Advisory Committee's latest report shows that more career registrar posts have already been approved[5] than were envisaged in the "do nothing" projections in *Achieving a Balance*.

Preferentially increasing the staff grade while delaying or curtailing the expansion of consultant numbers will appeal to hard pressed managers seeking to provide adequate services with inadequate budgets. Indeed there may still be, as Dowie contends, "a substantial reservoir" of people qualified and willing to undertake such posts. Most junior doctors, however, undoubtedly still want to train as consultants.

Many of the report's recommendations should receive the wholehearted support of all doctors. They include the setting up of more preregistration posts in general practice, further research into the workloads of individual specialties, skill substitution, the costing of the use of hospital doctors' time, and better statistical support for planning medical manpower. We should not be content, however, to support a system in which many career grade hospital staff never achieve the status of fully trained specialists.

What is needed is a fundamental reappraisal of where services are delivered and by whom. This is particularly pertinent at present, given that we face a growing elderly population, a fall in the number of applicants for medical school, and the possibility of a pan-European shortfall of medical staff within the next 15 years.[6] Perhaps the time has come to blow the dust off the Short report[7] at last.

ANDREW CARNEY

Chairman, North West Thames Junior Doctors Committee, London WC1H 9SA

1 Dowie R. *Patterns of hospital medical staffing. Overview and specialty reports.* London: British Postgraduate Medical Federation, 1991.
2 Dowie R. *Patterns of hospital medical staffing. Interim report: junior doctors' hours.* London: British Postgraduate Medical Federation, 1990.
3 UK Health Departments, Joint Consultants Committee, and Chairmen of Regional Health Authorities. *Hospital medical staffing: achieving a balance—plan for action.* London, DHSS, 1987.
4 Department of Health. *Hospital medical staff—England and Wales.* London: DoH, Statistics and Management Information Division, 1991: national table 2 (30/09/90).
5 Joint Planning Advisory Committee. *Report for the years 1990/1.* London: DoH, 1991.
6 Saugmann P. Medical manpower in West Europe: towards a balance between supply and demand by the Year 2000. In: *Medical manpower in Europe. From surplus to deficit?* Copenhagen: Permanent Working Group of European Junior Hospital Doctors, 1991.
7 House of Commons Select Committee on Social Services. *Fourth report from the social services committee, session 1980/1.* London: HMSO, 1981. (Medical education, HC 31; Short report.)

Medical treatment for benign prostatic hyperplasia

Surgery still gives the best results

Transurethral prostatectomy remains the treatment of choice for symptomatic benign prostatic hyperplasia unless the prostate is large enough to warrant the retropubic approach. Recently attention has turned to alternative treatments for the condition: now we are in the middle of a surge of interest in possible medical treatments.

Interpreting the results of drug treatment requires an understanding of the very variable course of untreated prostatic obstruction: short term improvements may reflect the natural course of the disease rather than the effects of treatment. Patients should be carefully assessed before treatment is begun—not only to allow an accurate assessment of the drugs' subsequent effects but also to exclude prostatic malignancy, complications of prostatic obstruction, and other coexisting diseases.

The exclusion of prostatic cancer is currently provoking debate among urologists. Without a tissue diagnosis (automatically available with prostatectomy) focal carcinomas which would otherwise be diagnosed, will be missed in up to one in five patients. Most of these tumours will probably remain occult and not influence patients' survival. Rectal examination will miss most of them; measuring the serum concentration of prostate specific antigen before starting drug treatment[1] and performing transrectal ultrasonography of the prostate when the concentration of antigen is raised will provide the best diagnostic accuracy.

The first attempt at medical treatment was chemical castration with hormones, but this never achieved widespread acceptance because of its inevitable effects on sexual function. Interest in hormonal treatment has revived with the advent of a new class of drug that inhibits the conversion of testosterone to the more active dihydrotestosterone within the prostate by inhibiting the enzyme 5α-reductase (this avoids any effect on circulating androgens and hence the unwanted systemic effects associated with castration). The results of clinical trials are awaited.

Currently the main medical treatment for benign prostatic hyperplasia is α adrenergic blockade. Phenoxybenzamine, which blocks α_1 and α_2 adrenoceptors, has improved urinary flow and clinical symptoms in most clinical studies.[2,3] Some 30% of patients experience side effects,[2] which have been attributed to the blockage of presynaptic α_2 adrenoceptors. This is believed to interfere with the normal negative feedback control of the release of noradrenaline at the presynaptic adrenergic nerve terminal, thus resulting in a high circulating concentration of noradrenline. Phenoxybenzamine's side effects and evidence of its mutagenicity in bacterial and mouse cell cultures[4] have limited its use in the treatment of benign prostatic obstruction.

