Coronary Heart Disease and Physical Activity—A Fresh Look

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SUMMARY

Incidences of fatal coronary heart disease in White South African males increased significantly between 1951 and 1970. In those under 60 years of age, the incidence exceeds that in USA. Prevention lies in the early recognition of raised blood lipids and hypertension, and in stopping smoking. The role of physical inactivity is less certain. The physiological knowledge needed for determining the correct speed of walking and running is proposed.

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The rapid industrialisation of Southern Africa since World War II has thrown a tremendous burden upon the small White male population. From this section of the population have come the entrepreneurs in industry and commerce, the professionals, the scientists and technologists, and the administrators. Coronary heart disease (CHD) is taking a heavy toll of these men when they are at the height of their productivity and are in responsible positions. This contention is borne out in Table I which shows that in the 20-year-olds the main causes of death in White males in South Africa in 1970 were accidents (mainly on the road) and suicides. In the 40-year-olds CHD outstrips other causes of death, and among 50-year-olds it is the main cause of death. The last reliable determination of the incidence of fatal CHD in White males in South Africa was by Dr A. Adelstein in his 1963 paper.[1] He estimated the annual death rates in 5-year class intervals for the period 1954 - 1958.

Has the CHD problem in the White male population of South Africa remained static since Dr Adelstein's survey, or is it getting worse? I have calculated the incidences of death from CHD in 5-year class intervals of age for the 3 census periods, 1951, 1960 and 1970, and the results are given in Table II and Figs 1 and 2. This shows a clear trend towards a higher incidence of CHD for the period 1951 - 1970 at all ages, which becomes significant at 35 - 40 years of age. This trend is also discernible in the USA (Fig. 3), where the incidence of CHD for the years 1954 and 1967 is given.

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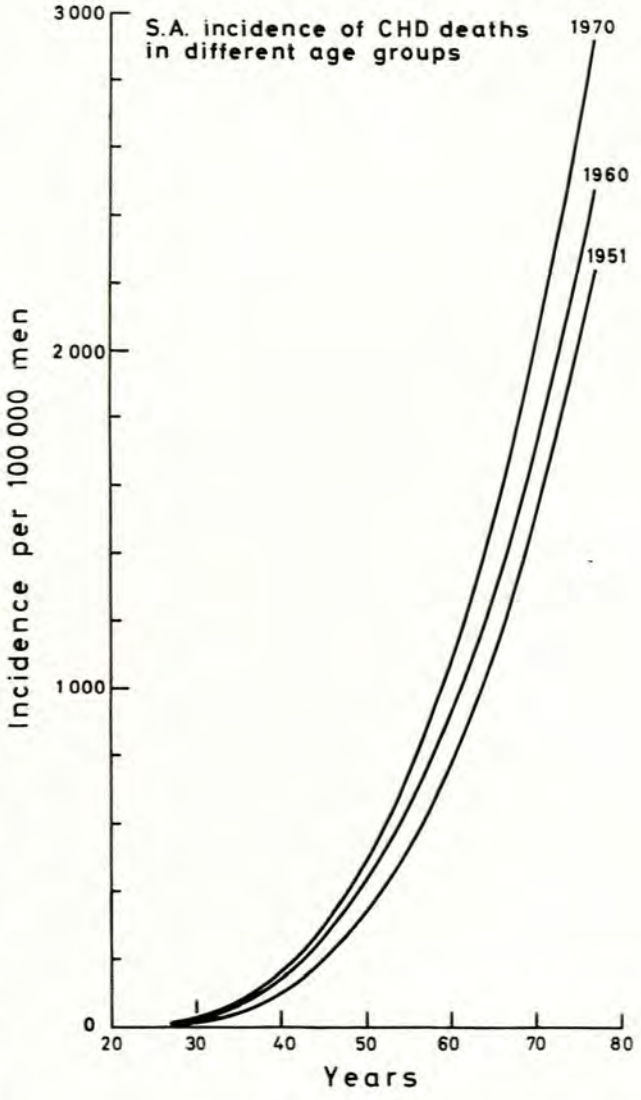

Fig. 1. See text.

How do the South African and USA figures compare? The incidences in 5-year class intervals for the USA for 1967 and for South African White males for 1970 are compared in Fig. 4. This shows that there is a higher incidence of deaths from CHD in the South African White male population under 50 years old, but that after the age of 60 years the incidence of deaths from CHD in the USA exceeds that in South Africa.

