by dilating quite heavily calcified valves in patients who are unfit for surgery.[20] As in aortic stenosis, it is important that patients who would be better treated with surgery are not subjected to valvuloplasty simply because of age if they are fit for surgery in other respects. Echocardiography is the best technique for assessing the suitability of the valve for dilatation.[18] Although transthoracic imaging is usually satisfactory, the transoesophageal approach provides better detail of the valve, subvalvar apparatus, extent of mitral regurgitation, and any intra-atrial clot[21] (a contraindication to the procedure).

Balloon mitral valvuloplasty usually converts severe mitral stenosis to a mild to moderate narrowing, with at least a doubling of the valve's area and significant improvement in the patient's clinical state. Unlike in aortic stenosis, these results are well maintained.[22,23] In experienced hands the mortality is less than 1%—considerably less than that of mitral valve replacement, although a fairer comparison would be with closed mitral valvotomy, which probably has a similar risk.[24] Important complications of balloon valvuloplasty include cardiac perforation leading to tamponade, embolic stroke due to displacement of clot from the left atrium, and damage to the valve leading to severe regurgitation. These complications are more likely with inexperienced operators.[25] Traditionally, surgical treatment of mitral stenosis has been withheld until symptoms become severe because of the risks and inconvenience associated with it. Now that the mitral valve can be dilated with excellent relief of symptoms, a mortality of 1%, and only a few days in hospital it is reasonable to consider the procedure in patients with mild symptoms in the hope that early intervention may postpone the onset of atrial fibrillation and pulmonary vascular disease and allow the patient to have near normal exercise tolerance.

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Reaccrediting general practice

GMSC, RCGP, and regional advisers in general practice should get together

In a survey earlier this year of general practitioners working in the NHS nearly two thirds disagreed with the statement that "Once a GP has acquired a basic level of competence no further form of reappraisal is necessary during the rest of his/her active professional life."[1] Given that no professions currently reaccredit their members, here is evidence that most general practitioners are forward looking and have the interests of their patients in mind.

Why should the profession with one of the longest periods of professional training now be initiating reaccreditation? The sheer quantity of new knowledge and the pace of change demand unprecedented intellectual and emotional commitment from medical generalists.

Reaccreditation is mainly for patients. Like the original licence to practise, it should be an assurance that education has been effective, competencies have been acquired, and patients can consult with confidence. It ought also to give something to the profession, both individually and collectively. For the individual doctor the process should be stimulating and educational, while for the profession there should be corporate pride in a job well done.

There are three ways in which reaccreditation could be done in general practice, all with advantages and disadvantages. These are by re-examination, a requirement to attend educational sessions, and assessment of performance in the practice itself.[2] An examination is the easiest and cheapest option, and there will be pressure for it. In British general practice this is likely to be the examination for membership of the Royal College of General Practitioners (MRCGP) or a variation of it. This may not, however, be the most popular proposal, and there are problems. Firstly, the MRCGP examination has been designed to assess vocational training, and though three quarters of all trainees now take it (examination board of Royal College of General Practitioners, personal communication, 1992), it is not designed as a test of competence for experienced general practitioners. Although there is now a debate about "qualifications and quality of care"[3] and a defence body offers discounts to doctors with the MRCGP,[4] it is not an ideal examination for reaccreditation.

Attendance at courses is an easy option and the approach favoured by the Royal College of Obstetricians and Gynaecologists.[5] Given that attendance at courses is already

included in the 1990 general practitioner contract adopting it for accreditation smacks of double counting, and there is little evidence that it is associated with better performance. Selecting and monitoring educational events and monitoring attendance—let alone how much learning actually goes on—would be difficult.