The experimental work of Caine and associates[5] and the incidental clinical observation reported in the *BMJ* in 1978 that urinary incontinence developed in women during antihypertensive treatment with prazosin[6] contributed to the recognition of the potential role of selective α_1 adrenoceptor blockade as treatment for diseases of the lower urinary tract. Subsequent studies by several groups of workers have shown the functional and ultrastructural pre-eminence of α_1 receptors over α_2 receptors within the stromal compartment of the prostate,[7] thereby providing a scientific basis for the use of specific α_1 blockade in non-surgical management of benign prostatic hyperplasia. Most studies of the specific α_1 antagonists have been conducted over relatively short periods of two to four weeks[8-11] and have shown objective improvements in mean and maximum urinary flow rates of 14-96% with subjective improvements in patients' symptom scores.

Longer term studies, which are of greater relevance to clinical practice, have investigated terazosin,[12] indoramin,[13] prazosin,[14] doxazosin,[15] and alfuzosin.[16] Only the study of alfuzosin reported by Ramsay *et al* failed to show any improvement in the outflow obstruction.[17] All the other studies showed that drug treatment significantly improved urinary flow rates, although the improvement in flow rates after at least three months' treatment was less than that previously reported in shorter term studies. For example, in a study of the longer term use of terazosin, Lepor and Knapp-Maloney reported an increase in maximum flow rate of 4·7

ml/s at six weeks, reducing to 1·6 ml/s at 72 weeks.[12] These observations suggest the development of tolerance. Alternatively a more likely explanation is that the α blockade results in a change in both voiding pressure and flow during voiding and that these effects on detrusor function take more than a month to occur, reducing the potential for any increase in flow rate. Supporting the suggestion that there is a gradual change in detrusor function is the observation that detrusor instability may take up to six months to improve after prostatectomy.

The best way of assessing clinical efficacy is currently the subject of considerable debate. Though the effect of any drug on symptoms is extremely relevant to clinical practice, symptom scores are difficult to quantify and validate. Conversely, estimations of flow rate provide objective data, allowing comparisons with the "normal" range of 25-35 ml/s for healthy young controls, but represent the consequence of the combined influences of detrusor contraction and bladder outflow resistance.

Surgery produces better results than α adrenergic blockade. A recent study comparing α_1 adrenoceptor blockade using prazosin with surgical prostatectomy found that the flow rate increased by 28% after α_1 blockade compared with 96% after surgery. Symptom scores improved by 45% after surgery and only 6% after prazosin.[18]

α Blockade with drugs clearly has a long way to go before it can rival surgery. Increasing the efficacy of adrenoceptor blockade by defining an α_1 subtype within the prostate that is distinct from the subtype located on blood vessels is one way forward. This would allow the administration of higher doses of α antagonist without increasing the risk of cardiovascular effects.[19] Alternatively, combinations of an α_1 antagonist and a 5α-reductase inhibitor both to relax the prostatic smooth musculature and to shrink the epithelial component of prostatic tissue are another possibility. In contemporary practice selective α_1 adrenergic blockade has an adjunctive role in treating symptomatic benign prostatic hyperplasia and provides an alternative to surgery for those patients who are unfit for surgery, do not want surgery, or those who are on waiting lists for surgery.

