Changes in total death rate and in ischaemic heart disease death rate in interethnic South African populations, 1978 - 1989

A. R. P. WALKER,   A. ADAM,   H. G. V. KÜSTNER

Abstract Ischaemic heart disease (IHD) in prosperous Western populations rose markedly in the 1940s, peaked between 1970 and 1975 and then fell variably — by about 50% in the USA and Australia. Undoubtedly, decreases in serum cholesterol levels, in the incidence of hypertension and in smoking frequencies are largely responsible. In South Africa, in all population groups other than blacks, IHD rates rose analogously, with Asians and whites attaining very high rates. However, from 1978 to 1989, the total death rate among white males (per 100 000 world population) fell from 1 002 to 631 (37%), and the IHD mortality rate from 312 to 139 (56%). Rates for Asians were 1 306 - 1 130 (14%) and 355 - 226 (36%), respectively, and for coloureds 1 691 to 1 392 (18%) and 171 to 110 (36%). For blacks, the total mortality rate remained unchanged; IHD rates were low, but these data are unreliable. Percentage falls in the IHD rate exceed those in the total death rate, especially among whites, indicating true decreases in IHD rate. Understandably, the accuracy of the data, as with such data overseas, is questionable. Local falls are none the less in line with those noted in Western countries. Rates for whites remain very high, and are even higher for Asians. While knowledge of the reasons for the rises and falls in IHD rates remains incomplete, whites have none the less taken some preventive action, although Asians and coloureds apparently little.

S Afr Med J 1993; **83**: 602-605.

H istorically, coronary atherosclerosis was a disease of antiquity, evident in the thickening and calcification of coronary arteries in Egyptian mummies dating from about 1000 BC.[1] In about 800 BC, Indian physicians, who knew of the circulation of the blood, described anginal pain as 'a sense of constriction in the precordium, stitching pain and sensations of churning, or bursting or rubbing'.[2] In Greece, the Hippocratic school recognised angina pectoris.[1] Seneca, the Roman philosopher, in 65 AD described his own experience as follows: 'The attack is very short and like a storm. It usually ends within one hour . . . To have another malady is only to be sick; to have this is to be dying.'[1]

In the late 18th century, both Jenner (of smallpox fame) and Parry 'related angina pectoris to occlusive disease of the coronary arteries'.[3] Herrick's[4] classic article, published in 1912, helped bring coronary artery

Human Biochemistry Research Unit, Department of Tropical Diseases, University of the Witwatersrand and South African Institute for Medical Research, Johannesburg
A. R. P. WALKER, D.SC.

Directorate: Epidemiology, Department of National Health and Population Development, Pretoria
A. ADAM, B.SC., M.B. B.CH., M.F.G.P., D.T.M. & H., D.P.H.
H. G. V. KÜSTNER, B.SC., M.B. B.CH., M.D.

obstruction and myocardial infarction to the attention of the medical profession. Almost unbelievably, as late as 1912, it was stated that angina pectoris 'is a rare disease in hospitals; a case a month is about the average, even in the large metropolitan hospitals'.[5]

Since the 1940s and 1950s enormous, indeed catastrophic, increases took place in the incidence of ischaemic heart disease (IHD). Whereas previously it was responsible for less than 1% of all deaths, its incidence in many Western populations rose so rapidly that, until recently, it caused up to one-third of *all* deaths.[6] Thus, within the lifetime of many, IHD changed from a disease of negligible public health importance to the No. 1 killer.

Within the past 2 decades, however, there have been marked, although variable, falls in the IHD mortality rate in many Western populations. These have been maximal (approximately 50%) in the USA and Australia.[7-10] While reasons for the falls are not completely clear, decreases in serum cholesterol levels, in hypertension incidence and smoking frequencies are undoubtedly strongly implicated.[10-11] Simultaneously, there have also been some falls in total death rates.

In South Africa, much has been written on IHD mortality rates.[12-17] Information on IHD in all population groups has been forthcoming.[18-22] Studies have clearly indicated high rates in whites, very high rates in Asians, lower rates in coloureds and very low rates among blacks.

