virological investigation are negative. These data suggest that though virus may be causally related the mechanism is more complex than straightforward viral myocarditis; some immunological idiosyncrasy seems likely.

Immunological studies in dilated cardiomyopathy have shown preferential binding of IgG and IgA.²⁰ Studies of cell mediated immunity have shown abnormal transformation of lymphocytes to phytohaemagglutinin,²¹ that the percentage of circulating T lymphocytes is reduced,²² that leucocyte migration is inhibited,²³ and that T suppressor cell function is defective.^{24 25} A more recent study found that in two fifths of patients the ratio of helper to suppressor cells was higher than in normal controls.²⁶

These studies and other experimental evidence strongly suggest that viral infection evokes an immunological response that results in dilated cardiomyopathy.²⁷⁻²⁹ Possibly the viral infection may trigger antibody production directed at suppressor cells. These antibodies might then affect T cell receptors, which modulate β cell function; the result would be increased β cell activity and production of autoimmune antibodies. Dysfunction of T suppressor cells might also affect cell mediated immunity.23 Nevertheless, by no means all workers accept the autoimmune hypothesis.³⁰

Other avenues of investigation need to be explored in patients with dilated cardiomyopathy, but an infectiousimmune mechanism seems likely to be the cause in about half.

With rare exceptions,¹⁹ searching for virus in the myocardium using the standard techniques has failed even when immunofluorescence techniques specific for Coxsackie virus have been used. This is not altogether surprising: replication usually takes place early on, and by the time the patient comes to the physician morphologically recognisable virus may no longer be present.³¹ Recently a pilot study using a deoxyribonucleic acid hybridisation probe technique to detect ribonucleic acid sequences specific for Coxsackie B virus has yielded promising results when applied to endomyocardial tissue recovered by bioptome. Even if myocarditis is not present evidence may be found of virus in the myocardium.³² This may even apply to patients whose biopsy specimen has shown no evidence of active myocarditis or myocarditis in the past.

The pieces of the jigsaw are now beginning to fall into place, but much work is still necessary. Central to all studies is examination of tissue obtained by bioptome and the accurate diagnosis of myocarditis. Newer diagnostic techniques such as two dimensional echocardiography, Doppler ultrasonography, magnetic resonance, and scintigraphy (particularly gallium scanning) may help in the diagnosis of myocarditis. These techniques are, however, unlikely to replace the study of specimens obtained by bioptome in monitoring the response to treatment. So far as treatment is concerned the studies have been without proper controls. Patients with myocarditis may recover spontaneously²⁸ and hence a multicentre controlled trial is mandatory.

Consultant Histopathologist, National Heart Hospital, London W1M 8BA

1 World Health Organisation/International Society and Federation of Cardiology Task Force. Report on the definition and classification of cardiomyopathies. Br Heart 7 1980;44:672-3

ECKHARDT G J OLSEN

Bristow MR. The adrenergic nervous system in heart failure. N Engl 7 Med 1984;311:850-1.
 Factor SM, Cho S, Sonnenblick EH. Verapamil treatment of cardiomyopathic Syrian hamsters:

- 5 Bagger JP, Baandrup U, Rasmussen K, Moller M, Vesterlund T. Cardiomyopathy in western Denmark. Br Heart 7 1984;52:327-31.
- 6 Williams DG, Olsen EGJ. Prevalence of overt dilated cardiomyopathy in two regions of England. B Heart 7 1985;54:153-5
- Sakakibara S, Konno S. Endomyocardial biopsy. Jpn Heart J 1962;3:837-43.
- Richardson PJ. King's endomyocardial bioptome. Lancet 1974;i:660-1. Olsen EGJ. Histomorphologic relations between myocarditis and dilated cardiomyopathy. In: Bolte HD, ed. Viral heart disease. Berlin: Springer-Verlag, 1984:5-12. 10 Fowles RE. Progress of research in cardiomyopathy and myocarditis in the USA. In: Sekiguchi M,
- Olsen EGI, Goodwin IF, eds. Myocarditis and related disorders. Proceedings of the interne symposium on cardiomyopathy and myocarditis. Berlin: Springer-Verlag, 1985:5-7. (Also published as suppl 1 to Heart and Vessels 1985.)
- 11 Baandrup U, Florio RA, Olsen EGJ. Do endomyocardial biopsies repres the myocardium? A semiquantitative and light microscopic study of single versus multiple biopsies with the King's bioptome. Eur Heart J 1982;37:171-8.
- Olsen EGJ. Myocarditis-a case of mistaken identity? Br Heart 7 1983;50:303-11
- Richardson PJ, Morgan-Capner P, Daly K, McSorley C, Olsen EGJ. Endomyocardial biopsy and viral heart disease. Verh Disch Ges Herz Kreislaufforsch 1983;49:141-8.
- 14 Billingham ME, Olsen EGJ, Fengolio JJ Jr, et al. Myocarditis pathology panel, Dallas, Texas, 24 March 1984. Am J Cardiovasc Path 1986 (in press).
- Mason JW, Billingham ME, Ricci DR. Treatment of acute inflammatory myocarditis assisted by endomyocardial biopsy. Am J Cardiol 1980;45:1037-44.
 Daly K, Richardson PJ, Olsen EGJ, et al. Acute myocarditis. Role of histological and virological examination in the diagnosis and assessment of immunosuppressive treatment. Br Heart J 1004 (5):2020.
- 1984:51-30-5 17 Cambridge G, MacArthur CGC, Waterson AP, Goodwin JF, Oakley CM. Antibodies to Coxsackie
- B viruses in congestive cardiomyopathy. Br Heart J 1979;41:692-6. 18 Richardson PJ. Viral myocarditis and cardiomyopathy. In: Maseri A, ed. Hammersmith cardiology workshop series. Vol 2. New York: Raven Press, 1985:111-7.
- 19 Kawai C. Idiopathic cardiomyopathy: a study on the infectious-immune theory as a cause of the disease. Jpn Circ J 1971;35:765-70.
- 20 Bolte HD, Schultheiss P. Immunological results in myocardial diseases. Postgrad Med J 1978;54:500-3.
- 21 Das SK, Petty RE, Meengs WL, Turbergen DJ. Cell mediated immunity in cardiomyopathy. Circulation 1976;53, 54:(suppl 2):22.
- 22 Sachs RN, Lanfranchi J. Cardiomyopathies primitives et anomalies immunitaires. Coeur et Medecine Interne 1978;17:193-8.
- 23 Das SK, Stein LD, Reynolds RT, Thebert P, Cassidy JT. Immunologic studies in cardiomyo pathy and pathophysiologic implications. In: Goodwin IF, Hialmarson A, Olsen EGI, eds. Congestive cardiomyopathy, Kruna, Sweden, 1980. Molad, Sweden: A B Hässle, 1981:87:93.
 Fowles RE, Biober CP, Stinson EB. Defective in vitro suppressor cell function in idiopathic
- congestive cardiomyopathy. Circulation 1979;59:483-91
- 25 Eckstein R, Mempel W, Bolte HD. Reduced suppressor cell activity in congestive cardiomyopathy and in myocarditis. Circulation 1982:65:1224-9
- 26 Sanderson JE, Koech D, Iha D, Ojiambo HP. T-lymphocyte subsets in idiopathic dilated cardiomyopathy. Am J Cardiol 1985;55:755-8.
 27 Ablemann H, Miklozek C, Modlin JF. The role of viruses in the aetiology of congestive
- cardiomyopathy. In: Goodwin JF, Halmarson A, Olsen EGJ, eds. Congestive cardiomyopathy. Kiruna, Sweden, 1980. Mölndal, Sweden: A B Hässle, 1981:76-84.
- 28 Goodwin JF. On the possibility of viral myocarditis as an important aetiologic agent of cardiomyopathy. In: Sekiguchi M, Olsen EGJ, Goodwin JF, eds. Myocarditis and related disorders. Proceedings of the international symposium on cardiomyopathy and myocarditis. Berlin: Springer-Verlag, 1985:4. (Also published as suppl 1 to Heart and Vessels 1985.)
- 29 Sekiguchi M, Yu Zu-Xi, Hasumi M, Hiroe M, Morimoto S, Nishikawa T. Histopathologic and ultrastructural observations of acute and convalescent myocarditis: a serial endomyocardial biopsy study. In: Sekiguchi M, Olsen EGJ, Goodwin JF, eds. Proceedings of the international
- symposium on cardiomyopathy and myocardiis. Berlin: Springer-Verlag, 1985:143-53. 30 Thompson RA, Trueman T, Harvey MR, Haschett M, Littler WA, Retieff L. Immunological methods and cardiomyopathy. Lancet 1980;i:47.
- Ablemann WH. Congestive cardiomyopathy. In discussion. Postgrad Med J 1978;54:509.
- 32 Bowles NE, Richardson PJ, Olsen EGJ, Archard LC. Detection of Coxsackie B virus-specific RNA sequences in myocardial biopsy samples from cases of myocarditis and dilated cardiomyopathy. Lancet 1986; (in press)

Whatever happened to the **Black report?**

Like the Bible, the Black report on inequalities in health is much quoted, occasionally read, and largely ignored when it comes to action.¹ Six years after its publication virtually none of its 37 recommendations have been implemented (even the 23 that would have cost little or nothing), and all the data available suggest that the gap between the rich and poorboth in their income and in their health-is widening.

Worried by the plight of the growing number of poor in Britain, the British Medical Association, the Trades Union Congress, and the Health Education Council joined together last week and held a conference to try to find a way forward. The mood of the conference was that radical measures were necessary, and Sir Richard O'Brien, formerly chairman of the Manpower Services Commission and speaking at the conference in his role as chairman of the Archbishop of Canterbury's commission on urban priority areas,² suggested

effects on the micro circulation and the extent of myocardial necrosis. Fed Proc 1981;4:758. 4 Torp A. Incidence of congestive cardiomyopathy. In: Goodwin JF, Hjalmarson A, Olsen EGJ, eds. Congestive cardiomyopathy, Kiruna, Sweden, 1980. Mölndal, Sweden: A B Hässle, 1981: 18-21

that the whole issue was a question of morality and values. If defeating poverty and improving housing and health were to be put at the top of the nation's agenda then "we could start tomorrow and solve these problems."

