Saturday 26 April 1997
BMJ

Modern treatments for internal haemorrhoids

Scalpel surgery is now rarely needed

Almost everyone suffers from haemorrhoids at some time in their lives. The symptoms include bleeding, prolapsing tissue, fullness after defecation, and pain. Bleeding can mimic or mask the diagnosis of cancer and must be thoroughly evaluated. In most cases, however, swift, simple, and effective treatment can be given in an outpatient clinic or a health centre.[1-4] The key to understanding the feasibility of outpatient treatment is that there are no sensory nerve fibres above the dentate (pectinate) line in the anus, which is at the squamomucosal junction. Internal haemorrhoids arise above this line, so they can be treated without an anaesthetic. External haemorrhoids develop below the dentate line and are exquisitely sensitive. Little preparation is needed for the treatment of internal haemorrhoids, but an enema will make them easier to see as well as making the procedure more aesthetically acceptable.

Haemorrhoids are graded by the degree of prolapse, and this grading determines the most appropriate methods of treatment. First degree haemorrhoids are merely visible vessels, second degree lesions prolapse with defecation but return spontaneously, third degree lesions prolapse and require manual replacement, and fourth degree lesions remain prolapsed out of the anal canal despite attempts to reduce them.

The treatment choices for internal haemorrhoids include infrared coagulation, radiofrequency coagulation, direct current coagulation, rubber band ligation, sclerotherapy, cryosurgery, scalpel surgery, and laser surgery.[5] Scalpel surgery is generally reserved for advanced fourth degree haemorrhoids and is most often done on inpatients. Laser surgery is said to be less painful, but this has proved difficult to verify.[6] Sclerotherapy is usually indicated only in first and second degree lesions,[7] and in the United States it is now little used because of the frequency and severity of complications and the technical difficulties of proper placement of the sclerosant. A recent report in the *BMJ* described three patients who became permanently impotent after sclerotherapy for their haemorrhoids.[8] Cryotherapy is also little used because of the profuse and prolonged discharge, the complications such as excessive sloughing and sphincter injury, and the poor results.[7]

The least expensive and possibly the most widely used equipment is a rubber band ligator. This is suitable for first to third degree haemorrhoids. The bands are easy to apply, but the drawback is that two people are needed, one to hold the anoscope and the other to apply the bands. The treatment can cause severe pain if the bands are placed too low, and there is a small risk of perineal sepsis, which can, very rarely, be fatal.[9] Sepsis is a medical emergency signalled by fever, pain, swelling, and the inability to pass urine.

The infrared coagulator is gaining rapid acceptance for outpatient treatment of internal first and second degree haemorrhoids and some third degree ones. A special bulb provides high intensity infrared light that coagulates vessels and tethers the mucosa to subcutaneous tissues. The flat tip probe measures 6 mm in diameter and is applied for 1.5-2 seconds three to eight times to a localised area of haemorrhoids. Generally only one section of the haemorrhoids is treated per visit. Patients generally have two to four areas that need treatment and so have to return several times at monthly intervals until all have been controlled. Infrared coagulation is quick (10-15 minutes a visit), effective, and painless, and patients can return to work immediately or the next day. Eighty per cent of patients treated by this method are reported to be free of symptoms at three months.[10] In a meta-analysis comparing infrared coagulation, rubber band ligation, and injection sclerotherapy, infrared coagulation came out best.[11]

The radiofrequency coagulation unit uses a disposable probe with an electrical current flowing between two flat electrodes (positive and negative) aligned at the tip. Activating the unit for two seconds in three or four areas of the same haemorrhoid complex effectively coagulates the vessels. Although the manufacturer claims that all haemorrhoids present can be treated in a single session, I believe it is preferable to treat one area at a time to avoid excessive pain and bleeding.[12]

The direct current units use a probe with two sharp points as electrodes. They are promoted for use in all grades of haemorrhoids but seem to have two drawbacks. Firstly, each treatment takes eight to 12 minutes of probe contact. This is considerably longer than the six to 10 seconds required for infrared and radiofrequency units.[4 13] Secondly, the probes can penetrate deeply unless the operator is careful to stabilise them during treatment.