To learn to what extent the falls in IHD reported overseas have occurred in the South African populations, assessments have been made of age-standardised (world population) mortality rates, both total death rates and IHD rates in interethnic populations in 1978 and in 1989. Additionally, IHD mortality rates of population segments aged 30 - 69 years have been calculated to allow comparisons with rates in other populations.

Subjects and methods

Population data

Population estimates for 1978 and 1989 were obtained from the Directorate: Epidemiology of the Department of National Health and Population Development in Pretoria.

Registered deaths and mortality rates

Mortality rates from all causes and IHD for the years 1978 - 1989 were calculated from the number of deaths registered, which data were provided by the Central Statistical Services of Pretoria.[23] Data for the age standardisation of rates to world population were obtained from South African population denominators and from international standard population data.[24] The age-standardisation of rates for Western European populations was calculated in the manner described by Uemura and Pisa.[9]

Code definitions

For the period 1978 - 1989, the 9th revision of the *International Classification of Diseases*[25] (ICD 410 - 414)

was used. This includes acute myocardial infarction, other acute and subacute forms of IHD, old myocardial infarction, angina pectoris and other forms of chronic IHD.

Age group 30 - 69 years

To obtain the mortality rate for this truncated category, calculations were made of the rate for each 5-year age group. This was multiplied by the weight assigned to that age category to allow for standardisation with European populations. The products were then added; this yielded the mortality rate for the 30 - 69-year age group.

Results

In Table I, age-standardised mortality rates (world population) are given for total deaths and IHD deaths in South African populations in 1978 and 1989. Falls in both are marked although variable. The data on the black population are unfortunately unreliable. For the other population groups, percentage falls in total mortality rates, especially in respect of whites, are exceeded by percentage falls in mortality from IHD; i.e. there have been true falls in deaths from IHD. Among whites, falls are much the same in both sexes. Indian women are affected slightly more than men, but coloured men are affected more than women.

In Table II, data are given for age-standardised mortality rates from IHD in South African populations aged 30 - 69 years, in 1985 as well as in 1989; this permits comparison with rates reported for Western populations in 1985.[9] The diversity of the data on South African population groups will be noted. The extremely high rates for Asians are conspicuous, as are those for whites, which are still very high.

Discussion

There are numerous perplexities in IHD epidemiology.[26] Internationally, the mortality rate in Scotland is almost three times that in France.[9] The rate in the UK is double that in Switzerland. In Greece and Japan, rates are remarkably low.[9] None of the differences in IHD experience described is fully explained by differences in the incidence of the risk factors so extensively studied.[8,26,27] There must therefore be some powerful unknown factors in operation, aggravating or inhibiting the development of the disease.

The rises and falls in IHD rates should not be considered in isolation, as similar falls have taken place in stroke-related mortality.[28] Falls have also occurred in such contrasting diseases as stomach cancer,[29] appen-

dicitis,[30] dental caries,[31] and spina bifida.[32] A lower mortality rate from IHD should therefore be regarded as one of a number of declines in deaths from many important diseases, for reasons not wholly understood.

In almost all countries where the IHD mortality rate has fallen the data have been carefully examined with regard to underreporting, overreporting and other sources of error.[33,34] The conclusion has been reached that definite declines in IHD rates have taken place overseas.

While the local falls are also undeniable, their precise extent can only be gauged by investigating the clinical and pathological antecedents or a series of white, Asian and coloured patients, all certified as dying from IHD. In the case of the black population particularly, it is humbling that such an examination has not been carried out. The falls in the total death rates of whites agree with falls noted in other Western populations.[9]

TABLE II.
Age-standardised mortality rates from IHD in South African populations in 1985 and 1989 compared with rates in other countries in 1985;[9] rates per 100 000 European population aged 30 - 49 years

Country	Male		Female	
	1985	1989	1985	1989
South Africa				
Blacks	18	17	11	10
Asians	499	417	234	182
Coloureds	245	198	134	113
Whites	391	257	117	83
Northern Ireland	406		130	
Finland	390		79	
Scotland	398		142	
England	318		94	
USA	235		80	
Denmark	251		69	
Australia	247		76	
Canada	230		66	
Israel	183		73	
Switzerland	40		30	
France	94		20	
Japan	38		13	

