

inflation is associated with more severe ischaemia than the second or subsequent inflations. Deutsch *et al* have shown that the first inflation causes more pain, ST segment change, lactate production, and reactive hyperaemia than the second inflation.⁸ This study used a balloon inflation for 90 seconds, which on the basis of animal studies is too short to trigger preconditioning. A more likely explanation for the protection is the opening of collateral vessels in response to the first inflation. This change in collateral support to the ischaemic area between first and subsequent balloon inflations has been visualised directly in another preconditioning study by injecting contrast media into the right and left coronary arteries.⁹ A further difficulty with the use of angioplasty to investigate preconditioning results from the possible ischaemia caused by crossing a tight coronary artery stenosis with a wire and then the deflated balloon, which means that recordings made during the first balloon inflation may already come from preconditioned myocardium. In summary, the evidence for preconditioning during percutaneous transluminal coronary angioplasty is unconvincing and this model has several drawbacks.

In intermittent cross clamp fibrillation the aorta is clamped above the coronary ostia to isolate the coronary circulation, the heart is fibrillated, and the lower end of the bypass conduit is anastomosed to the coronary artery distal to the obstruction. The aortic clamp is then removed and the heart reperfused while the upper end of the bypass conduit is joined to the aorta. This procedure is repeated for each graft. We have recently reported that the fall in myocardial ATP concentration during the standard duration of cross clamp fibrillation can be dramatically reduced if it is preceded by two three minute periods of ischaemia.¹⁰ This pattern of metabolic protection in humans is analogous to the change in myocardial ATP observed in the original preconditioning studies by Murry *et al*.¹ As changes in coronary collateral flow do not complicate the interpretation of these results this probably represents preconditioning in humans.

What use might be made of the cardioprotective effect of preconditioning? Early reperfusion is the most effective available treatment for acute myocardial infarction. Unfortunately, the benefits diminish if treatment is delayed. The recent finding that mortality can be reduced just by increasing the rate of infusion of tissue plasminogen activator¹¹ further underlines the importance of early reperfusion. The ability of preconditioning to delay the onset and slow the progress of myocardial necrosis would therefore increase the time available for effective reperfusion and further decrease deaths.

Similar considerations are likely to apply in patients with unstable angina and in those undergoing cardiopulmonary bypass or high risk coronary angioplasty. In addition, the preservation of explanted hearts before transplantation may be improved. In all these situations the ability of preconditioning to delay the progression of ischaemic myocardial damage would improve the outcome.

The discovery of ischaemic preconditioning has changed our perception of the pathophysiology of brief intermittent ischaemia. It is likely to enhance our understanding of how ischaemia ultimately leads to the death of myocardial cells. But, most important of all, it has focused attention on the myocardium's ability to protect itself. The momentum of current research suggests that the exact mechanisms underlying this powerful endogenous form of myocardial protection will soon become clearer. It should then be possible to induce preconditioning pharmacologically to protect patients with ischaemic heart disease.

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GP facilitators and HIV infection

With increasing numbers of HIV positive patients on their lists GPs could use some help

"the greatest misery of sickness is solitude; when the infectiousness of the disease deters them who should assist from coming."¹

So wrote John Donne in 1627 when he thought he was dying. Although the infectious diseases of his time were bubonic plague, leprosy, and syphilis rather than AIDS, the hysteria and misinformation surrounding them are all too familiar.

Some 20 000 people in Britain are known to be HIV positive, with many others unaware of their HIV status. On average every general practitioner may therefore expect to

have one HIV positive patient on his or her list. Although most HIV positive patients currently live in large cities (particularly Edinburgh and London), as more people become infected more practices will have such patients on their lists; already many patients move out of the cities back to their families after AIDS has been diagnosed.

As the rate of HIV infection increases general practitioners will encounter more HIV related illness, and in patients not previously thought of as being at risk—for example, heterosexual adolescents. Although the number of cases of heterosexually acquired HIV infection is low in Britain, it is

increasing rapidly and heterosexual transmission is now the commonest form of spread in Scotland (and in the rest of Europe outside Britain).²

Until now HIV disease has generally been treated in hospitals and many patients have been reluctant to consult their general practitioner. The reasons for this have included fear of hostility and rejection, lack of confidentiality, and lack of knowledge. General practitioners have similar fears and anxieties about HIV and AIDS to those of the public and also feel insecure with a disease that is new and ever changing. Indeed, several surveys have shown a "lack of confidence in dealing with the issues surrounding HIV"³ and a "lack of knowledge" of the topic among general practitioners and trainees.⁴ But hospitals will be unable to cope as more people become infected, and more care for HIV disease will have to be provided in the community.