Whatever treatment is used, postoperative management is the same. The goal is to keep patients' stools soft by giving a high bulk diet and lots of fluids. Non-steroidal anti-inflammatory drugs will usually control any discomfort. Sitz baths may help in rare cases where needed. Suppositories are rarely necessary.

The clear advantages of the modern methods for outpatient treatment of internal haemorrhoids are that they are quick and relatively painless. Patients lose little if any time from work, the complications are minor, and the cure rates are high.[14 15] Pain is generally attributable to placing the treatment probes too far distally.

Patients may have a little spotting of blood for a few days and slightly more bleeding may occur after 10-14 days, when the eschar sloughs, but major haemorrhages do not occur as in the old style surgical approaches. No episodes of perineal sepsis, death, or impotence have been reported with the newer methods. The failure rates are reported to be 10-20%, but all that is needed is further treatment. A complication seen in 1-2% of patients (but not reported in the literature) is external haemorrhoidal thrombosis, usually associated with treatment of too extensive an area of internal haemorrhoids at one visit.

Formal surgical intervention is still occasionally necessary, but patients dislike it because of the associated severe pain and morbidity. Modern treatment methods may be mastered by doctors working in primary care, and they provide a prompt effective treatment in most cases.

John L Pfenninger *Director*

The National Procedures Institute,
Midland, Michigan 48640, USA
jpfenninger@msms.org

1 Zainea G, Randall G, Norman DA, Pfenninger JL. Office treatment of hemorrhoids. In: Pfenninger JL, Fowler GC, eds. *Procedures for primary care physicians*. St Louis: Mosby, 1994:929-53.
2 Pfenninger JL, Surrell J. Nonsurgical treatment options for internal hemorrhoids. *Am Fam Physician* 1995;52:821-37.
3 Bleday R, Pena JP, Rothenberger DA, Goldberg SM, Buls JG. Symptomatic hemorrhoids: current incidence and complications of operative therapy. *Dis Colon Rectum* 1992;35:477-81.
4 Yang R, Migikovsky B, Peicher J, Laine L. Randomized, prospective trial of direct current versus bipolar electrocoagulation for bleeding internal hemorrhoids. *Gastrointest Endosc* 1993;39:766-9.
5 Schussman LC, Lutz LJ. Outpatient management of hemorrhoids. *Prim Care* 1986;13:527-41.
6 Smith LE. Hemorrhoidectomy with lasers and other contemporary modalities. *Surg Clin North Am* 1992;3:665-79.
7 Liebach JR, Cerda JJ. Hemorrhoids: modern treatment methods. *Hosp Med* 1991;August:53-68.
8 Bullock N. Impotence after sclerotherapy of haemorrhoids:case report. *BMJ* 1997;314:419.
9 Russell TR, Donohue JH. Hemorrhoidal banding: a warning. *Dis Colon Rectum* 1985;28:291-3.
10 Walker AJ, Leicester RJ, Nicholls RJ, Mann CV. A prospective study of infrared coagulation, injection and rubber band ligation in the treatment of hemorrhoids. *Int J Colorectal Dis* 1990;5:113.
11 Johanson JF, Rimm A. Optimal nonsurgical treatment of hemorrhoids: a comparative analysis of infrared coagulation, rubber band ligation, and injection sclerotherapy. *Am J Gastroenterol* 1992;87:1600-6.
12 Dennison A, Whiston RJ, Rooney S, Chadderton RD, Wherry DC, Morris DL. A randomized comparison of infrared photocoagulation with bipolar diathermy for the outpatient treatment of hemorrhoids. *Dis Colon Rectum* 1990;33:32-4.
13 Norman DA, Newton R, Nicholas GV. Direct current electrotherapy of internal hemorrhoids: an effective, safe, and painless outpatient approach. *Am J Gastroenterol* 1989;84:482-7.
14 Templeton JL, Spence RJ, Kennedy TL, *et al*. Comparison of infrared coagulation and rubber band ligation for first and second degree hemorrhoids. *Am J Gastroenterol* 1989;84:475-81.
15 Zinberg SS, Stern DH, Furman DS, Wittles JM. A personal experience in comparing three nonoperative techniques for treating internal hemorrhoids. *Am J Gastroenterol* 1989;84:488-92.