Blacks

The mortality rate from IHD in this population, although low, is unreliable. However, the numbers of patients treated at large city hospitals are few. At King Edward VIII Hospital in Durban (2 500 beds), only 25 - 35 patients are admitted annually with IHD (personal communication — Y. K. Seedat). At Baragwanath Hospital (3 000 beds), the number is 70 - 80 patients annually (personal communication — P. Serelli). In

TABLE I.
Age-standardised mortality rates per 100 000 (world population, all ages) for total deaths and for IHD deaths in South African populations in 1978 and 1989, with percentage falls in mortality

	Blacks		Asians		Coloureds		Whites	
	M	F	M	F	M	F	M	F
1978								
Total mortality rate	1 072	661	1 306	980	1 691	1 108	1 002	615
IHD mortality rate	16	9	355	196	171	94	314	133
1989								
Total mortality rate	1 068	832	1 130	755	1 392	911	631	366
IHD mortality rate	11	8	226	113	110	71	139	55
% fall in total mortality rate	–	–	14	23	18	18	37	41
% fall in IHD mortality rate	–	–	36	43	36	25	56	59

each of these hospital's regions the population at risk is about 3 million. Most rural hospitals have not yet seen a case,[35] although 3 deaths from IHD in 1989 - 1990 were reported at Tshepong Hospital near Klerksdorp.[36] IHD appears relatively rare in blacks, and is certainly very rare in other African countries.[37] Interestingly, it remains very uncommon in Caribbean and African immigrants to the UK.[38] On the other hand, it is now a common disease in some West Indian countries[39] and in the USA.[40]

Asians

Rates in this population, for which most data originate from research done in India, have been high in all local studies.[12,14,15] It is relevant that no matter to which Western country Indians emigrate, in time they attain IHD rates which *exceed* those in the host country. This is the case in the UK,[41] and also in South Africa. The reasons are not clear, as orthodox risk factors do not appear to be excessively high.[19]

Coloureds

IHD was formerly uncommon, but the incidence has increased considerably.[14,15,20] As noted by Steyn et al.,[20] this population has a high level of risk factors.

Whites

The major fall in IHD rate is considerable, and as can be concluded from the CORIS study,[18] is due in part to changes in lifestyle, more especially to decreased smoking[42] and hypertension frequency. From his early observations, Wyndham[14,15] concluded that the mortality rate from IHD in young whites aged 25 - 44 years was the highest extant. In 1989, calculations indicate that for the 35 - 44 and 45 - 54-year age groups, mortality rates in white men were 48 and 176/100 000. In the UK, according to the Registrar-General's report for 1990, the corresponding figures for British men for 1988 were somewhat lower, 35 and 154/100 000.[43]

From comparisons made in Table II, there should be strong motivation to reduce the high IHD rate in the Asian and white populations, especially among those at high risk.

Measures to avoid IHD

General dietary measures for the avoidance of IHD, as well as diet-related cancers and other degenerative diseases, have been set out by numerous international nutritional bodies.[44-47] The most recent report calls for a doubling of bread consumption, principally wholemeal bread, a doubling of vegetable consumption and increased consumptions of potatoes and fruit, each by 50%.[46] The World Health Organisation also urges considerable increases in vegetable consumption; 400 g daily is recommended.[47] Nutrition is certainly a very important health regulating factor in the 'prudent' lifestyle. However, it is important to recognise that non-nutritional adverse factors, which include smoking, excessive alcohol consumption, physical inactivity and low socio-economic status can reduce, or even nullify, the beneficial effects of a 'prudent' diet.[6,11]

Outlook for the future

It is of course fervently hoped that the falls in IHD in our populations will continue, whatever the reasons.