TABLE I. MAIN CAUSES OF DEATH IN WHITE MALES IN SOUTH AFRICA (1970)

Age group	Accidents	Suicide	CHD	Cancer		CVS
				Gut	Lung	
20 - 29	375	84	(21)	—	—	—
30 - 39	162	80	148	—	—	—
40 - 49	145	79	644	—	—	—
50 - 59	—	—	1 319	195	198	—
60 +			2 530	—	—	1 377

TABLE II. INCIDENCES OF FATAL CHD IN WHITE SOUTH AFRICAN MALES IN 1951, 1960 AND 1970

Class intervals of age	Pop. census	CHD cases (A81 420 - 422)				Incidences
1951	(White males)	1950	1951	1952	mean	per 100 000
20 - 24	104 064	4	3	4	4	4
25 - 29	98 703	7	7	4	6	6
30 - 34	96 995	18	20	22	20	21
35 - 39	96 380	54	57	50	55	57
40 - 44	92 142	114	126	101	114	123
45 - 49	73 073	194	188	196	194	266
50 - 54	54 891	222	228	262	237	432
55 - 59	49 326	314	328	358	333	675
60 - 64	39 881	327	380	417	375	939
65 - 69	31 642	374	432	450	419	1 322
70 - 74	24 520	377	399	455	410	1 673
75 - 79	15 290	302	339	398	346	2 263
		(A81 420 - 422)				
1960		1959	1960	1961	mean	
20 - 24	118 361	7	1	2	4	3
25 - 29	104 294	16	8	12	12	12
30 - 34	102 988	37	26	36	33	32
35 - 39	98 132	78	84	77	80	82
40 - 44	92 710	172	147	167	161	174
45 - 49	92 551	270	281	255	269	290
50 - 54	83 788	409	441	433	428	511
55 - 59	60 243	431	406	527	455	756
60 - 64	44 341	508	505	492	502	1 133
65 - 69	35 303	561	555	569	562	1 592
70 - 74	29 605	501	541	549	530	2 154
75 - 79	16 793	423	430	453	435	2 589
		Revised classif. 410 - 414				
1970		1969	1970	1971	mean	
20 - 24	161 650	11	7	11	10	6
25 - 29	146 111	15	14	17	15	10
30 - 34	130 100	49	49	40	46	35
35 - 39	114 520	115	99	110	108	94
40 - 44	104 460	237	263	230	243	233
45 - 49	100 320	361	381	296	346	345
50 - 54	87 180	515	544	484	515	591
55 - 59	81 540	674	779	664	706	866
60 - 64	67 740	883	922	855	887	1 314
65 - 69	43 090	703	763	845	770	1 751
70 - 74	27 750	650	698	641	663	2 385
75 - 79	17 700	508	529	524	522	2 949

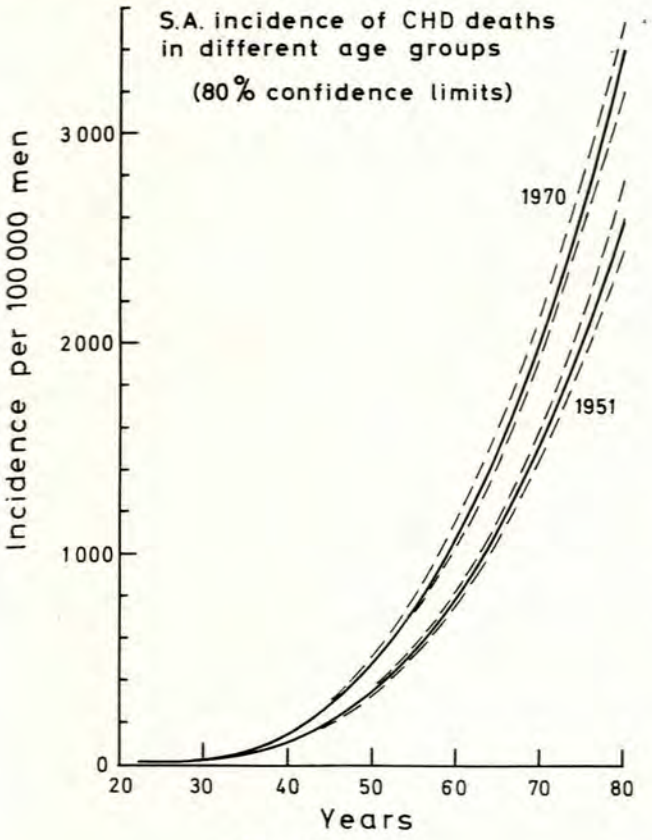

Fig. 2. See text.