The future of locality commissioning

Making it universal raises some difficult issues

GP fundholding was introduced by the Conservative government to involve general practitioners in shaping local health services[1]: individual practices acted as agents for their patients, using their own budgets to bring about quality and efficiency improvements in hospital and community health services. A range of alternative, unofficial schemes has emerged at local level (non-fundholding, general practitioner led commissioning, and locality commissioning[2-4]) led by non-fundholders who want to help shape local health services but are sceptical about holding an individual practice budget. These schemes have received less attention than fundholding and its derivatives,[5 6] but this issue of the *BMJ* includes a rare attempt to evaluate locality commissioning in Avon, where there seem to be modest benefits at a much lower cost than with fundholding (p 1264).[7]

See p 1246

No single model exists, but a locality commissioning group is generally a collaboration between practices which together cover a geographical population. The group aims either to influence the local health authority's purchasing or to work directly with local providers to agree changes which can then be incorporated in health authority contracts. The group does not normally take a delegated budget from the health authority, though some schemes work with indicative or "shadow" budgets. In most cases, lead general practitioners are paid a small amount for their time by the health authority, and health authority staff work to support the schemes.

Groups are free to choose which services they wish to change. Again, there is no single way in which change is engineered, but the size of some groups has enabled them to exert considerable influence over local providers.[2]

In 1995-6 the NHS Executive was still promoting fundholding and its variants as the only effective means of general practitioner involvement in purchasing.[5] Since then, locality commissioning has come in from the cold, principally because it appears to offer a lower cost, less divisive form of engagement for general practitioners than single practice fundholding. The main reservations relate to its effectiveness, especially in the face of resistant providers. Nonetheless, the Conservatives are now prepared to allow pilot schemes,[8] although it is not clear whether commissioning groups will be given management allowances like fundholders. The Liberal Democrats support the idea of all local practices becoming collective budget holders.[9] Labour's model is that all general practitioners in an area with a population of 50 000 to 150 000 would take part in a group which would be delegated a comprehensive budget by its local health authority (C Smith, speech 6 December 1996); fundholders would continue with their single practice elective budgets only with the consent of the locality group.

The distinctions between approaches based on fundholding and those developed in reaction to it are crumbling.[10] Whoever is in government after 1 May will have to identify which models work best in which circumstances and at least cost. For locality commissioning to secure its future, at least two difficult issues will have to be resolved: how to persuade practices in the same locality to work together where they would not otherwise choose to do so (some practices want to work with others but want to choose their collaborators; others do not want to take part at all); and how to give local groups the benefit of influence while persuading them to share the health authority's responsibility for managing the use of healthcare resources overall, particularly for emergency care; this is crucial when health authorities are struggling to remain within budget.[11]

Some form of general practitioner participation in commissioning does, however, seem to be here to stay. This in turn raises a further and linked question for the future: if the benefits of such collaborative commissioning are proved, will participation in commissioning become a requirement for all practices, so that some patients are not disadvantaged?

Nicholas Mays *Director of Health Services Research*

Policy Institute, King's Fund, London W1M 0AN

1 Secretaries of State for Health, Wales, Northern Ireland and Scotland. *Working for Patients*. London: HMSO, 1989 (Cm 555).
2 Black DG, Birchall AP, Trimble IMG. Non-fundholding in Nottingham. *BMJ* 1994;309:930-2.
3 Pickin C, Popay J. *Locality approaches in purchasing: a review of current practice in the NHS*. Salford: Public Health Research Resource Centre, University of Salford, 1994.
4 Graffy JP, Williams J. Purchasing for all: an alternative to fundholding. *BMJ* 1994;308:391-4.
5 NHS Executive. *Developing NHS purchasing and GP fundholding*. Leeds: NHSE, 1994 (EL(94)79).
6 Mays N, Goodwin N, Bevan G, Wyke S on behalf of Total Purchasing National Evaluation Team. *Total purchasing: a profile of national pilot projects*. London: King's Fund, 1997.
7 Hine CE, Bachmann MO. Locality commissioning in Avon: what does it offer? *BMJ* 1997;314:1246-50.
8 Secretaries of State for Health, Scotland and Wales. *Primary care:delivering the future*. London: Stationery Office, 1996 (Cm 3512).
9 Make the difference: the Liberal Democrat Party manifesto 1997. London: Liberal Democrat Party, 1997.
10 Mays N, Dixon J. *Purchaser plurality in UK health care: is a consensus emerging and is it the right one?* London: King's Fund Publishing, 1996.
11 Dixon J, Harrison £. Funding the NHS: a little local difficulty? *BMJ* 1997;314:216-9.