With regard to individual efforts at avoidance — is the public listening and really responding? Although a decrease in energy intake has been urged for most Western populations, only marginal, if any, decreases are occurring.[48,49] In the USA[50] and other countries,[8] obesity is increasing, and despite a decade of recommendations, in prosperous European countries the amount of energy supplied by fat remains very high, about 40 - 45%.[51] It has fallen to about 36% in the USA. In most countries, there has been a reduced intake of saturated fat. (butter consumption has fallen considerably) and an increased intake of polyunsaturated oils, margarine and spreads.[48] This change is undoubtedly of importance. Reductions in serum cholesterol levels have been significant in some countries,[52] although not in others.[53] The consumption of fibre-containing foods remains low in most countries[48,54] and, as noted above, requires doubling.[46]

In most Western countries smoking frequency has fallen, although less so in women than men;[55] high blood pressure is now better controlled than before,[56] physical activity has increased very slightly, but only in certain segments of populations. Present-day white children are lazy.[57,58]

On the one hand, in *national* populations, insofar as preventive action has been taken, the outlook for the control of IHD and other lifestyle-related diseases is not propitious. However, with community studies, the results of interventions with regard to primary and secondary prevention of the disease give grounds for hope. Two studies illustrate the possibilities.

In the USA, 'to assess the combined influence of blood pressure (BP), serum cholesterol level and cigarette smoking on death from coronary heart disease (CHD) and to describe how these associations vary with age, data on those factors and on mortality for 316 099 men screened for the Multiple Risk Factor Intervention Trial (MRFIT) were examined. Vital status of participants has been determined after an average follow-up of 12 years . . . Smokers with serum cholesterol and systolic BP levels in the highest quintiles had CHD death rates that were approximately 20 times greater than non-smoking men with systolic BP and cholesterol levels in the lowest quintile. Systolic and diastolic BP, serum cholesterol level and cigarettes per day were significant predictors of death due to CHD in all age groups . . . . These results, together with the findings of clinical trials, offer strong support for intensified preventive efforts in all age groups.'[59]

In the UK, 'a randomised controlled trial was done to examine the effects of dietary intervention in the secondary prevention of myocardial infarction (MI). Two thousand and thirty-three men who had recovered from MI were allocated to receive or not to receive advice on each of three dietary factors: a reduction in fat intake and an increase in the ratio of polyunsaturated to saturated fat, an increase in fatty fish intake, and an increase in cereal fibre intake. The advice on fat was not associated with any difference in mortality, perhaps because it produced only a small reduction (3 - 4%) in serum cholesterol. The subjects advised to eat fatty fish had a 29% reduction in 2-year all-cause mortality compared with those not so advised. The 2-year incidence of reinfarction plus death from ischaemic heart disease was not significantly affected by any of the dietary regimens. A modest intake of fatty fish (two or three portions per week) may reduce mortality in men who have recovered from MI.'[60]

The results of these studies strengthen the need for people to adapt to the 'prudent' lifestyle measures now universally recommended by international health organisations.