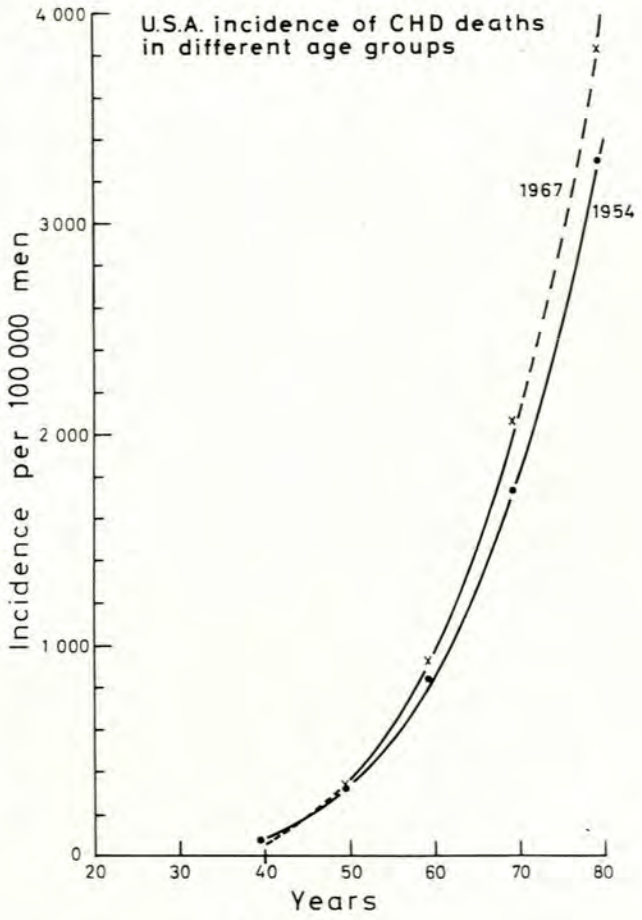

Fig. 3. See text.

There can be little doubt that in South Africa we have a CHD problem in White males in the 30 - 50 age group which is unparalleled elsewhere in the world. What can be done to reduce the death rate among White males from CHD?

Improved curative medicine, such as more intensive care units, undoubtedly has its place in reducing the death rate from CHD, but we are still left with the fact that one-third of the deaths occur before they can receive medical attention. The more logical approach is to prevent the disease. For prevention to be effective we need a sound strategy. This is the main lesson we have learnt from preventive medicine in eradicating epidemic diseases. Today we are in a fortunate position in this regard. A sound strategy demands a knowledge of the causative factors. To quote from Morris *et al.*[2] of the London busmen study fame, '. . . the dominant hope is that action in the stage of the precursor pathology may achieve primary prevention. Individuals shown to be susceptible will be identified and prophylactic measures directed towards them. Prevention will be translated into the clinical field.'

Much could be achieved, following Morris *et al.*'s[2] suggestion, if all White males over the age of 40 years were examined annually to detect the first signs of precursor pathology and immediate steps were taken to either halt the progress of the disease or reverse the processes along the following lines: the planning of the diet and/or the use of drugs to reduce blood lipids in patients with raised serum cholesterol or triglyceride levels; treatment of patients with diastolic blood pressure maintained in excess of 95 mmHg or systolic blood pressure above 160 mmHg; and stopping the smoking of cigarettes, especially if one or both of the above precursor pathologies are present.

Few clinicians would disagree about the role of the above factors in the causation of CHD, and with the need for their early recognition and treatment in preventing CHD.

The role of physical inactivity in the causation of CHD and of regular exercise in preventing it is more open to debate. In 1953 Morris *et al.*[3] in a study of London busmen concluded that 'men in physically active jobs (bus conductors) have a lower incidence of CHD than men in physically inactive jobs (bus drivers) . . . more important, the disease is not so severe in physically active workers, tending to have a smaller case fatality and a lower early mortality rate.' Morris *et al.*'s[3] conclusions have support in the studies of Breslow and Beull[4] in California, Taylor *et al.*[5] on 2 groups of railway workers, Kahn's study[6] of postmen and clerks in Washington, and Frank *et al.*'s study[7] of 11 000 persons in greater New York. Frank *et al.*[7] used a physical activity questionnaire