Is one arterial graft enough?

Total arterial revascularisation seems promising

The natural course of coronary revascularisation with saphenous vein grafts is well documented. Within 10 years half will have become blocked by a combination of thrombosis, intimal hyperplasia, and atherosclerosis.[1] Since the long term benefit largely depends on the grafts remaining patent, arterial grafts are being used increasingly in the hope that they will remain patent for longer and so give better long term results.

The concept of using arterial grafts is not new. Vineberg used the mammary artery for his pioneering procedure in the 1950s,[2] and reports of the left internal mammary artery being used as a direct coronary graft appeared in the late 1960s.[3] Histological and functional studies have shown that the internal mammary artery has biological properties that help it to resist thrombosis, intimal hyperplasia, and atherosclerosis,[4 5] suggesting that it may have better patency than saphenous vein. These hopes are borne out in practice. Left internal mammary artery grafts to the left anterior descending coronary artery have given consistently better patency rates than saphenous vein grafts.[6] Retrospective clinical reviews and registry data both show that this improved patency is associated with prolonged survival of patients, a reduction in late myocardial infarction and other non-fatal cardiac events, and a lower rate of reoperation.[7 8]

These superior results from single internal mammary artery grafts have prompted the use of bilateral grafts. Using both internal mammary arteries increases the complexity of the operation, but this has not increased operative mortality or morbidity.[9] Early retrospective comparisons of single and bilateral mammary artery grafts suggested improved long term survival with bilateral grafts,[10] but a later reanalysis of the data—with better follow up of patients and an independent statistical review—showed that the benefit was confined to a small reduction in the incidence of recurrent angina at 15 years.[11] A recent large prospective study showed no clinical benefit at a four year follow up.[12] Clearly, longer follow up is required before definitive conclusions can be drawn from these data.

Since most patients need three or more grafts, supplementary vein grafts—with their limited patency—will usually be needed even with bilateral mammary grafts. The alternative is to use additional arterial grafts with the aim of providing "complete arterial revascularisation." Most clinical experience has been with the right gastroepiploic, inferior epigastric, and radial arteries. In experienced hands all these conduits have been used without any increase in mortality and morbidity and with encouraging early patency rates.[13] No long term data are yet available on the performance of these grafts.

Ideally, the value of multiple arterial grafts would be clarified by a prospective randomised trial. Such a study would face enormous practical difficulties. Variables other than choice of conduit influence patency; these would have to be taken into account, and vast numbers of patients would have to be recruited. The combinations of arterial grafts and coronary arteries available to surgeons would be further complicated by the need for many of the decisions about the use of arterial grafts to be made intraoperatively. Detailed follow up of clinical status—and, ideally, of graft patency—would be necessary for at least 10 years since evidence from the Cleveland Clinic has already indicated that differences in long term

mortality and reoperation rates between cohorts of patients receiving single or multiple arterial grafts are likely to become apparent only after this time (B Lytle, personal communication).[14] Most observers believe that no such trial will ever be organised.

Nevertheless, the concept of "total arterial revascularisation" has a sound theoretical appeal, and there is already compelling evidence for using the left internal mammary artery to bypass the left anterior descending coronary artery. Clearly, if an unrealistically large prospective randomised trial would be needed to determine the value of complex arterial grafting other methods of analysis will have to be used to try to clarify this issue.

One way forward might be to establish a national registry with specific data fields about arterial grafts. At present, the only national perspective on arterial revascularisation comes from the United Kingdom Cardiac Surgical Register, which collects simple data on all cardiac operations performed in NHS hospitals. This has already shown that use of the internal mammary artery has grown in frequency from 20% of all coronary operations in 1985 to 86% in 1994. The growth in multiple arterial revascularisation has been slower. In 1994, 21 223 patients underwent coronary artery surgery with arterial grafts, of whom 6% received two arterial grafts and 2% received three or more.

