Why doctors must grapple with health economics

Medical journals are publishing more and more articles that report on not just the medical but also the economic consequences of treatment. The main constraints on health care are now more financial than medical or technical, and the separation of clinical judgment from financial responsibility will soon end—clinical budgeting, for example, is just around the corner. More doctors are becoming formal managers, and their medical ethics will conflict with their new status. Increasingly doctors will have to take decisions with an economic component, and therefore they must be able to understand and critically evaluate papers that contain health economics.

On p 339 Olsson and others present the economic consequences of using a β blocker, metoprolol, for prophylaxis after myocardial infarction. Their conclusion is that such treatment seems cost effective. They observed over three years that patients treated with metoprolol were on average healthier than patients treated with a placebo and that their treatment cost less in terms not only of health care (direct costs) but also of days off work and early retirement (lost production or indirect costs). The short term economic message is that using metoprolol in this way saves money both for the health care agencies and "society." This straightforward message should, however, be read with reservation. In this short space I cannot give a comprehensive critique of the paper, but examples are raised of specific questions that should be addressed by any cost effectiveness analysis. (More detailed expositions of health economics are available elsewhere.[1-4])

Most, if not all, economic studies rely on the robustness and integrity of the underlying medical study. The final message of the metoprolol paper must be read in the light of answers to questions such as: Were the sizes of control and trial groups adequate? Was the follow up long enough? In brief, the usual criteria for establishing proof of effectiveness need to be applied. The metoprolol study has three medical aspects that directly affect the economic analysis: sickness leave and early retirement (indirect costs); hospital costs (direct costs); and health outcome. A much longer study might show a change in the results and partly or wholly reverse the authors' economic conclusions. The lack of data makes it risky to extrapolate from the three year results. No refinement in the economic analysis can eradicate doubts about the long term effects of metoprolol. This does not invalidate the study, and the authors take care to mention the uncertain effects of treatment with metoprolol beyond the three years, but it does muddy the waters for taking a clear cut economic decision on whether to use metoprolol.

The dangers of extrapolating the authors' results arise from the possibility that in the long term the longer survival rates of patients treated with metoprolol will increase hospital costs. This comes perilously close to stating that euthanasia is the only valid medical action on economic grounds. This conclusion, rightly rejected by the authors, is based on the false premise that the only criterion that a cost effectiveness analysis promulgates is cost minimisation. Cost minimisation has a logical validity only when constrained by the desire to achieve positive health benefits. Early death—while cheap for the health service—is not usually considered one of the goals of medical intervention. The study's economic analysis reflects this by including indirect costs, which as well as representing some notional loss of productivity place in a crude sense an economic value on health outcome.

Economic analysis is an art requiring assumptions and proxies. In its imperfect way cost effectiveness analysis attempts to bridge the difficult gap between the inevitable use of scarce resources and the benefits that flow from a treatment or service. Cost effectiveness analysis should not be seen as having the power of veto but rather as providing one part of the information (in addition to political, social, medical, and philosophical criteria) necessary to achieve a truly informed decision about using society's resources.

JOHN L APPLEBY

District Health Economist,
South Birmingham Health Authority,
Birmingham B29 6JF

1 Drummond MF. *Principles of economic appraisal in health care*. Oxford: Oxford University Press, 1980.
2 Drummond MF. *Studies in economic appraisal in health care*. Oxford: Oxford University Press, 1981.
3 Blades CA, Cuyler AJ, Wiseman J, Walker A. *The international bibliography of health economics: a comprehensive guide to English language studies since 1914*. Brighton: Wheatsheaf Books, 1986.
4 Mooney GH, Drummond MF. Essentials of health economics. Parts I-IV. *Br Med J* 1982;285:949-50, 1101-2, 1191-2, 1263-4, 1329-31, 1405-6, 1485-6, 1561-3, 1638-9, 1727-8.

The third heart sound

The third heart sound is a series of low frequency vibrations in early diastole best heard with the bell of the stethoscope at the cardiac apex. It is normal in children and young adults but usually disappears after 40. It also occurs in patients with high cardiac output caused by anaemia, fever, pregnancy, and thyrotoxicosis. If the patient is over 40 the third heart sound nearly always shows that something is wrong—usually left ventricular failure, but less commonly mitral regurgitation or constrictive pericarditis. The timing and quality of the third heart sound remain remarkably constant regardless of its cause—except in constrictive pericarditis when the "pericardial knock" is early and high pitched.

