would do wonders for the morale of the rest of the staff concerned with the actual care of patients. Furthermore, whileit has recognised the demographic trends, the Government seems to have taken little account of them in their wider implications. If the proportion of old people in the population is rising and will continue to rise, then we cannot afford to encourage a trend towards earlier retirement. A man of 60 in average health now has an expectation of life of over 15 years and a woman of 60 can expect to reach her eightieth year; even 70-year-olds can expect to live 10 and 12 years respectively.[5] Many of these old people are willing, eager, and fit to work, and they should surely be encouraged to do so by tax concessions and any other incentives. We need to strike a more reasonable balance between the numbers in the work force and the size of the population they have to support.

In the medium to long term, however, it is becoming increasingly clear that the NHS cannot balance its books and has no chance of doing so. The DHSS plans depend on cutting investment (capital expenditure) to well under 10% of turnover and by implication accept that patients in Britain will have poorer medical services than those available in many other western countries. This is a policy of despair, and it reflects the Department's refusal over the years to recognise the really fundamental issues that must be resolved if the NHS is to survive as a first-class health service.

Firstly, the NHS is underfinanced. No doubt the reason that Britain spends less of its national wealth on health than do our neighbours reflects the way in which the Service is paid for. Countries which use compulsory health insurance schemes to finance a comprehensive service (such as Denmark) seem to be able to extract more money from their populations. The many possible ways of augmenting NHS income include some payment by patients for drugs or for hospital "hotel charges," national lotteries, an expansion of the private sector, or switching to an insurance-based State system. However arranged (and this is a political decision), an increase in national spending on health is a vital priority and one that should be examined in detail by the Royal Commission.

Secondly, the career structure in the NHS is responsible for most of the problems of hospital staffing and low job satisfaction among recent graduates. There is little point in the consultative document talking of "a medical school student intake of some 4000 a year by 1980 which will enable a higher proportion of posts in hospitals and general practice to be filled by British graduates" so long as there is the present disparity between the numbers of training and career posts in the hospital service. We need a rational policy, possibly closer to the European pattern, and this is urgent:[6] here the responsibility rests squarely with the medical profession to cease interspecialty demarcation disputes and agree to a solution that makes mathematical sense.

Finally, and without apology, we return to a familiar theme. As medical technology continues to advance, the treatment of coronary thrombosis, stroke, lung, bowel, and breast cancers, degenerative arthritis, and the other really common illnesses in our society will continue to improve and become more expensive. Faced with a life-threatening illness, every citizen has a right to expect the NHS to provide him with the best treatment available, or, at the very least, treatment along the lines agreed to be most effective by orthodox medical opinion. Not too long ago, the NHS could claim to satisfy this duty, but it can no longer do so, and the gap between performance and expectation is widening daily. In part, the answer lies in finding more money for the acute services, but that is likely to be deferred until the report of the Royal Commission. The alternative approach is to decrease the incidence of these common disorders. We know how to do this: most of them are linked, quite conclusively, with excess intake of food, alcohol, or tobacco smoke or are due to traffic accidents or industrial injuries that are often preventable. A sustained campaign of health education (at the profession as well as the public) offers the only rational long-term solution to the financial equations of medical care.

The Department's philosophy then, should, be rejected as defeatist. It accepts as inevitable a growing disparity between the NHS we can afford to provide and the medical skills that modern technology can offer. There is an alternative, and a practical and realistic one: it envisages concentration on prevention and especially on the prevention of birth handicap. An analogy may be drawn with the problem presented by rhesus haemolytic disease of the newborn in the early 1960s, when it demanded a greater and greater share of the services in obstetric, paediatric, haematology, and radiology departments. By cutting through to the cause of the disease, the problem has been solved. Talk about priorities should not be concerned with adjustments of 1-2% up or down in the established patterns of medical care: it should seek to identify when and where massive shifts should be made in our plans for health care. We need a new strategy, not a change in tactics.

