imaging have only a limited place in the management of patients with suspected acute dissection.

In experienced hands the emergency investigation of choice is transoesophageal echocardiography.³ This can be performed rapidly in the cardiac care unit, and the patient remains accessible to medical and nursing staff.⁶ It provides detailed anatomical information about the morphology of a dissection—including the involvement of the coronary artery ostia—and it also gives functional information about other associated complications, such as aortic regurgitation and tamponade.⁶ Biplane and multiplane probes have reduced the blind spot in the upper ascending aorta and causes of artefact such as calcification of the aortic wall are now well recognised.⁷ Transoesophageal echocardiography will also show the presence of an intramural haematoma,⁸ persistent flow in the false lumen, and communications between the lumens, each of which is associated with a poor long term outcome.⁹

Immediate surgery is mandatory when the ascending thoracic aorta is involved in the dissection (Stanford classification type A¹⁰).² This entails excision of the intimal tear, reapproximation of the dissected aortic layers, and interposition of a Dacron graft.¹¹ When the aortic valve is involved, it is repaired by resuspending the commissures unless the patient has Marfan's syndrome, when the ascending aorta and the aortic valve are usually replaced with a composite graft.¹¹

Attitudes about performing bypass surgery for "coincidental" coronary disease at the same time as emergency aortic surgery are changing.¹² Coronary disease can be assessed fully only by coronary angiography, with its attendant risks and preoperative delay. In patients with no history of ischaemic heart disease many surgeons now operate without performing coronary angiography if transoesophageal echocardiography shows that the dissection does not affect the coronary ostia.

Aggressive control of the blood pressure and prolonged bed rest is the usual initial treatment for patients with uncomplicated dissection sparing the ascending aorta (Stanford type B)² as emergency surgery to the descending thoracic aorta carries a substantial mortality when compared with medical treatment. Surgery should be reconsidered if there is evidence of aortic rupture, proximal extension of the dissection, or ischaemic complications.¹³

The optimal management of patients with suspected dissection requires close liaison between district hospitals and cardiac surgical centres and use of local guidelines for

investigation that reflects the available skill. Patients with a low clinical likelihood of dissection who are in a stable cardiovascular state should undergo prompt local investigation with a nominated non-invasive technique. Unstable patients with a high likelihood of dissection should receive medical treatment and be transferred immediately to the surgical centre for both diagnostic imaging and management. If skill in transthoracic or transoesophageal echocardiography is available locally these procedures can be performed while transport is awaited, but doing so should not delay transfer. A videotaped record of the study should accompany the patient to the surgical centre, where repeat transoesophageal echocardiography can be performed in the anaesthetic room if necessary. This approach minimises delay, an essential step in lowering the mortality of acute dissection.

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GPs, patients, and the distance between them

Planning would help to increase access

See p 100

The issue of coherent catchment areas for primary care is raised again by Murray and colleagues in this week's journal (p 100).¹ The problem can be viewed from two perspectives: the distance that the patient is from the doctor and the distance that the doctor is from the patient.

For most people the convenience of the nearest surgery is the main determinant of where they register.² Nevertheless, many go further afield, and historic factors may be important in their choice. When they move, those in urban areas often remain with the devil they know—"the desire to maintain a link with a doctor known to the family . . . appears to be stronger than any wish to minimise the distance travelled to the practice premises."³ The availability of a woman doctor,

and, for patients from ethnic minorities, doctors from their own cultural group, may also be important.

Since 1948, when a salaried component was added to the simple equation between capitation and income, there has been little evidence that doctors retain migrants or accept allcomers for financial reasons. The majority of patients on most urban general practitioners' lists live within the practice's catchment area. This limits unnecessary travelling, facilitates care with local teams, and ensures that local populations do not lose out to more distant but mobile patients. But maintaining the right of patients to choose within limits seems a desirable option and one that services should be capable of accommodating.

The more acute problem is the distance that doctors are from their patients. Although most people report that distance is not an obstacle to access, for those most in need, including elderly and disabled people and those dependent on public transport, distance is often an important constraint. The negative effect of distance on the use of general practice services, both at the surgery and at home, has been repeatedly documented over the past 20 years.^{4,5} Knox has described how the changes in the locations of general practices in Aberdeen between 1950 and 1973 reinforced the geographical patterns of disadvantage.⁶ (Their amalgamation into group practices and their failure to devolve from city centre to peripheral housing estates made access more rather than less difficult.) Most general practitioners breathe a sigh of relief when new premises are built. Siting, however, is often a parochial affair, dependent on personal preference, the availability of a site, and the location of the list rather than on any wider considerations of geographical equity.

