Shopping around

In 1974-5 the NHS spent £100m on medical supplies in England alone. The shopping was done by districts, areas, regions, retained boards of governors, and the DHSS with little or no co-ordination; so that while large numbers of, say, electrocardiographs might be supplied to the NHS no one purchasing organisation handled enough to be able to compare the many varieties available. Production of medical equipment is just as fragmented and disorganised. There are 2000 firms supplying health products in Britain. Some are small workshops employing a handful of people, some are large firms with exclusively medical interests, some are subsidiaries of large corporations with interests well outside medicine, and some are foreign companies.

With this background it is reassuring that the DHSS recently set up a committee to “review existing policy for NHS procurement and to make recommendations as to the most cost-effective policy and its implementation, bearing in mind the need to strengthen the home market as a basis for exports.” Most of its recommendations¹ should help to reduce the present chaos. The committee has recommended a reduction in the variety of essentially similar equipment. There are, for example, over 53 models of ECG monitors being supplied. Clearly someone should be evaluating these so that the least effective may be withdrawn. The evaluation system should, the report states, consider the safety, technical performance, performance in clinical use, reliability, and cost of equipment, and it should be carried out in hospitals and academic departments. Such a policy should result in an increase in orders for the better pieces of equipment, so that their manufacturers would be able to increase production and efficiency and reduce costs. Already Britain is a net exporter of medical equipment (in 1975 there were £126.5m exports against £70m imports). More critical assessment of equipment by the British consumer to exclude the least satisfactory should increase exports further.

Another defect of the present system is the buying bonanza that tends to occur at the end of each financial year. The committee believes that this could be avoided by the use of a three-year rolling programme, the first year being firm, the second year provisional, and the third year planned, with annual revision. Many hospitals already have this sort of system in use. It is clearly an improvement on the alternative and apparently still common practice of haphazard ordering described in the report. However, the three-year cycle is not without problems. It requires constant updating; there should never be simply a question of Buggins’s turn, whereby an instrument is ordered because Buggins vaguely thought that it might be worth having three years ago. Revision of equipment lists should probably be undertaken by Cowgale divisions and the combined list assembled on behalf of the medical committee by a consultant who is prepared to take on the considerable task of deciding whether, for example, the division of surgery’s top priority should have precedence over the urgent request from the physicians.

Perhaps the most contentious recommendation is that authorities should be wary of the growth of district supplies organisations not directly accountable to the area supplies officer. This recommendation implies that the professional skill needed to evaluate equipment is to be found only among area staff, a belief for which there is no evidence. The area authorities need a monitoring and co-ordinating function, but the emphasis in the report on accountability seems wrong. Some current practices are exceptions to the criticisms in the report. The method used for purchasing disposable syringes is an example of what has already been achieved by central buying and storing against hospital orders, but such a system cannot be applied easily to nondisposable items bought in smaller numbers.

In many regions the regional scientific officer has become the officer concerned with the purchase of larger items of equipment, backed by a professional committee distributing a budget of perhaps £1.5m. Such a system may streamline purchasing but it has to be reconciled with financial control by the districts. While regions may be the best places for vetting larger items of equipment, areas and districts should

⁵ Freud, A. and Dann, S., Psychoanalytic Study of the Child, 1951, 6, 140.
⁶ Davis, K., American Journal of Sociology, 1947, 52, 432.
²⁶ Bell, R Q., Psychological Review, 1968, 75, 81.
Mitrail valve prolapse

The competence of the mitral valve depends on several features. Firstly, its structure must be normal. Secondly, there must be co-ordinated interaction of the mitral leaflets, annulus, chordae tendineae, papillary muscles, and left ventricular wall.3-5 Mitral regurgitation may result from any condition which deforms or immobilises these structures.

Our increasing knowledge of mitral valve function has focused attention on a fascinating syndrome, recently reviewed by Devereux et al.4 Various named Barlow's syndrome, the late systolic click syndrome, floppy mitral valve, and mitral leaflet prolapse syndrome, is not uncommon though readily missed; not overtly rheumatic in origin though this has been suggested. Occasionally familial, usually benign, in some patients it is associated with ill-explained complications such as chest pain, ventricular ectopic beats, angio- graphic abnormalities in left ventricular contractile pattern, and electrocardiographic abnormalities.