[1] DHSS, *Priorities for Health and Personal Social Services in England*. London, HMSO, 1976.
[2] *British Medical Journal*, 1976, **1**, 245.
[3] Edington, P T, Sibanda, J, and Beard, R W, *British Medical Journal*, 1975, **3**, 341.
[4] Chantler, C, and Barratt, T M, *Lancet*, 1976, **1**, 583.
[5] CSO, *Social Trends*. London, HMSO, 1975.
[6] *British Medical Journal*, 1976, **1**, 546.

Foresight prevents blindness

Blindness has been selected as the focus of attention on this year's World Health Day on 7 April. No captain of the men of death, blindness nevertheless has provided for thousands of years one of the chief symbols of suffering, exacting a huge toll in terms of the sum of individual misery and deprivation and the accompanying loss of potential achievement.

World Health Organisation statistics estimate that there are 10 million totally blind people in the world today. In at least an equal number sight is so impaired as to limit education and employment severely and enforce dependency on community or family. In the purely economic sense lack or loss of vision is a drain of brain and skill from any country's human resources. Much of the world's blindness is distributed and determined in the same way as malnutrition; originating from infection and dietary inadequacy, it is found most often in poor, squalid, and overcrowded environments.

Infection is the most frequent cause. Acute infection gives rise to neonatal ophthalmia (gonococcal and staphylococcal) and at a later age to severe conjunctivitis, often crucially damaging to corneal integrity, in the course of measles or severe malnutrition. Viruses such as herpes and variola have a smaller role. The rapidity of attack, delays in reaching treatment centres, and inadequate therapy often prove lethal to eyesight. The important chronic infections are trachoma, a viral keratoconjunctivitis of dry, dusty, overcrowded environs, and onchocerciasis, a filarial ophthalmitis of wetter forest areas. Their insidious onset hinders early diagnosis and promotes a tolerance, akin to that for flies and smoke, which delays the

search for advice and inexorably impairs vision. Trachoma alone has been estimated to affect 400 million people.[1]

Vitamin A deficiency is estimated to be responsible for 80 000 new cases of xerophthalmia a year,[2] mostly in the vast conurbations of South and East Asia, the Eastern Mediterranean, and Central America. In India 15 000 children under 5 lose their sight each year for this reason. Cataract, excluding the relatively rare congenital conditions, is a common global cause for blindness in the older age groups. Accidental trauma to the eyes is another, smaller source of loss of vision.

In most of the temperate zones of the world blindness was reduced considerably during the last century, even before the advent of modern medical therapy, through improvements in hygiene, water supplies, nutrition, and standards of living—measures still awaited in many countries. During this period major advances were made in the alleviation of the handicap of blindness itself: blind schools were opened and the Braille script developed.

Prevention of blindness in the third world has only relatively recently emerged as a practical proposition. The first line of attack includes all those improvements in living standards, environmental hygiene, water supplies, and overcrowding which developing countries are struggling to achieve. Specific mass methods of prevention, relatively inexpensive, are now available, such as vitamin A supplementation of the diets; vaccination against smallpox, measles, and trachoma; chemoprophylaxis against trachoma, gonococcal ophthalmia, and keratomalacia; and vector control for onchocerciasis. The problem lies in making the available knowledge applicable and acceptable to governments and people. The total annual cost, for instance, of protecting 100 million children aged 1-5 all over the world against the risk of xerophthalmia would be $3 million[3]—a drop in the ocean compared with the amounts spent on armaments and advertising.

The second line of attack depends on early diagnosis and the application of the appropriate treatment, often on a mass screening scale. Prevention and cure are inseparable activities in the immediate and adequate antibiotic therapy of conjunctivitis, the early and persistent treatment of trachoma and onchocerciasis, and the use of parenteral vitamin A in xerophthalmia. Here again the problem lies not in what to do but how to do it.