Faced with a disease that is relatively new, with unfamiliar and complex treatment regimens that alter frequently, and with a client group who may be better informed than they are, general practitioners might naturally feel threatened or confused. This is where a facilitator may help. A facilitator who is also a practising general practitioner is ideally placed to understand the specific problems of general practice. General practitioners encountering for the first time an HIV positive patient or one with AIDS may be unaware of the resources available in the community and from where and from whom to obtain help. This is especially important in the community care of terminal disease. Liaison between all groups working

with HIV infection, both statutory and non-statutory, is essential,⁵ and the facilitator has a role in developing links between these agencies.⁶ Another important objective of a facilitator should be to increase awareness of HIV among general practitioners and to promote education, so that as the numbers of affected patients rise general practitioners will be well prepared for their role in caring for them.

Although many general practitioners confidently manage patients with HIV infection, much remains to be done in the community. In places where the number of infected patients is low, HIV is understandably accorded a low priority: none the less, we should "trouble shoot" now rather than "fire fight" later. As Donne concluded in 1627, "the physician who dares scarce come . . . it is an outlawry, an excommunication upon the patient." Nearly 400 years separates these sentiments and the modern day, but they are still relevant and just as important.

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Fundholding: from solution to problem

Rigorous evaluation should precede any further extension of the scheme

General practice fundholding has become a policy problem for the NHS. Hailed as a success only a few months into its operation,¹ it now seems dogged by limited advantages, high costs, and unintended consequences.

So far the only demonstrable advantage to fundholders has been a reduction in prescribing costs.^{2,3} This is an eccentric achievement in a country whose prescribing costs historically have been among the lowest in western Europe.⁴ It tells us nothing about either the quality of care, which may decline as costs are cut, or the long term economic costs of short term savings on prescribing, which may be appreciable.⁵

The economic costs of fundholding are considerable and include both open costs (such as management fees, subsidies for computerisation, and the administrative costs of billing and reviewing contracts) and hidden costs (such as costs for staff in family health services authorities, hospitals, and the Audit Commission). The political costs may be equally important given the damage done to equity by "fast tracking," the growing doubts about the value of fundholding, and the government's persistent failure to devolve responsibility for underprovision of health services to purchasers.

Why has fundholding become so problematic? Firstly, fundholders usually cannot act as ruthless purchasers. Not only is there a contradiction between advocacy on behalf of patients and rationing of resources but local providers may not always be influenced by fundholders' interests and the choice of provider may be limited or non-existent.⁶ On the contrary, activity by provider units can create overspending for fundholders, a problem that may get worse as fundholders buy more services and as capitation based funding is intro-

duced. Secondly, fundholders are as much a threat as an opportunity for local health policy. Fundholders' decisions about placing resources are primarily budget led because the pressure to avoid overspending is so great. Overspent fundholders may simply lack the money to adhere to wider health policies.

How did this happen? Fundholding has developed as an ideological construct, not a scientific hypothesis. Fundholding is an incentive evolved from Bosanquet and Leese's microeconomic model of development in general practice⁷ and has been promoted by "ignorant experts" (in Alan Maynard's words) but never tested in pilot studies despite authoritative advice.⁸ Designed as a political solution to kick start the market, fundholding has become an end, not a means to an end.⁹

This idealisation of an untested economic mechanism meets the needs of some general practitioners: it addresses the division in British medicine between generalists and specialists¹⁰ by attempting to invert the power relationship while also touching on the omniscience beneath the surface of generalism. Fundholding also creates a managerial career structure within general practice in parallel with the vocational training structure, the local medical committee career path, and the hierarchy of academic practice. In a profession needing modernisation but locked within the egalitarianism of the independent contractor status, fundholding may represent a new opportunity for personal development.

Finally, no school of general practice sees itself as simply having a gatekeeper function, but fundholding as currently