The concept of long term follow up of a national cohort of patients has already been established by the United Kingdom Valve Registry, set up in 1986 at the Hammersmith Hospital, London. This register is sent details of all heart valves implanted in NHS hospitals. It maintains data on mortality through the Office of National Statistics and collects data on reoperation rates from individual units. Such a model could be applied to the outcome of coronary surgery, with specific reference to arterial grafts. This might not be an ideal solution, but it is probably the only way to collect data on large numbers of patients and define the place of complex arterial grafting in cardiac surgical practice. Registry data have provided the impetus for the increased use of a single mammary graft; before too long they may go some way to answering the question of whether one arterial graft is enough.

Malcolm Underwood *Clinical fellow*

Service de Chirurgie Cardiovasculaire et Thoracique, Universite Catholique de Louvain, avenue Hippocrate 10, B-1200 Bruxelles, Belgium

Graham Cooper *Consultant cardiothoracic surgeon*

Department of Cardiac Surgery, Northern General Hospital, Sheffield S5 7AU

Bruce Keogh *Consultant cardiothoracic surgeon*

Department of Cardiac Surgery, Queen Elizabeth Hospital, Birmingham B15 2TH

1 Fitzgibbon GM, Leach AJ, Kafka HP, Keon WJ. Coronary bypass graft fate: long-term angiographic study. *J Am Coll Cardiol* 1991;17:1075-80.
2 Vineberg AM. Treatment of coronary insufficiency by implantation of the internal mammary artery into the left ventricular myocardium. *J Thorac Surg* 1952;23:42-54.
3 Kolessov VI. Mammary artery-coronary artery anastomosis as a method of treatment for angina pectoris. *J Thorac Cardiovasc Surg* 1967;54:535-43.
4 Sims FH. Discontinuities in the internal elastic lamina: a comparison of coronary and internal mammary arteries. *Artery* 1985;13:127-43.
5 Yang Z, Stultz P, von Segesser L, Bauer E, Turina M, Luscher T. Different interactions between platelets with arterial and venous coronary graft vessels. *Lancet* 1991;337:939-43.
6 Lytle BW, Loop FD, Cosgrove DM, Ratliff NB, Easley K, Taylor PC. Long term (5-12 years) serial studies of internal mammary artery and saphenous vein coronary bypass grafts. *J Thorac Cardiovasc Surg* 1985;89:248-58.
7 Loop FD, Lytle BW, Cosgrove DM, Stewart RW, Goormastic M, Williams GW, *et al.* Influence of the internal mammary artery graft on 10 year survival and other cardiac events. *N Engl J Med* 1986;314:1-6.
8 Edwards FH, Clark RE, Schwartz M. Coronary artery bypass grafting: the Society of Thoracic Surgeons National Database experience. *Ann Thorac Surg* 1994;57:12-9.
9 Cosgrove DM, Lytle BW, Loop FD, Taylor PC, Stewart RW, Gill CC, *et al.* Does bilateral internal mammary artery grafting increase surgical risk? *J Thorac Cardiovasc Surg* 1988;95:850-6.
10 Fiore AC, Naunheim KS, Dean P, Kaiser GC, Pennington G, Willan VL, *et al.* Results of internal thoracic artery grafting over 15 years: single versus double grafts. *Ann Thorac Surg* 1990;49:202-9.
11 Naunheim KS, Barner HB, Fiore AC. Results of internal thoracic artery grafting over 15 years: single versus double grafts. *Ann Thorac Surg* 1992;53:716-8.
12 Morris JJ, Smith LR, Glower DG, Muhlbaier LH, Reves JG, Weschler AS, *et al.* Clinical evaluation of single versus multiple mammary artery bypass. *Circulation* 1990;82(suppl III):37-52.
13 Calafiore AM, Di Giammarco G. Complete revacsularisation with three or more arterial conduits. *Semin Thorac Cardiovasc Surg* 1996;8:15-23.
14 Cataldo G, Braga M, Pirotta N, Lavezzari M, Rovelli F, Marubini E. Factors influencing 1-year patency of coronary artery saphenous vein grafts. *Circulation* 1993;88(suppl II):93-8.

The private finance initiative

Undermines rational planning of health services

Among health service managers, healthcare professionals, and the public there is almost universal agreement that the private finance initiative has failed as an alternative source of capital funds for the NHS. The amount of public sector capital has reduced—by 15% in the past two years—but when the election was announced in March not one major privately financed hospital project had started. Most criticism has focused on this failure. In any case there is a belief that schemes under the private finance initiative can work only at a cost to the NHS (in terms of future payments for facilities) which will prove unacceptable. Hospital provision over 30 years is a high risk enterprise, and returns on private investment must reflect this risk.