Much is to be done in the future, but many partial, small-scale inroads have already been established. Mobile eye clinics are being used in rural areas not covered by static dispensaries. These clinics combine cure and prevention, protecting communities and individuals "at risk" by detecting early disease and by enumerating the partly and totally blind. Treatment includes surgery, which may vary from removal of a foreign body to cataract extraction. Auxiliaries are being selected and trained to a high degree of efficiency,[4] and this has made possible a far greater coverage for the screening and treatment of eye diseases and for the education of people in simple hygiene. The use of healed patients has also been mooted particularly in the elimination of trachoma.

The World Health Organisation is sharpening and extending the attack on the causes and effects of blindness. Will its theme of foresight and prevention include a vision of the day when blindness will be like the hunched back of Pott's disease, the pockmarked face of smallpox, or the bow legs of rickets, a historical curiosity?

[1] Duke-Elder, S, *System of Ophthalmology*, 1965, **8**, (1), 258. London, Kimpton.
[2] McLaren, D S, *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1966, **60**, 436.
[3] Amadou Mahtar M'Bow, *Cajanus*, 1975, **8**, 338.
[4] Burkitt, W R, *Tropical Doctor*, 1975, **5**, 30.

Histamine antagonists and peptic ulcer

Neutralising or inhibiting gastric acid secretion was thought essential for the healing of peptic ulcers until just over a decade ago, when liquorice derivatives were clearly shown to accelerate ulcer healing. The interest which has been maintained in such compounds since then has reflected their undoubted activity—and also the lack of convincing evidence that even vigorous treatment with alkalis or anticholinergic agents can alter the natural course of a gastric or duodenal ulcer. Recently two groups of compounds have promised to restore the dominant role of acid inhibition in the treatment of gastric and duodenal ulcer: the substituted prostaglandins and the histamine H_2 antagonists, which are potent inhibitors of acid secretion. The series of histamine antagonists so far produced includes burimamide, metiamide, and cimetidine.

Pharmacological responses to histamine in small intestinal smooth muscle or in the bronchi can be blocked by conventional antihistamines such as mepyramine,[1] but it has long been known that ordinary antihistamines will not prevent gastric acid secretion induced by histamine—a phenomenon exploited in the augmented histamine test of acid output devised over 20 years ago. This separation between histamine H_1 and H_2 receptors has been emphasised recently by the synthesis of histamine analogues (in which the side chain has been altered and extended) which are potent inhibitors of H_2 acid secretory responses but are devoid of H_1 activity.[2]

The first of these drugs, burimamide, was replaced by metiamide, which was more active, especially when given by mouth. However, metiamide had to be abandoned because it may cause agranulocytosis,[3] and a third drug, cimetidine, is now available. Since the pharmacological properties of the three compounds are very much the same the results obtained with metiamide remain of considerable interest. It proved to be a potent suppressor of both basal and stimulated acid secretion when given by mouth. In patients with duodenal ulcer it greatly reduced both meal-stimulated and nocturnal secretion,[3 5] and it almost completely abolished responses in those with gastric ulcers.[6] These observations led to clinical trials, which showed that duodenal ulcer symptoms could be relieved and ulcer healing could be shown far more often in patients receiving the drug than in those receiving placebos[7 8]—though in the second trial healing and symptom relief did not necessarily go in parallel. A more severe test of the compound was its use in the Zollinger-Ellison syndrome, but again acid hypersecretion was controlled, with corresponding clinical benefit to the patients.[9 10]

Cimetidine, the replacement drug, has the thiourea moiety of the molecule replaced by cyanoguanidine, and this change should prevent the haematological problems experienced with its predecessor[11]; one piece of evidence which supports this view is that a patient who developed bone marrow suppression while receiving metiamide had no ill effects while treated with cimetidine.[12] Experience obtained with cimetidine is necessarily limited; apart from inhibiting pentagastrin and histamine responses in the stomach, it is a potent suppressor of meal-stimulated acid output[13] and appreciably reduces the acidity of gastric contents when these are sampled throughout the day and night.[14–18]

Some important questions now need answering. Will treatment with cimetidine prove to be of clear benefit in healing duodenal and gastric ulcers; if it does work what will the