The third approach is to measure general practitioners' performance in their practice. Difficult and expensive, it requires national agreements on standards of performance, objective measurements, and visiting teams. It is, however, the one method that is already operating in general practice—through visits to approve trainers and to accredit members of the Royal College of General Practitioners as fellows. The survey earlier this year found that more than two fifths of general practitioners reported that their practice participated in training and so were being assessed.[1]

The survey also found that general practitioners have a preference regarding how they might be reaccredited: they would clearly prefer peer reviewed practice visits with external visitors.[1] Their responses were also analysed according to their degree of involvement in training—from no involvement, through training provided in the practice, to providing training personally. A clear gradient existed: the greater the general practitioner's participation in training the more likely he or she was to believe that reaccreditation was necessary. Trainers, who are reaccredited for teaching at least every five years and in some regions every three, value reaccreditation most.

General practitioners have given their representatives an effective mandate to put reaccreditation on their national agenda. Many groups, such as patients and managers, may now try to get in on the act. They would, however, be wise to stand back: this is a delicate time when the first shoots of a new growth could easily be damaged. General practice has taken the first step: it should now be encouraged and allowed to work out its own solution, probably by peer review. It must, for example, construct a sensitive, educational, and appropriate system for those who have problems in being reaccredited.

Three groups have a stake in this: the General Medical Services Committee, which organised the survey; the Royal College of General Practitioners, which developed vocational training[6] and higher professional education in general practice[7] and published the most advanced system of reaccreditation in general practice, *Fellowship by Assessment*[8]; and the university appointed regional advisers in general practice, who are already reaccrediting about two in five British general practices.[1]

In the mid-1970s general practice needed to invent a new organisation, the Joint Committee on Postgraduate Training for General Practice, to handle vocational training. Could the three national groups with an interest in reaccreditation now get together, share their skills, and take the process forward?

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Osteoporosis in men

Hormonal protection eventually fades and prevention is key

Clinical osteoporosis may be defined as thinning of the bone—with normal mineralisation—severe enough to lead to fracture with minimal trauma. The incidence of vertebral fractures is up to six times less common in men than women,[1] but osteoporosis and its problems are on the increase in both sexes. Men are relatively protected for several reasons.

Firstly, age for age their bone density is higher. Their bones are bigger; and they continue to increase in size owing to growth of periosteal bone.[2] Androgens have a direct effect on bone cells, probably in conjunction with insulin related growth factor I, on chondrocytes of the growth plate, and on osteoblasts.[3] The anabolic effect of testosterone on muscles probably increases the stresses on bones during exercise, and this in turn would be expected to lead to more deposition of bone in men.

Secondly, spermatogenesis is a production line process that does not progressively deplete the stock of germ cells; normally therefore, both the structure and function of the seminiferous tubules, which are important for the function of Leydig cells, are maintained into quite advanced old age. The production of testosterone (and oestradiol) is well sustained in old men.[4] This hormone pattern contrasts with the single batch process of oogenesis in women, which means that once stocks are depleted production of oestradiol virtually ceases—with the consequent menopausal acceleration of bone loss.

Thirdly, and probably as an adverse consequence of high concentrations of testosterone on other body systems, men on average live about five years less than women, so there are fewer old men to develop cortical osteoporosis and so to sustain fractures of the neck of the femur. The pattern of osteoporosis in men is different from that in women, with less loss of trabeculae and thinning of trabeculae being more prominent.[5,6]

Except in extreme old age, men with osteoporosis generally present with pain due to vertebral crush fractures. Several studies have shown that identifiable risk factors play a more prominent part than in women. These include predisposing illnesses—any condition leading to treatment with steroids or excess natural secretion of these hormones, hypogonadism (with or without hyperprolactinaemia), gastrointestinal surgery, defective synthesis of type I collagen leading to osteogenesis imperfecta tarda, and, rarely, systemic mastocytosis.[6-11] A clinician in doubt about the diagnosis should measure the serum concentrations of luteinising hormone, follicle stimulating hormone, testosterone, and prolactin and do screening tests for Cushing's syndrome such as measurements of the 24 hour urinary free cortisol excretion and serum cortisol concentration after suppression the previous night with a single dose of 1·5 mg dexamethasone. Behavioural risk factors include heavy cigarette smoking, excessive drinking, a low calcium diet (which may result from alactasia), and