See pp 1222, 1266

An article by Pollock *et al* in this issue (p 1266) raises two further problems with the private finance initiative.[1] The first is a lack of openness surrounding the whole process, resulting from commercial sensitivity. The second lies at the heart of the planning process for public services: the questionable assumptions that lie behind many hospital schemes in the pipeline. These criticisms are linked. The ability to assess the appropriateness of schemes depends on the opportunity to examine publicly the factors behind one choice rather than another.

Much of this criticism would have applied equally well to health authority decision making before the private finance initiative—though the initiative has not helped. The basis of decisions was seldom adequately

placed before the public, and the rationale behind these decisions was equally questionable. Subjecting large capital schemes to the private finance initiative adds to problems in two ways. Firstly, although schemes may initially go through the usual NHS consultation process, what finally emerges may be very different from the scheme originally agreed. Secondly, private consortia base their decisions on the viability of each individual scheme without reference to more fundamental health objectives. Even though health authorities are supposed to ensure these, it may be hard for them to do so.

When major changes to the provision of services are intended the usual process of NHS consultation applies. The responsible health authority issues a consultation document on the proposed scheme, laying out potential options for change. Theoretically, subsequent decisions must take account of the result of this consultation. In the past consultation was often regarded as a charade, irrespective of the method of capital financing. However, the process under the private finance initiative—producing outline business cases, assessing tenders, making judgments about the core parts of schemes—is shrouded in commercial sensitivities which do indeed make independent assessment difficult if not impossible.

At a late stage the scheme may change so much that the proposal should theoretically go back for further public consultation. Private consortia find it very difficult to take part in such an open ended process. At issue is the control of schemes as they go through the lengthy tendering process to full Treasury approval. How much change to a scheme is allowed before further consultation is necessary or, indeed, whether any further consultation is feasible if the private sector option is to work, is unanswered. But if a completely new site emerges, if the private sector insists on a change of use of facilities, or if more private facilities are added, all raise the issue of what was consulted on. Even the composition of consortia can change after the preferred contractor is agreed, and planning assumptions continually change. In this welter of complexity and uncertainty little wonder that accountability to the public comes a poor second.

Large schemes involving rationalising hospital sites must be set in the context of overall strategic planning of health services in the UK. Most health authority plans were based on assumptions about structure and levels of hospital activity similar to those outlined in current privately financed proposals. If these are wrong the fundamental problem does not lie with the introduction of the private finance initiative. However, the initiative makes matters worse.

Schemes are generated by individual trusts. It is not clear how effectively trusts take account of the interaction between their schemes and those of other trusts or with other parts of the health service. For example, are the plans for rationalisation at the Wellhouse Trust consistent with intentions for hospitals in other parts of north London? What assumptions are made about other health service provision—continuing care, family doctors, social services? Taking account of such interactions has always been a problem for planners. Health authorities are responsible for maintaining this overview but often struggle to keep pace with ever-changing privately financed schemes. It is doubtful whether they are capable of this task or able to resist the political pressure to achieve a successful privately financed scheme, practically at all costs. Moreover, the introduction of private finance brings different considerations. Private consortia will tend to err on the side of caution over the size of a facility, since from a commercial perspective excess demand is less of a problem than excess capacity, whereas lack of capacity may be the most serious problem from a public health viewpoint.

Although the problems of openness and the rationale behind health service decision making go beyond the question of how to raise capital—indeed, they are inherent in the 1991 framework of independent trusts—the private finance initiative has exposed them in a particularly acute way. Making private capital available for health service facilities is a commercial decision, not necessarily linked to benefit in terms of the overall system of healthcare delivery. The private finance initiative has accentuated an existing tendency to less open planning of services within a very narrow strategic framework, neither of which are good in the long term.

Seán Boyle *Fellow in health policy analysis*
King's Fund, London W1M 0AN

1 Pollock AM, Dunnigan M, Gaffney D, Macfarlane A, Majeed FA on behalf of the NHS Consultants' Association, Radical Statistics Health Group, and the NHS Support Federation. What happens when the private sector plans hospital services for the NHS: three case studies under the private finance initiative. *BMJ* 1997;314:1266-71.