REFERENCES

1. Brothwell D, Sandison AT. *Diseases in Antiquity. A Survey of Diseases, Injuries and Surgery of Early Populations.* Springfield, Ill.: Charles C. Thomas, 1967.
2. Mukerjee AB. Heart disease in India. *J Indian Med Assoc* 1975; **65:** 156-158.
3. Lie JT. Recognizing coronary heart disease. Selected historical vignettes from the period of William Harvey (1578 - 1657) to Adam Hammer (1818 - 1878). *Mayo Clin Proc* 1978; **53:** 811-817.
4. Herrick B. Clinical features of sudden obstruction of the coronary arteries. *JAMA* 1912; **59:** 2015-2020.
5. McCrae T. *Osler's Principles and Practices of Medicine.* London: Appleton, 1912; 836.
6. Pedoe HT. Coronary heart diseases. In: Miller DL, Farmer RDT, eds. *Epidemiology of Diseases.* Oxford: Blackwell, 1982.
7. Sytkowski PA, Kannel WB, D'Agostino RBD. Changes in risk factors and the decline in mortality from cardiovascular disease. *N Engl J Med* 1990; **322:** 1635-1641.
8. Leeder S, Gliksman M. Prospects for preventing heart disease. *BMJ* 1990; **301:** 1004-1005.
9. Uemura K, Pisa Z. Trends in cardiovascular disease mortality in industrialized countries since 1950. *World Health Stat Q* 1988; **41:** 155-178.
10. Ilsley C. Admissions to coronary care units. *NZ Med J* 1985; **98:** 380-382.
11. Tunstall-Pedoe H, Smith WCS. Cholesterol as a risk factor for coronary heart disease. *Br Med Bull* 1990; **46:** 1075-1087.
12. Walker ARP. Mortality from coronary heart disease and cerebral vascular disease in different racial populations in South Africa. *S Afr J* 1963; **37:** 1155-1159.
13. Shrire V. Heart disease in Southern Africa with special reference to ischaemic heart disease. *S Afr Med J* 1971; **45:** 634-644.
14. Wyndham CH. The epidemiology of cardiovascular disease in South Africa. *Medicine International* 1982: S811-S817.
15. Wyndham CH. Trends with time of cardiovascular mortality rates in the populations of the RSA for the period 1968 - 1977. *S Afr Med J* 1982; **61:** 917-983.
16. Duvenhage H, Sinclair GS, Küstner HGV. Deaths from ischaemic heart disease in white South Africans, 1963 - 1977. *S Afr Med J* 1982; **62:** 231-234.
17. Küstner HGV, Adam A. A further decline in the annual mortality rates from ischaemic heart disease in white South Africans, 1963 - 1988. *Epidemiol Comments* 1991; **18:** 156-168.
18. Rossouw JE, Jooste PL, Steenkamp HJ, Thompson ML, Jordaan PC, Swanepoel AS. Socio-economic status, risk factors and coronary heart disease: The CORIS baseline study. *S Afr Med J* 1990; **78:** 82-85.
19. Seedat YK, Mayet FGH, Khan S, Somers SR, Joubert G. Risk factors for coronary heart disease in the Indians of Durban. *S Afr Med J* 1990; **78:** 447-454.
20. Steyn K, Jooste PL, Langenhoven ML, *et al.* Coronary risk factors in the coloured population of the Cape Peninsula. *S Afr Med J* 1985; **67:** 619-625.
21. Steyn K, Jooste PL, Bourne L, *et al.* Risk factors for coronary heart disease in the black population of the Cape Peninsula. *S Afr Med J* 1991; **79:** 480-485.
22. Vermaak WJH, Ubbink JB, Delport R, Becker PJ, Bissbort SH, Ungerer JPJ. Ethnic immunity to coronary heart disease? *Atherosclerosis* 1991; **89:** 155-162.
23. Central Statistical Services. Reports on Deaths (Report Nos. 07-03-17 to 24; 07-05-01 to 09; 03-09-01 and 03-10-01). Pretoria: Government Printer, 1978 - 1989.
24. Doll R, Muir C, Waterhouse J. *Cancer Incidence in Five Continents.* Vol II. Berlin: Springer-Verlag, 1970; 338.
25. International Classification of Diseases (ICD). *Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death* (9th revision rubric E969). Geneva: World Health Organisation, 1977.
26. Walker ARP, Walker BF, Labadarios D. Perplexing changes and variations in the epidemiology of common diseases: cancer and coronary heart disease. *Int Clin Nutr Rev* 1991; **11:** 131-139.
27. Hart JT. Coronary prevention in Britain: action at last? *BMJ* 1987; **294:** 725-726.
28. Grotta JC. Current medical and surgical therapy for cerebrovascular disease. *N Engl J Med* 1987; **317:** 1505-1517.
29. Nobrega FT, Sedlack HD, Sedlack RE, Dockerty MB, Ilstrup DM, Kirland LT. A decline in a carcinoma of the stomach: a diagnostic artifact? *Mayo Clin Proc* 1983; **58:** 255-260.
30. Pokorny WJ. Appendicitis. In: Oski FA, De Angelis D, Feigin RD,

