Wales*. London: HMSO, 1988.
- 9 Working Group. Acquired immunodeficiency syndrome in England and Wales to end 1993. Projections using data to end September 1989. *Communicable Disease Report* January 1990:1-12.
- 10 Fernand D, Gerard J. Don't believe the hype. *Sunday Times* 1992 March 4:1.
- 11 May RM, Anderson RM. Transmission dynamics of HIV infection. *Nature* 1987;326:137-47.
- 12 Wellings K, Field J, Wadsworth J, Johnson AM, Anderson RM, Bradshaw SA. Sexual lifestyles under scrutiny. *Nature* 1990;348:276-8.

Diagnosing pulmonary embolism

If the lung scan is non-diagnostic attention should turn to the proximal leg veins

The importance of establishing the diagnosis in a case of suspected pulmonary embolism is beyond doubt. Clinical trials in the early 1960s established the untreated condition's high mortality (30%) and the effectiveness of giving anticoagulants.¹ Continuous intravenous heparin provides immediate and almost complete protection against recurrence of embolism² and should therefore be started as soon as the diagnosis is suspected. On the other hand, anticoagulants are not without risk: heparin given by continuous intravenous infusion for seven days carries a 5% risk of major haemorrhage; warfarin given for three months an 8% risk.³ A vigorous attempt should therefore be made to substantiate the diagnosis before anticoagulants are continued long term.

Although reviews traditionally conclude that pulmonary angiography should be used much more widely to diagnose pulmonary embolism,⁴ little evidence exists that this advice is taken. A recent British survey found that only one third of acute hospitals could provide the service, and in these pulmonary angiography was performed on average four times a year for suspected pulmonary embolism.⁵

Since its introduction in the 1960s the radioisotope ventilation-perfusion lung scan has dominated the investigation of this disease: a simple, minimally invasive test that is cheap and quick to perform and has reasonable specificity. A steady flow of retrospective studies in the 1970s comparing this technique with the definitive investigation of pulmonary angiography has recently been refined by two prospective

studies.^{6,7} These have identified two groups in whom a therapeutic decision can be made. The first is patients with a "high probability" scan result (10-20% of patients), which means the presence of multiple segmental or lobar defects in perfusion that are unmatched by defects in ventilation. Patients with such a scan result have a near 90% chance of embolism and should be given anticoagulants^{6,7} unless there is a history of previous embolism—probably the commonest cause of a false positive result—in which case comparison with old scans is desirable.

The second group consists of patients with a normal or near normal scan (15-40% of patients), in whom the diagnosis can be virtually excluded. In these patients another cause for the symptoms should be sought. In all other cases (40-70% of patients) the scan result should be regarded as non-diagnostic. Although scans from such patients may be categorised into various subsets with different probabilities of embolism, this does not solve the clinician's problem because the overall rate of angiographically proved embolism in this group is about 40%.^{6,7}

These findings have not, apparently, greatly influenced current clinical practice. Doctors are still relying on results of lung scans to decide patients' management without resorting to pulmonary angiography to clarify the diagnosis.⁸ The reasons for this are clear: angiography is relatively expensive, time consuming, and perceived to be risky. The last of these is a misconception. In skilled hands the procedure carries little

risk: in several large series the overall mortality was less than 0.5%, and all the deaths were in patients with severe pulmonary hypertension or right ventricular failure.⁹⁻¹¹

The recognition that pulmonary embolism is usually a complication of deep venous thrombosis provides a solution to the problem posed by a non-diagnostic lung scan. In nine out of ten cases the thrombus is located in the legs,¹² and the association with embolism is much stronger when the thrombus involves the proximal leg veins.¹³ Only rarely are other sites (for example, axillary, pelvic, or renal veins) or other sources (for example, fat or tumour emboli) implicated. Three good methods are available for identifying proximal leg vein thromboses: phlebography, Doppler ultrasonography, and impedance plethysmography. Phlebography remains the most reliable method,¹⁴ but Doppler ultrasonography and impedance plethysmography have the advantage of being less invasive and serial studies may be made in uncertain cases.¹⁵ Moreover, impedance plethysmography will detect the presence of 95%, and Doppler ultrasonography the presence of 91%, of phlebographically demonstrable acute thrombi in the popliteal veins or above.^{15 16}

Patients with a non-diagnostic lung scan, which most doctors currently regard as a negative result,⁸ therefore require phlebography, Doppler ultrasonography, or impedance plethysmography. If the result is positive the patient clearly warrants anticoagulant treatment. If the results are negative excluding pulmonary embolism completely is not possible: pulmonary embolism is associated with a negative phlebogram in 30% of cases.¹² The chance of subsequent clinical embolism in these patients is, however, small.^{14 17} Thus it is safe merely to observe such patients, giving them subcutaneous heparin if risk factors for thrombosis are present and to rescan them if the clinical situation changes. If the results of phlebography or non-invasive tests are inconclusive pulmonary angiography must be considered. If this is not

available locally the patient should be given heparin and transferred to a hospital that performs angiography.