Drug treatment for benign prostatic hyperplasia

Decreasing muscle tone within the prostate gland may be more effective than reducing the size of the prostate

Benign prostatic hyperplasia is a common condition in men.[1] Patients may present with filling symptoms (frequency, urgency, and urge incontinence) or voiding symptoms (hesitancy, poor urinary stream, straining, intermittent stream, and a feeling of incomplete bladder emptying), or both.[2] The aim of treatment is primarily to relieve symptoms. Until the early 1980s the only treatment options were surgery (usually with transurethral resection) or simply waiting to see whether natural resolution of symptoms occurred.[3] After the identification of adrenoceptors in the smooth muscle of the prostate gland in the 1970s, urologists began to consider the possibility of using selective α_1 adrenoceptor antagonists (α blockers) to relieve symptoms.[3] An alternative drug treatment became available in 1992 with the introduction of fin-

asteride, a 5α reductase inhibitor. Until recently, the relative effectiveness of these two types of drugs was uncertain, but further information has now become available.[4]

Symptoms of benign prostatic hypertrophy can begin as early as the third decade of life. They increase with age, until about a fifth of men aged 40-64 and two fifths of men aged 65-79 have urinary symptoms or a urinary flow rate below 15 ml/sec.[5] As many as half of men aged 60 have histological hypertrophy, rising to 88% in men aged 80.[1] The degree of discomfort and inconvenience from these symptoms may be measured by a formal symptom scoring scale such as that developed by the American Urological Association.[6] The total score represents the overall severity of symptoms (0-7 mild; 8-19 moderate; 20-35 severe), and the scale has been shown to be a useful means of quantifying symptoms and measuring changes over time.[7]

Selective α_1 adrenoceptor antagonists (such as indoramin, prazosin, terazosin, alfuzosin, tamsulosin, and doxazosin) work by relaxing smooth muscle in the bladder neck and prostate. Although titration of the dose is necessary, most patients respond to treatment within weeks.[8] Side effects include orthostatic hypertension, tiredness, dizziness, and headache.[8 9] A randomised trial showed a 32-44% improvement in symptoms in patients treated with terazosin compared with a 23% improvement in the control group. There is no evidence that α blockers reduce complication rates or the eventual need for surgery.

5α Reductase inhibitors such as finasteride block the conversion of testosterone to dihydrotestosterone —the form of testosterone found in the prostate gland, which is thought to be responsible for the development of benign prostatic hypertrophy. The drug, taken orally, reduces the size of the prostate and leads to an increase in peak urinary flow rate and a reduction in symptoms.[10 11] The improvement in symptoms takes place over six months. Side effects include decreased libido, problems with ejaculation, and impotence. There has also been some concern about treatment with finasteride leading to a halving of prostate specific antigen levels, which could possibly mask early prostate cancer.[12] There is no evidence that 5α reductase inhibitors reduce complication rates or the need for surgery.

Since 5α reductase inhibitors and α blockers work by different mechanisms it has not been possible to assess their relative efficacy or their potential to work synergistically. However, a recently published study compared finasteride, terazosin, and a placebo against each other and in combination.[4] The study screened 1686 men aged 45-80 for benign prostatic hypertrophy and recruited 1229 who fulfilled the criteria, which included an American Urological Association score of at least 8 and a peak urinary flow rate of no more than 15 ml/sec and no less than 4 ml/sec. After a four week run in period with placebo, patients were randomised by telephone to four groups with a mean age of 65, 80% of white race, a mean score of 16, a peak urinary flow rate of 10.5 ml/sec, and a mean prostatic volume of 36 cm^3. At one year, the placebo, finasteride, terazosin, and combination groups showed mean decreases in symptom score of 2.6, 3.2, 6.1, and 6.2 respectively; mean increases in peak urinary flow rates of 1.4, 1.6, 2.7, and 3.2 ml/sec respectively; and changes in prostatic volume of an increase of 0.5 cm^3, a decrease of 6.1 cm^3, an increase of 0.5 cm^3, and a decrease of 7.0 cm^3 respectively.