Warshaw JB, eds. *Principles and Practice.* Philadelphia: Lippincott and Co., 1990; 1738-1739.
31. Löe H. Dawes C. Dental science and dental health: now and a century hence (Guest editorial). *J Dent Res* 1988; **67:** 1054-1055.
32. Robinson R. Fewer neural tube defects: more Down's. *BMJ* 1991; **302:** 612.
33. Ueshima H, Tatara K, Asakura S. Declining mortality from ischaemic heart disease and changes in coronary risk factors in Japan, 1956 - 1980. *Am J Epidemiol* 1987; **125:** 62-72.
34. Hammer N, Ahlbom A. Recent trends in mortality from and incidence of myocardial infarction in Stockholm. *BMJ* 1987; **294:** 609-610.
35. Gilpin TP, Walker ARP, Walker BF, Evans JA. Admissions of rural black patients to Murchison Hospital, Port Shepstone, Natal: causes of admissions and prospects of improvements. *S Afr J Food Sci Nutr* 1989; **1:** 11-15.
36. Steenekamp JHB, Simson IW, Theron W. Cardiovascular causes of death at Tshepong Hospital in 1 year, 1989 - 1990. *S Afr Med J* 1992; **81:** 142-146.
37. Parkin DM. *Cancer Occurrence in Developing Countries* (IARC Scientific Publications No. 75) Lyons: International Agency for Research on Cancer, 1986.
38. Beevers DG, Cruickshank JK. Age, sex, ethnic origin and hospital admission for heart attack and stroke. *Postgrad Med J* 1981; **57:** 763-765.
39. Miller GJ. Some recent advances in non-communicable diseases in the tropics. Coronary heart disease and associated characteristics in a tropical developing community. *Trans Soc Trop Med Hyg* 1991; **85:** 332-335.
40. Watkins LO. Coronary heart disease and coronary risk factors in black populations in underdeveloped countries: The case for primordial prevention. *Am Heart J* 1984; **108:** 850-862.
41. McKeigue PM, Marmot MG. Mortality from coronary heart disease in Asian communities in London: *BMJ* 1988; **297:** 903.
42. Yach D, Townsend GS. Smoking and health in South Africa. *S Afr Med J* 1988; **73:** 391-399.
43. *Report of the Registrar General.* London: HMSO, 1990.
44. Koop E. *The Surgeon-General's Report on Nutrition and Health.* Washington, DC: Department of Health and Human Services, 1988.
45. Peterkin BB. Dietary guidelines for Americans. *J Am Diet Assoc* 1990; **90:** 1725-1727.
46. Bingham S. Dietary aspects of a health strategy for England. *BMJ* 1991; **303:** 353-355.
47. Report of a Study Group. *Diet, Nutrition and the Prevention of Chronic Diseases* (Tech Rep. Ser. No. 803). Geneva: World Health Organisation, 1991.
48. Ministry of Agriculture, Fisheries and Food. *Household Food Consumption and Expenditure, 1987* (Annual Report of the National Food Survey Committee). London: HMSO, 1988.
49. McBride G. US diet industry under fire. *BMJ* 1990; **300:** 1401-1402.
50. Shah M, Hannan PJ, Jeffrey RW. Secular trends in body mass index in the adult population of three communities from the upper mid-western part of the USA: the Minnesota Heart Health Program. *Int J Obes* 1991; **15:** 499-503.
51. O'Connor M. Europe and nutrition: prospects for public health. *BMJ* 1992; **304:** 178-180.
52. Burke GL, Sprafka JM, Folsom AR, *et al.* Trends in serum cholesterol levels from 1980 to 1987. Minnesota Heart Survey. *N Engl J Med* 1991; **324:** 941-946.
53. Dargie HJ. Scottish hearts but British habits. *BMJ* 1989; **299:** 1475-1476.
54. Block G, Lanza E. Dietary fiber sources in the United States by demographic group. *J Natl Cancer Inst* 1987; **79:** 83-91.
55. Wald N, Kiryluk S, Darby S, Pike M, Peto R, eds. *UK Smoking Statistics.* Oxford: Oxford University Press, 1988.
56. Foote A, Erfurt JC. The benefit to cost-ratio of work-site blood pressure control programs. *JAMA* 1991; **265:** 1283-1286.
57. Armstrong N, Balding J, Gentle P, Kirby B. Patterns of physical activity among 11 to 16 year old British children. *BMJ* 1990; **301:** 203-205.
58. Dyment PG, Goldberg B, Haefele SB, *et al.* Physical fitness and the schools. *Pediatrics* 1987; **80:** 449-450.
59. Neaton JD, Wentworth D. Serum cholesterol, blood pressure, cigarette smoking and death from coronary heart disease. *Arch Intern Med* 1992; **152:** 56-64.
60. Burr ML, Fehily AM, Gilbert JF, *et al.* Effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction: Diet and Reinfarction Trial (DART). *Lancet* 1989; **2:** 757-761.