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Equalities and inequalities in health

Five years ago Sir John Brotherston's Galton Lecture[1] at the annual symposium of the Eugenics Society asked the question "Is inequality inevitable?" The tenor of his reply was that equality might not be attained in health for biological reasons but that inequality in health care certainly could be reduced and that the redistribution of resources was "the major instrument we must employ."

The recent report by Sir Douglas Black and his colleagues on *Inequalities in Health*,[2] so grudgingly made available by the DHSS, presents in 417 pages much detail that Brotherston could not give us, rather selectively culled from a bibliography (not well presented). The recommendations have a certain detachment from reality which explains, though it does not justify, Patrick Jenkin's dismissive foreword—which says, in effect, that they are too expensive now or in the foreseeable future and anyway do not necessarily provide the right formula. It may be true that the Government cannot at present provide the money for an ideal policy within the overall budget on health and welfare. That is no reason, however, for the Secretary of State failing to look at the pattern of services which could be developed even within present resource limitations. After all, his own estimate that the recommendations would cost another £2000m is only slightly more than the sum required to bring per caput spending on the NHS in England and Wales to that already spent in Scotland and Northern Ireland. In fact, the extra money the Scots and Irish have been given in the past 30 years has been spent on hospital-based services rather than on the community services the group wants to see developed; the outcome of that policy is worthy of a study that the group did not attempt.

Recent DHSS publications on priorities, prevention, and resource allocation have been largely endorsed by the group. In particular, it would like to see the redistribution recommended by the Resource Allocation Working Party[3] hastened and a much greater effort made on prevention. Some of its comments seem a little facile. All those concerned with "acute hospital services" must be growing tired of being told that they must cut back to allow some of the money saved to be spent outside the hospitals. Furthermore, the recommendation in the priorities document that savings should be made in the maternity services has recently been rebutted comprehensively by Mrs Renée Short's Parliamentary Select Committee,[4 5] but the report agrees with Mrs Short that the professionals concerned must make maternity care more patient-centred. This sort of approach could be extended to other disciplines, in association with community health councils.

High-technology medicine is the usual target for those who demand a larger share of NHS resources for community services and prevention. This latest report is even more emphatic than others and equally vague about the elements to be cut. Admittedly, the specialist services give plenty of openings to their attackers. The recent series of articles by Card and Emerson[6 7] show how expensive technical investigations could be reduced without lowering the quality of care given to patients—indeed imposing less stress on them and saving their time. The economics possible in prescribing, including the use of generic names of drugs, are frequently extolled, but the educational services in therapeutics which would most help this have been developed in few districts. Professor Bryan Jennett's four radio talks on "Doctors, patients, and responsibility" presented some of the dilemmas to the public in true perspective: when a service operates under a resource ceiling anything extra must be at the expense of giving up something else.

It may be that inpatient time could be saved by further shortening the stay for many conditions, though more staff and other resources might be needed to do it. Whether time thus saved should be used to reduce hospital bed use, probably by closing wards or small hospitals, or to shorten waiting time for other patients, should be decided by rational processes not by guesswork. Some kind of social assessment of gains and losses is needed, whether in the cash terms of Bunker *et al*[8] or some social index. Too little attention has been paid in this and other reports to the misery entailed for some patients or families by long waiting times for surgical treatment or admission for long-term care. Some commentators in their understandable concern for long-stay patients fail to realise that these are less than one-twentieth of the total admitted in a year, though they occupy over half the beds at any time.

Sir Douglas Black's group has done a valuable service in bringing out the importance of social welfare, housing and education services, and social security payments in maintaining health. Britain could draw important lessons from some other EEC countries as well as from the Nordic group; for example, sheltered housing has been used extensively in West Germany.

The emphasis in the report on prevention is welcome but a little vague. Certainly health education should be strengthened, but it cannot be effective without the full participation of clinicians, particularly general practitioners, and school staff. Statutory control of some of the anti-health promotion of alcohol, tobacco products, and the wrong foods is seen as essential. Indeed, the most explicit recommendations largely follow those of the WHO Expert Committee on Smoking Control and the Fourth World Conference on Smoking and Health. The report gives justified prominence to the importance of environmental changes to reduce accidents, especially to children, and to make it easier for the urban population to take exercise. It examines the effects of unemployment but gives greater emphasis to nutrition, the need for adequate heating for young and old, and overcrowding. How depressing that it should still be necessary to parade

evidence that the Medical Research Council found convincing 50 years ago.

One of the most interesting proposals is that the 10 areas with the worst mortality records should become special development areas to which £30m of extra funds might be given. This is a far more ambitious proposal than Sir Keith Joseph's demonstration centres for such services as geriatric care and rehabilitation eight years ago or the earlier attempt to start health centres in underdoctored areas. Such an overall effort might indeed help, though it would inevitably produce new inequalities.