These results show that, in this group of men with moderate prostatic symptoms, terazosin was significantly better than both finasteride and placebo at relieving symptoms. Despite finasteride causing the prostate to shrink, the increase in peak urinary flow with this drug was only just better than with placebo and much less than with terazosin.

α Blockers remain the drug of first choice for the medical management of benign prostatic hypertrophy, whereas 5α reductase inhibitors do not seem to be as effective at relieving symptoms, although there may be a benefit when there is substantial prostatic enlargement.[13] For patients who have not developed complications (such as hydronephrosis, recurrent urinary tract infection, and chronic urinary retention), there is still a choice between drug or surgical treatment. The uncertainty about the long term outcomes of drug treatment and the effectiveness of surgery in providing rapid relief of symptoms offers the opportunity for involving patients in decisions about their management.[14]

Andrew Farmer *General practitioner and research fellow*

Health Services Research Unit,
Department of Public Health and Primary Care,
University of Oxford, Oxford OX2 6HE

Jeremy Noble *Consultant surgeon*

Department of Urology,
Oxford Radcliffe Hospital, Oxford OX3 7LT

1 Berry SJ, Coffey DS, Walsh PC, Ewing LL. The development of human benign prostatic hyperplasia with age. *J Urol* 1984;132:474-9.
2 Abrams P. New words for old: lower urinary tract symptoms for "prostatism." *BMJ* 1994;308:929-30.
3 Abrams PH, Shah PJR, Stone R, Choa RG. Bladder outflow obstruction treated with phenoxybenzamine. *Br J Urol* 1982;54:527-30.
4 Lepor H, Williford WO, Barry MJ, Brawer MK, Dixon CM, Gormley G, *et al.* The efficacy of terazosin, finasteride or both in benign prostatic hyperplasia. Veterans Affairs Cooperative Studies Benign Prostatic Hyperplasia Study Group. *N Engl J Med* 1996;335:533-9.
5 Garraway WM, Russell EBAW, Lee RJ, Collins GN, McKelvie GB, Hehir M, *et al.* Impact of previously unrecognised benign prostatic hyperplasia on the daily activities of middle aged and elderly men. *Br J Gen Pract* 1993;43:318-21.
6 Barry MJ, Fowler FJ, O'Leary MP, Bruskewitz RC, Holtegrewe HL, Mebust WK, *et al.* The American Urological Association symptom index for benign prostatic hyperplasia. The Measurement Committee of the American Urological Association. *J Urol* 1992;148:1549-57.
7 Lawrence K. Measurement properties of the AUA symptom score: a methodological clarification. *Br J Urol* 1996;77:175-80.
8 Chapple CR. Selective α1-adrenoceptor antagonists in benign prostatic hyperplasia: rationale and clinical experience. *Eur Urol* 1996;29:129-44.
9 Lepor H, Auerbach S, Puras-Baez A, Narayan P, Soloway M, Lowe F, *et al.* A randomized, placebo controlled multicenter study of the efficacy and safety of terazosin in the treatment of benign prostatic hyperplasia. *J Urol* 1992;148:1467-74.
10 Andersen JT, Ekman P, Wolf H, Beisland HO, Johansson JE, Kontturi M, *et al.* Can Finasteride reverse the progress of benign prostatic hyperplasia? A two-year placebo-controlled study. *Urology* 1995;46:631-7.
11 Gormley GJ, Stoner E, Bruskewitz RC, Imperato-McGinley J, Walsh PC, McConnell JD, *et al.* The effect of finasteride in men with benign prostatic hyperplasia. *N Engl J Med* 1992;327:1185-91.
12 Waldstreicher J, Tocker PF, Cheng J, Shown TE, Hampsey JP, Roy JB, *et al.* The effects of finasteride on serum prostatic specific antigen (PSA) in men with benign prostatic hyperplasia. *J Urol* 1995;153:136(s).
13 Boyle P, Gould AL, Roehrborn CG. Prostate volume predicts outcome of treatment of benign prostatic hyperplasia with finasteride. Meta-analysis of randomised clinical trials. *Urology* 1996;48:398-405.
14 Barry MJ, Fowler FJ, Mulley AG, Henderson JV, Wennberg JE. Patient reactions to a program designed to facilitate participation in treatment decisions for benign prostatic hyperplasia. *Med Care* 1995;33:771-82.