Sir Richard described how his commissioners had been "shocked and disturbed" when they walked the streets and talked to the people of the urban priority areas (and he apologised for the name). They saw a "different kind of life in a different Britain" and unanimously found its quality unacceptable. The poverty that these "good and great" saw, some of them for the first time, swept away complacency. Most members of the government have not had that experience and were able to dismiss the commissioners' report as unrealistic, Marxist, or simply too expensive to implement.

But the government does have to hand the statistics that describe Britain's expanding poverty and its effects. In 1976 the poorest fifth of the population received 7.4% of national income while the richest fifth received 37.9%; by 1983 the poorest fifth received 6.9% and the richest fifth 39.3%. In 1984 4.6 million people were receiving supplementary benefit compared with 2.7 million in 1974, and if the amount received on supplementary benefit is taken as the poverty line 7.7 million were living on or below the line in 1981 and 2.8million were below it (the latest figures available). Because of the huge increase in unemployment since 1979 young families now make up a much higher percentage of the poor, and the number of children growing up in poverty doubled between 1979 and 1981. Homelessness is one of the worst consequences of poverty, and 140 000 heads of households were registered homeless in Britain in 1984 compared with 41000 in 1979. Shelter estimates that 9000 families in England are living in "bed and breakfast" accommodation-4000 of them in London at an estimated cost of £26m.

The link between poverty and excess mortality and morbidity is clearly established, and so this increasing poverty must be leading to more death and sickness. The Black report showed that the mortality of unskilled manual workers and their families was at all ages never less than twice that of professional men and their families-and between 1 month and 11 months of age it was more than four times as high. The latest occupational mortality data, which are due to be published any day (and may have been delayed because of the conference), are expected to show that the mortality differences have increased. Depressing data were also presented at the conference that suggested that, although overall death rates from diseases amenable to treatment are falling, the ratio of excess mortality among the poor compared with the rich for those diseases is increasing. Thus effective medical treatments are failing to reduce inequalities in health, and even preventive methods may be increasing them-smoking rates, for instance, have fallen much faster among the better off.

So what could be done? Money alone will not solve the problem, but little is likely to be achieved unless large sums are made available mainly to reduce inequalities in income but also to improve services. In dismissing the proposals of the Black report in 1979 the government said that to implement them would cost upwards of £2 billion a year, and the proposals of the Archbishop of Canterbury's commission were costed by an economist from the Institute of Fiscal Studies at £4 billion.³ This, the economist said, would mean 4p on the standard rate of income tax. Other possible sources would be the £4.5 billion that would be raised by abolishing income tax relief on mortgages, or the large sum that would result from abolishing the married man's allowance. Many speakers at the conference also referred to the money spent on defence—particularly on nuclear weapons and on the Falklands. But despite these suggestions the conference was well aware that increases in income tax or drastic cuts in defence expenditure with the money being diverted to the poor are unthinkable to the present government. Indeed, the current political reality is that the Social Security Review is likely to make many of the poor still poorer.

Whether or not they are given extra resources are the health services capable of reducing inequalities in health? Mr Michael Schofield, general manager of Rochdale Health Authority, thought that the services as they are organised now are structurally incapable of doing much. Too high a proportion of health service money, he thought, is spent on hospitals, which are "isolated" from the communities around them (often poor communities) and infiltrated with a culture wholly different from that of the deprived. More money should be spent on community services, and the Resource Allocation Working Party, instead of taking money from hospitals in the south and giving it to hospitals in the north, should really be shifting health service money into community services. It remains perhaps an article of faith that community services could do much to reduce the health consequences of poverty, but Mr Schofield was particularly impressed by a child development programme operating in various districts in Britain that had used local helpers who had themselves been through hard times to work with socially disadvantaged children. The money for this project had come from the Netherlands. Mr Schofield's thoughts were supported in part by Sir John Crofton, former president of the Royal College of Physicians of Edinburgh and one of the authors of a Scottish report on health education and multiple deprivation: he argued that projects to remove inequalities in health worked best if they started at the grass roots and worked up.

The truth is that despite the Black report and the conference we probably do not know the best ways to remove inequalities in health—because there has never been a wholehearted commitment to try to do so. Nevertheless, paradoxically, the government has now made such a commitment: almost without anybody noticing, it has endorsed the 38 targets of the European region of the World Health Organisation, the first of which is to reduce inequalities in health by 25% by the year 2000. Let us hope that this is a serious commitment and that the government will put up money and encourage experiments to achieve the reduction.

Assistant editor, BMJ

¹ Department of Health and Social Security. Inequalities in health: report of a research working group. London: DHSS, 1980.

² Archbishop of Canterbury's Commission on Urban Priority Areas. Faith in the city: a call for action by church and nation. London: Church House Publishing, 1985.

by church and nation. London: Church House Publishing, 1985. 3 Dilnot A. Quoted in: Lipsey D. How much would it cost? Sunday Times 1985; Dec 8:17.