CHRISTOPHER CHAPPLE

Consultant Urological Surgeon,
Royal Hallamshire Hospital,
Sheffield S10 2JF

1 Catalona WJ, Smith DS, Ratliff TL. Measurement of prostate-specific antigen in serum as a screening test for prostate cancer. *N Engl J Med* 1991: **324**:1156-61.
2 Caine M, Perlberg S, Meretyk S. A placebo-controlled double-blind study of the effect of phenoxybenzamine in benign prostatic obstruction. *Br J Urol* 1978;**50**:551-4.
3 Abrams PH, Shah PJR, Stone AR, Choa RG. Bladder outflow obstruction treated with phenoxybenzamine. *Prog Clin Biol Res* 1981;**78**:269-75.
4 Phenoxybenzamine for symptoms of bladder neck obstruction. *Drug Ther Bull* 1983;**21**:15-6.
5 Caine M, Raz S, Zeigler M. Adrenergic and cholinergic receptors in the human prostate, prostatic capsule and bladder neck. *Br J Urol* 1975;**47**:193-202.
6 Thien T, Delaere KP, Debruyne FMJ, Koene RAP. Urinary incontinence caused by prazosin. *BMJ* 1978;i:622-3.
7 Chapple CR, Aubry ML, James S, Greengrass M, Burnstock G, Turner-Warwick RT, *et al.* Characterisation of human prostatic adrenoceptors using pharmacology receptor binding and localisation. *Br J Urol* 1989;**63**:487-96.
8 Hedlund H, Andersson KE, Ek A. Effects of prazosin in patients with benign prostatic obstruction. *J Urol* 1983;**130**:275-8.
9 Kirby RS, Coppinger SWC, Corcoran MO, Chapple CR, Flannigan M, Milroy EJG. Prazosin in the treatment of prostatic obstruction: a placebo-controlled study. *Br J Urol* 1987;**60**:136-42.
10 Stott MA, Abrams PH. Indoramin in the treatment of prostatic bladder outflow obstruction. *Br J Urol* 1991;**67**:499-501.
11 Kawabe K, Ueno A, Takimoto Y. Use of an α_1-blocker, YM617, in the treatment of benign prostatic hypertrophy. *J Urol* 1990;**144**:908-12.
12 Lepor H, Knapp-Maloney G. Outcome assessment of terazosin for benign prostatic hyperplasia (BPH): 18 month follow-up. *J Urol* 1991;**145**:263A.
13 Chow W, Hahn D, Sandhu D. Multicentre controlled trial of indoramin in the symptomatic relief of benign prostatic hypertrophy. *Br J Urol* 1990;**65**:36-8.
14 Chapple CR, Christmas TJ, Milroy EJG. A twelve-week placebo-controlled study of prazosin in the treatment of prostatic obstruction. *Urol Int* 1990;**45**(suppl 1):47-55.
15 Chapple CR, Carter P, Christmas TJ. A three month double-blind placebo controlled study of doxazosin as treatment for benign prostatic bladder outflow obstruction. *Neurourol Urod* **10**:308-9.
16 Jardin A, Bensadoun H, Delauche-Davallier MC, Attali P, BPH-ALF Group. Alfuzosin for treatment of benign prostatic hypertrophy. *Lancet* 1991;**337**:1457-61.
17 Ramsay JWA, Scott GI, Whitfield HN. A double-blind controlled trial of a new α-1 blocking drug in the treatment of bladder outflow obstruction. *Br J Urol* 1985;**57**:657-9.
18 Noble JG, Chapple CR, Milroy EJG. Long term selective α1 adrenoceptor blockade versus surgery in the treatment of benign prostatic hyperplasia. *Neurourol Urod* 1991;**10**:296-8.
19 Chapple CR, Burt R, Marshall I. α1 Adrenoceptor subtypes in the human prostate and inferior epigastric artery. *Neurourol Urod* 1991;**10**:306-8.

Behçet's disease

Retains most of its mysteries

In 1937 Hulusi Behçet, a Turkish dermatologist, described a disease associating uveitis with genital and oral aphthous lesions. Fifty years later Behçet's disease has expanded and is now recognised as a chronic, multisystem disorder with vasculitis as its underlying pathological process. It is an illness of the second to fourth decades, rarely seen in childhood and in patients over 50; a diagnosis of late onset Behçet's disease, even with consistent symptoms, will usually prove wrong.

The vascular manifestations are the main clinical features. Phlebothrombosis may affect all parts of the body, including dural sinus thrombosis (one third of the neurological lesions in our series).[1] Damage to arteries is observed in 5% to 35% of patients[2,3]—mostly aneurysms and arterial thrombosis. Arterial lesions carry a poor prognosis because the aneurysms often rupture, especially those in the pulmonary vessels.[4] Cardiac lesions include intraventricular thrombosis and thickening.[5] Vasculitis of the coronary arteries may lead to infarction or to aneurysm and usually requires surgical treatment.

The articular manifestations of Behçet's disease are rarely destructive. The association with ankylosing spondylytis (1-3%) seems coincidental. Renal damage is unusual and is often due to amyloidosis.[6,7]

For most patients Behçet's disease is not life threatening, and the outlook is dominated by disability due to blindness or neurological lesions—the identification of which is greatly facilitated by magnetic resonance imaging.[8]

Nevertheless the disease has many mysterious features. The first of these is its aetiology: infection may play a part ranging from herpes viruses to streptococci.[9,10] No case to case transmission has been described. The second mystery is the pathophysiological mechanism of Behçet's disease. The possibilities include autoimmune mechanisms, immune circulating complexes, and chemical agents. Among more recent suggestions have been heat shock proteins and hyperactivity of vascular endothelial cells.[10] None of these hypotheses has led to a clear understanding of the disease.

The epidemiology of Behçet's disease is also mysterious. An association with HLA-B5 antigens has been reported in certain parts of the world and in some familial clusters,[11] with a prevalence varying from 0·6/100 000 in Yorkshire,[12] and 7-8·5/100 000 in Japan to 370/100 000 in a recent survey in Turkey.[13] Behçet's disease seems more common between