The third heart sound is associated with rapid left ventricular filling, which occurs during relaxation of the ventricle in early diastole. When relaxation is almost over filling decelerates abruptly. This deceleration is widely held to convert kinetic energy to vibratory energy, which if sufficiently vigorous causes the third heart sound.[1-3] This theory has been challenged by Prewitt *et al*, who could find no consistent relation between any aspect of rapid ventricular filling and the timing of the third heart sound.[4] The left ventricle is, however, ellipsoid and these investigators' conclusions were based on measurements made in the transverse (short) axis; more recent work suggests that completion of long axis ventricular relaxation is the event that triggers the third heart sound.[5,6]

Van de Werf *et al* have emphasised that theories on the mechanism of the third heart sound must account for both its normal and abnormal occurrence.[7] They found that the volume and velocity of inflow and the completeness of ventricular relaxation were important determinants of deceleration during rapid ventricular filling.[8] These observations readily account for the pathological third heart sound in patients with high cardiac output or mitral regurgitation, both of which are associated with increased volume and

Br Med J (Clin Res Ed): first published as 10.1136/bmj.294.6568.326-a on 7 February 1987. Downloaded from http://www.bmj.com/ on 8 April 2024 by guest. Protected by copyright.

velocity of inflow. The observations also account for the third heart sound in left ventricular failure and constrictive pericarditis because ventricular relaxation is incomplete in both conditions, causing sudden deceleration of inflow.[9]

Age related changes in the kinetics of ventricular relaxation provide a plausible explanation for the physiological third heart sound disappearing in adults.[7] In children and young adults the left ventricle is thin and relaxes rapidly, ensuring the high inflow velocity that generates the third heart sound. With advancing age, however, the left ventricular mass increases, possibly because of rising blood pressure. The thickened ventricle relaxes more slowly thus reducing inflow velocity and ultimately causing the physiological third heart sound to disappear.

ADAM J TIMMIS

Senior Registrar,
Department of Cardiology,
Guy's Hospital,
London SE1 9RT

1 Kuo PT, Schnabel TG, Blakemore WS, Whereat AF. Diastolic gallop sounds, the mechanism of production. *J Clin Invest* 1957;**36**:1035-42.
2 Crevasse L, Wheat MW, Wilson JR, Leeds RF, Taylor WJ. The mechanism of the generation of the third and fourth heart sounds. *Circulation* 1962;**25**:635-42.
3 Arevalo F, Meyer EC, Maccanon DM, Luisada AA. Hemodynamic correlates of the third heart sound. *Am J Physiol* 1964;**207**:319-24.
4 Prewitt T, Gibson D, Brown D, Sutton G. The "rapid filling wave" of the apexcardiogram. Its relation to echocardiographic and cineangiographic measurements of ventricular filling. *Br Heart J* 1975;**37**:1256-62.
5 Ozawa Y, Smith D, Craige E. Origin of the third heart sound I. Studies in dogs. *Circulation* 1983;**67**:393-8.
6 Ozawa Y, Smith D, Craige E. Origin of the third heart sound II. Studies in human subjects. *Circulation* 1983;**67**:399-404.
7 Van De Werf F, Geboers J, Kesteloot H, De Geest H, Barrios L. The mechanism of disappearance of the physiologic third heart sound with age. *Circulation* 1986;**73**:877-84.
8 Van De Werf F, Minten J, Carmeliet P, De Geest H, Kesteloot H. The genesis of the third and fourth heart sounds. A pressure-flow study in dogs. *J Clin Invest* 1984;**73**:1400-7.
9 Van De Werf F, Boel A, Geboers J, *et al*. Diastolic properties of the left ventricle in normal adults and in patients with third heart sounds. *Circulation* 1984;**69**:1070-8.

Royal Free disease: perplexity continues

Epidemic neuromyasthenia, persistent myalgia following sore throat, Otago mystery disease, Icelandic disease, and myalgic encephalitis are just some of the names used for a chronic debilitating illness presumed to be a sequel of viral infection. The medley of names reflects the protean nature and worldwide distribution of the condition whose cardinal feature is extreme exhaustion after exercise; this is usually accompanied by a range of somatic, psychological, and "flitting" neurological symptoms. The Myalgic Encephalitis Study Group, formed in 1975, met last week at the Royal Society of Medicine to review the findings in the 70 or so suspected outbreaks and to discuss strategies for diagnosis and management.