Under the present system some measure of social planning is at least conceivable. Patch and locality planning is beginning to emerge, adapting services to the needs of local populations and their providers.⁷ Whether this option to rationalise primary care services will remain is questionable. Perhaps we should be thankful for two things: firstly, that each patient does not have 87 different doctors as may happen in market driven systems that have abandoned gatekeepers, generalists, and continuity, instead favouring primary access to a multiplicity of specialists.⁸ And we should also be thankful that general practice is not yet beset by the problems of the education service, with basic provision limited by distance in the state sector and choice limited by ability to pay in the private sector. Equity, of either access or quality, does

not appear on the agenda of a government currently bent on establishing such a two tier service for primary care.⁹

Even forward thinking administrative authorities are floundering. There are neither adequate structures nor policies capable of dealing with coherent planning for primary care. In many places the destabilisation of hospital services has compounded the problem, and fewer managers and planners at district and regional levels are likely to add to the chaos.

Additional payments for deprivation have improved some unacceptably low medical incomes. But more substantial measures will be needed to prevent the widening divergence in access to and quality of primary care services, which is increasingly apparent both locally and regionally.¹⁰ Efficient distribution of resources is inextricably related to equity. Current policy does not address this, and, as Murray *et al* point out, the problems have not gone away.

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Clonality in Langerhans' cell histiocytosis

Women are more informative than men

Most human tumours are monoclonal, which suggests that they originate from a single cell. "Clonality" can be investigated by several techniques. In plasma a dominant monoclonal class of immunoglobulin suggests a neoplasm derived from a single altered plasma cell, which gives it a survival and growth advantage over non-neoplastic cells. In lymphoid malignancies, studies on rearrangements of genes coding for antigen receptors provide an excellent system of clonal markers, with consistent rearrangement of clonal immunoglobulin heavy chains (in B cell leukaemias and lymphomas)¹ and T cell receptor β chains (in T cell tumours). In other tumours careful cytogenetic and molecular analysis has shown consistent reciprocal chromosomal translocations and partial and complete chromosomal deletions or duplications which reflect tumour clonality as well as indicating stretches of altered sequences of DNA that may have a role in the initial neoplastic change. Cytogenetic and DNA analysis has not yet, however, provided clonal markers for many solid tumours.²

An alternative approach, applicable only to females, is to take advantage of the phenomenon of "X-inactivation" (X inactivation mosaicism).^{3,4} Early in the development of female embryos one X chromosome of each pair is randomly inactivated in each somatic cell. Some enzymes occur in two forms (isoenzymes)—if they are encoded by the X chromosome one form but not the other is represented in each cell.

The result of clonal expansion of a tumour cell reflects this event—only one form of the isoenzyme is expressed, as shown by Fialkow by using isoenzymes of glucose-6-phosphate dehydrogenase.³ These X inactivation studies have now been extended by molecular techniques, including polymerase chain reaction,⁵ and examination of very small samples of tissue is now possible.

Applying this approach, Willman and colleagues have clearly shown the clonal nature of Langerhans' cell histiocytosis (histiocytosis X).⁶ The disorder, first described over 100 years ago,⁷ had previously been known as Hand-Schüller-Christian disease, eosinophilic granuloma of bone, Letterer-Siwe disease, and self healing reticulocytosis. Abnormal Langerhans-like cells are pathognomonic of the condition, and they and their normal counterpart (the Langerhans histiocyte, which presents antigen) are rare in expressing the CD1a antigen. The clinical presentation and course of Langerhans cell histiocytosis vary from a spontaneously regressing solitary lytic lesion of bone to an intermediate form with lesions in bone, skin, mucous membranes, liver, and spleen with variable organ dysfunction to a lethal leukaemia-like disorder most often seen in infants.⁸

This variation has led to much speculation about the nature of the condition. A viral cause has been suggested, but ultrastructural and serological studies yield negative results,^{9,10} and no consistent evidence for an immune