Gallevardin described the distinctive auscultatory features of mitral valve prolapse back in 1913, and he also observed the effects of posture on the timing of midsystolic clicks and the often associated late systolic murmurs. He mistakenly attributed these findings to pleuropericardial adhesions, and this was not refuted until 1961, when Reid suggested that the click was caused by abrupt tensing of the chordae and the late systolic murmur by late systolic mitral regurgitation.5 Barlow first showed angiographically that there was late systolic prolapse of the posterior mitral leaflet.6 In 1968 Engle drew attention to an abnormality of left ventricular contraction,7 and this has been extended8-9 to designate mitral valve prolapse as a segmental cardiomyopathy in which the altered shape of the ventricle in systole leads to slackening of the chordae and leaflets and consequent prolapse into the left atrium.10 The most recent theory suggests that the underlying abnormality is congenital or acquired dilatation of the mitral annulus11 or defective systolic contraction of the annulus, possibly associated with defective contraction of the basal portion of the left ventricle.9

After a period of confusion and over-diagnosis echocardiography has greatly aided the recognition and understanding of mitral prolapse.12-13 Prolapse of one or both leaflets into the left atrium can be directly visualised, and the click has been shown to occur at the moment of prolapse in late systole. Both the click and prolapse occur at a constant ventricular diameter, explaining the effect of posture and drugs on their timing.14-15 Prompt squatting or infusion of a pressor agent increases left ventricular volume and so reduces or abolishes prolapse, but they may induce chest pain.14 In one individual clicks may be multiple or absent and move their position in systole.15 The murmur may vary from absent to a honk or whoop which can be heard across the room.16 The auscultatory features are highly variable, and prolapse which is evident angiographically or echocardiographically may occasionally be clinically silent. Other inconstant clinical features are jerky pulses and a frankly bifid left ventricular impulse.16-17

Electrocardiographic abnormalities are common.8,15 There is T wave inversion in inferior and sometimes also lateral leads in about one-third of cases. Ventricular premature beats have also been reported and supraventricular tachycardia is common. Abnormalities of rhythm are often provoked by effort.

Most patients have no symptoms; in those who do these are often ill explained. Some patients are aware of an intermittent noise in the heart. Complaints of fatigue, faintness, shortness of breath, and decreased exercise tolerance may be due to exercise-induced dysrhythmias or to chest pain—which is common, often atypical, but sometimes highly suggestive of coronary origin. Woolley has recently suggested (in half fun, whole earnest) that a previously well-known but now extinct disease might in truth have been mitral valve prolapse syndrome:18 the non-entity emotively described as “soldier’s heart” or “the effort syndrome,” eponymously as Da Costa’s syndrome, or simply as neurocirculatory asthenia or “dis-orderly action of the heart.” In the era of “soldier’s heart” diagnosis had to remain conjectural unless the outcome of a disease was frequently fatal. Who can ever find out whether Woolley is right?

The incidence of mitral valve prolapse depends on how hard it is sought. The syndrome is uncommon in childhood, becomes more frequent in women than in men, but then gradually increases in frequency and in old age is probably equally common in either sex. Though mitral prolapse can result from ischaemic papillary muscle and segmental contraction abnormality, coronary artery disease is an uncommon association.

Nearly all patients with Marfan’s syndrome have echocardiographic or clinical evidence of mitral prolapse, and Marfan’s syndrome has been recognised in about 4% of reported cases.5 Myxomatous degeneration leads to ballooning of leaflets and thinning and elongation of chordae. The mitral annulus may also become appreciably dilated. Abnormalities of the thoracic cage without Marfan’s syndrome have also been described in mitral prolapse: a narrow anteroposterior diameter to the chest with a straight back (making the auscultatory features much easier to recognise). The frequency of angio- graphic mitral prolapse in secundum atrial septal defect has only recently been fully recognised; it has been reported in up to one-third of patients in whom it has been specifically sought.

The prognosis is usually benign, but four major complications may supervene. Sudden chordal rupture may lead to the abrupt development of severe mitral regurgitation, and this medical emergency is now well recognised. Gradually progressive mitral regurgitation due to increasing prolapse accounts for about 10% of valve replacements for isolated mitral regurgitation in adults. Infective endocarditis is rare, though an undoubted hazard, and this too can be responsible for the advent of a serious leak. Sudden death is the least common complication; it may be related to ventricular dysrhythmias19 or to an associated re-entry pathway leading to a catastrophic rapid ventricular rate should atrial fibrillation develop.