Postviral fatigue syndrome or myalgic encephalitis are the currently favoured names for the illness, but in Britain a well known outbreak gave rise to the more familiar sobriquet, Royal Free Disease.[1] Dr Melvin Ramsay, the first speaker, was in charge of the infectious diseases unit at the Royal Free Hospital during the outbreak that affected 292 medical, nursing, ancillary, and administrative staff. The outbreak started on 13 July 1955 when a resident doctor and a ward sister were admitted with an obscure illness. By 25 July more than 70 members of staff were similarly affected and the hospital was closed until 5 October, when most of the outbreak had subsided. Although at the time a diagnosis of hysteria was seriously considered, Dr Ramsay and others at the hospital never doubted that in most cases there was an infectious cause. Because they found fever in 89%, lymphadenopathy in 79%, ocular palsy in 43%, and facial palsy in 19% they considered that a diagnosis of hysteria was untenable in all but a few patients who had panicked about the mystery illness.

An analysis by two psychiatrists in 1970 of the case notes of 100 Royal Free nurses and 100 matched controls led them to conclude, however, that the Royal Free outbreak was the result of pure hysteria.[2] Those who thought that an organic disease had caused the outbreak vigorously resisted this suggestion, claiming that including in the analysis patients who had succumbed to panic had led to a mistaken conclusion. Nor did supporters of the infection theory think that sufficient weight had been given to the observation that sporadic cases showing a similar clinical picture had occurred in north west London both before and after the Royal Free outbreak. The division between those who believe that the outbreak was caused by an infection and those who favour a psychological explanation continues to echo down the years. Only two weeks ago in a letter to the *Lancet* a professor of neurology, currently working at the Royal Free Hospital, reported that at follow up some of the original patients showed simultaneous activation of agonist and antagonist muscles, raising the possibility of simulation of symptoms.[3]

Epidemiological evidence that an infectious agent is responsible for postviral fatigue syndrome has come from other countries. Some epidemics taken to be poliomyelitis, such as one in Los Angeles in 1934, have with hindsight been identified as postviral fatigue syndrome, and in five other outbreaks a subtle relation between poliomyelitis and postviral fatigue syndrome was discernible. For example, in 1955 an epidemic of type 1 poliomyelitis spread round the coast of Iceland but failed to become established in townships where seven years earlier there had been an outbreak of "epidemic neuromyasthenia." Prior infection with the agent causing neuromyasthenia was presumed to have inhibited the cytopathological effects of the poliovirus. This led to the suspicion that the causal agent of the epidemic neuromyasthenia had been another enterovirus, because enteroviruses are known to be mutually antagonistic. Other viruses, including herpes viruses and Epstein-Barr virus, are also believed to be able to trigger the syndrome; but Coxsackie B virus has been the culprit suspected in most investigations.

Until recently most cases of postviral fatigue syndrome have been epidemic. But since 1980 evidence that the illness may be endemic has accumulated from the west of Scotland. General practitioners in rural and seaside practices in Ayrshire, Stirlingshire, and Dunbartonshire recognised that considerable numbers of previously healthy adults, predominantly aged 30 to 40, were presenting with an illness characterised by profound chronic exhaustion and other ill defined symptoms including emotional lability.[4-6] The many similarly affected patients drew attention to the condition; in the absence of abnormal laboratory findings or of persistent physical or neurological deficits individual patients might well have been regarded as neurotic, depressed, or hypochondriacal. But because they had not shown these traits before the illness an organic cause was considered.

The only consistent positive laboratory finding was a raised but static titre of Coxsackie B neutralising antibody. A controlled study, reported last month by Calder *et al*, of sera from the Dunbartonshire practice found that 46% of 140 patients with postviral syndrome and 25% of 100 controls

Br Med J (Clin Res Ed): first published as 10.1136/bmj.294.6568.326-a on 7 February 1987. Downloaded from http://www.bmj.com/ on 8 April 2024 by guest. Protected by copyright.