N W MORRELL
Research Fellow
W A SEED
Professor of Medicine

Department of Medicine,
Charing Cross and Westminster Medical School,
London W6 8RF

- 1 Barritt DW, Jordan SC. Anticoagulant drugs in the treatment of pulmonary embolism. *Lancet* 1960;i:1309-12.
- 2 Girard P, Mathieu M, Simmoneau G, Petitpretz P, Cerrina J, Herve P, et al. Recurrence of pulmonary embolism during anticoagulant treatment: a prospective study. *Thorax* 1987;42:481-6.
- 3 Seed WA. Pulmonary embolism: part 1. *Vascular Medicine Reviews* 1991;2:71-83.
- 4 Windebank WJ. Diagnosing pulmonary thromboembolism. *BMJ* 1987;294:1369-70.
- 5 Cooper TJ, Hayward WJ, Hartog M. Survey on the use of pulmonary scintigraphy and angiography for suspected pulmonary thromboembolism in the UK. *Clin Radiol* 1991;43:243-5.
- 6 Hull RD, Hirsh J, Carter CJ, Raskob GE, Gill GJ, Jay RM, et al. Diagnostic value of ventilation-perfusion lung scanning in patients with suspected pulmonary embolism. *Chest* 1985;88:819-28.
- 7 The PIOPED Investigators. Value of the ventilation/perfusion scan in acute pulmonary embolism. *JAMA* 1990;263:2753-9.
- 8 Frankel N, Coleman RE, Pryor DB, Sostman HD, Ravin CE. Utilization of lung scans by clinicians. *J Nucl Med* 1986;27:366-9.
- 9 Dalen JE, Brooks HL, Johnson LW, Meister SG, Szucs MM, Dexter L. Pulmonary angiography in acute pulmonary embolism: indications, techniques and results in 367 patients. *Am Heart J* 1971;81:175-85.
- 10 Bell WR, Simon TL. A comparative analysis of pulmonary perfusion scans with pulmonary angiograms. *Am Heart J* 1976;92:700-6.
- 11 Mills SR, Jackson DC, Older RA, Heaston DK, Moore AV. The incidence, etiologies and avoidance of complications of pulmonary angiography in a large series. *Radiology* 1980;136:295-9.
- 12 Hull R, Hirsh J, Carter CJ, Jay RM, Dodd PE, Ockelford PA, et al. Pulmonary angiography, ventilation lung scanning and venography for clinically suspected pulmonary embolism with abnormal perfusion scans. *Ann Intern Med* 1983;98:891-9.
- 13 Moser KM, LeMoine JR. Is embolic risk conditioned by location of deep venous thrombosis? *Ann Intern Med* 1981;94:439-44.
- 14 Hull R, Hirsh J, Sackett DL, Taylor DW, Carter C, Turpie AGG, et al. Replacement of venography in suspected venous thrombosis by impedance plethysmography and 125-I fibrinogen leg scanning: a less invasive approach. *Ann Intern Med* 1981;94:12-5.
- 15 Huisman MV, Bulla HR, TenCate JW, Vreeken J. Serial impedance plethysmography for suspected deep venous thrombosis in outpatients. *N Engl J Med* 1986;314:823-8.
- 16 Lensing AW, Levi MM, Buller HR, Prandoni P, Vigo M, Agnelli G, et al. Diagnosis of deep-vein thrombosis using an objective Doppler method. *Ann Intern Med* 1990;113:9-13.
- 17 Hull R, Hirsh J, Sackett DL, Taylor DW, Carter C, Turpie AGG, et al. Clinical validity of a negative venogram in patients with clinically suspected venous thrombosis. *Circulation* 1981;64:622-5.

Harm minimisation for drug misusers

When second best may be best first

A quiet revolution is taking place in British responses to drug misuse. Harm minimisation is the new buzz word and refers to the component of care that makes reducing the harm that comes from drug use its main objective. Combating drug use then becomes the means to the end rather than the end itself.

At its centre the debate is about goals: working towards either stable abstinence or the less ambitious goal of reducing the harm from continued drug use in the belief that this is more achievable.¹ Essentially, harm minimisation is the triumph of pragmatism over purism: the acceptance that second best may be best first.

Elsewhere in medical practice harm minimisation is regarded as good secondary preventive health care. If cigarette smokers cannot give up they are advised to go for second best by switching to filter cigarettes or low tar varieties. Diabetic patients are advised on dietary and drug regimens but are also advised about self monitoring and emergency management in the event of loss of glycaemic control. People are regularly advised to drink moderately to avoid the health hazards associated with heavy alcohol consumption.

Even in drug misuse the concept is not new: 20 years ago some of the new drug clinics and day centres provided not

only needles and syringes but also instructions on injecting technique^{2 3} and special on site fixing rooms.⁴ In 1984 the *Prevention Report* made it clear that the goal of reducing drug use must exist alongside the goal of reducing the harm of continued drug use.⁵ Since then various British reports have recognised that some drug use will inevitably exist.^{1 6} They have legitimised efforts to reduce the harm of continued drug use while still endorsing efforts to counter drug use itself. The real issue becomes the balance between these strategies.

HIV has given harm minimisation a new prominence, and attempts to reduce sharing of syringes and unprotected sex have become key elements of this work. Harm minimisation is now being extended beyond HIV infection to overdose and hepatitis—as pioneered in Italy and the Netherlands.⁷⁻⁹ Descriptions of harm minimisation now appear in the Department of Health's new guidelines on managing drug misuse.¹⁰

When it comes to drug misusers the new guidelines from the Department of Health clarify the responsibilities for all doctors and the legitimacy and importance of strategies to minimise harm.¹⁰ Drug misusers have a right to health care regardless of doctors' moral indignation or prejudices. Abstinence (at least in the short term) is not the only