The report contains a mass of material that deserves full analysis. It is, however, far less well presented than and different in form from the US Health Department publication *Healthy People* of a year ago. It has a different purpose—furthering change in an established service—and concerns other departments as well as the DHSS. Surely it should be examined more closely than Patrick Jenkin's foreword suggests that it has been. Even though its objects are not attainable in the short term and may be wrong in some major respects, it could be modified so as to become a long-term development plan for health and social services. Mr Jenkin should think again.

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Laboratory features of pleural effusions

The clinical history, physical examination, and chest radiograph may give clues to the cause of a pleural effusion, but often this remains obscure. Examining the fluid may provide further clues. Blood staining suggests a traumatic or neoplastic origin, but may occur in infarction of the lung or pneumonia. Classically serous effusions are divided into transudates and exudates. Transudates have protein concentrations of 0·5-1·5% and a specific gravity of less than 1·015, and do not clot on standing. They occur in cardiac or renal failure or in deficiency disorders in which there is generalised oedema. Exudates result from inflammation of the pleura; they have a higher protein concentration of 3-5% and a specific gravity over 1·018, and they clot on standing. Microscopy will show the type of cells present—usually lymphocytes in tuberculous effusions and polymorphonuclear leucocytes in postpneumonic effusions, though exceptions occur. Cytological studies may show malignant cells. Bacteria may be visible or may grow under the correct culture conditions. These standard tests are useful in diagnosing the commoner causes of pleural effusions, but are less applicable to the more unusual causes.

Pleural lesions are a frequent manifestation of rheumatoid arthritis and systemic lupus erythematosus.[1] Up to a fifth of patients with systemic lupus erythematosus may develop effusions. In rheumatoid arthritis, however, although pleuritis is common, effusions occur in only 2-3% of patients. Here they may present a difficult diagnostic problem, especially when they antedate or accompany the rheumatic disorders. When acute febrile pleurisy occurs in rheumatoid arthritis, tests must establish whether this is part of the rheumatoid process or an infective or neoplastic condition in a possibly immunosuppressed individual.

The clinical usefulness of the various diagnostic tests for determining the cause of pleural effusions has recently been evaluated by Halla and his colleagues.[2] Several laboratory tests emerged as reliable discriminators between rheumatoid and systemic lupus effusions. All seven rheumatoid pleural fluids had an acidosis (pH $\leqslant$ 7·2), while all five systemic lupus fluids tested had a pH of 7·35 or more. This study also confirmed that rheumatoid pleural fluids are characterised by low glucose concentrations: all had less than 1·4 mmol/l (25 mg/dl) glucose, whereas all fluids from patients with systemic lupus erythematosus contained 4·4 mmol/l (80 mg/dl) or more. Lactic dehydrogenase concentrations exceeded 700 IU/l in rheumatoid effusions and were 500 IU/l or less in seven of the eight patients with systemic lupus erythematosus. Rheumatoid factor titres were measured by the latex fixation method; all rheumatoid effusions from seropositive patients were positive for rheumatoid factor with a titre greater than in the serum, while only one effusion from a patient with systemic lupus erythematosus was positive. Rheumatoid factor is not, however, specific for or limited to rheumatoid disease, for it is present in the pleural fluid of 41% of patients with bacterial pneumonia, 21% of patients with carcinoma, and 14% of patients with tuberculosis.[3]

More important, Halla and his colleagues[2] assessed the laboratory variables that might distinguish the effusions of systemic lupus erythematosus and rheumatoid disease from effusions due to other causes. Complement activity has been suggested as one such variable,[4] but Halla *et al*[2] found significant differences only for C4. The presence of immune complexes in the pleural fluid, however, appeared to provide a basis for a useful diagnostic test of rheumatoid disease. They used three detection reagents—monoclonal rheumatoid factor radioimmunoassay, C1q binding assay, and Raji cell radioassay. Each assay recognises a different biological property of immune complexes. The rheumatoid effusions contained immune complexes, nearly all at concentrations greater than those in the serum, on all three tests. In contrast, immune complexes in systemic lupus effusions were detected only by the Raji cell radioassay and then at concentrations similar to those in the serum. Only one patient with systemic lupus erythematosus gave a positive result with the monoclonal rheumatoid factor assay. Immune complexes in the pleural fluid from patients with other diseases were rare. The high concentration in rheumatoid pleural effusions suggests that immune complexes are produced within the pleural cavity in rheumatoid disease, whereas in systemic lupus erythematosus immune complexes in effusions seem to reflect the serum concentration. This is in keeping with the current theory that systemic lupus erythematosus is an intravascular and rheumatoid arthritis an extravascular immune complex disorder.

Halla and his colleagues[2] believe that measuring soluble immune complexes aids the differentiation of pleural effusions of rheumatic and non-rheumatic diseases. Measurement can be based on agar gel diffusion with monoclonal rheumatoid factor and human C1q. The test is technically simple and, although not quantitative, it yields frequencies of positive