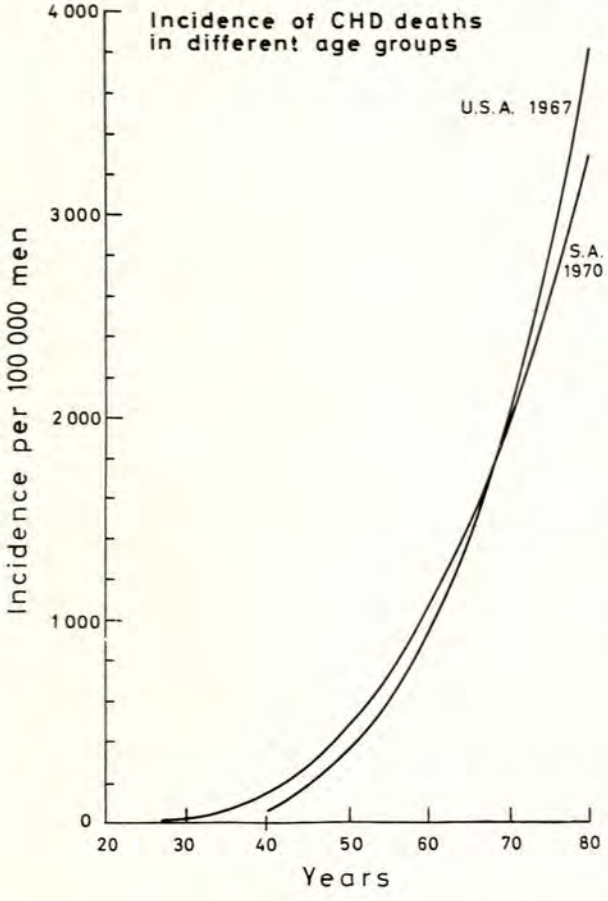

Fig. 4. See text.

and state that inquiry about the customary physical activity on and off the job permitted the delineation of a group of least active men who were more liable to experience a severe episode and die within 4 weeks of its onset from men who were relatively more active . . . clearly mortality in the least active men was three times that of the group of most active men . . . physical activity off the job was found to play an important role in the relationship.

There is strong support for Frank *et al.*'s[7] conclusions in Morris *et al.*'s[8] 1973 paper. They studied some 17 000 male executive grade civil servants aged 40 - 64 years in the UK, getting them to record their physical activities on Fridays and Saturdays. The questionnaire drew particular attention to vigorous physical activities (graded as an energy expenditure of 30 kJ/min or an oxygen consumption of 1,5 litres/min) which included swimming, brisk walking, jogging, heavy digging in gardens, etc. Such physical exercise could be expected to stress the cardiorespiratory system. There were 232 episodes of CHD in the period of the study, and these cases were matched with 464 men from the 17 000 population in regard to age, etc. During the 2 sample days, 11% of the men who developed CHD reported vigorous exercise compared with 26% of the controls. In men recording vigorous exercise the relative risk of developing CHD was about one-third of that in comparable men who did not exercise. Light exercise did not protect against CHD. Morris *et al.*[3] sum up as follows, habitual vigorous exercise during leisure time reduces the incidence of CHD in middle age among male sedentary workers. Vigorous activities which are normal for such men are sufficient. Training of the heart and cardiovascular system is one of the mechanisms of protection against common risk factors and the disease.

It is unfortunate that neither Frank *et al.*[7] nor Morris *et al.*[8] subjected subsamples of the vigorously exercising and the sedentary groups to a physiological test to measure their capacities for endurance exercise. Significant differences between the 2 groups in maximum oxygen intakes, VO_{2max}, the physiologist's measure of the individual's capacity for endurance exercise, would have been conclusive evidence of real differences between the 2 groups in their state of physical conditioning.

There are other authors who are not so convinced about the preventive effect of exercise. Fox and Paul[9] for example, cite Chapman *et al.*'s[10] prospective study of 955 male civil servants, in which there was, if anything, a higher rate of CHD in the physically active men than in the sedentary group. Paul also draws attention to the selective factor in Morris *et al.*'s[8] subjects. The conductors were lighter, had smaller skinfold thicknesses, lower serum cholesterols and lower blood pressures than the drivers. Similar selective factors could have been present in Taylor *et al.*'s train drivers and conductors, and in Frank *et al.*'s[7] postal delivery men and postal clerks.

It is also not known what pathophysiological mechanisms are involved in the protection against CHD given by vigorous exercise. Claims have been made that coronary artery collaterals proliferate,[11] that blood pressure is lowered and that serum cholesterol is decreased[12,13] as a result of a physical conditioning programme. These claims have been disputed.[9] One reason for the lack of unequivocal evidence of a halt of the precursor pathology, or of its reversal, is that the physical conditioning programmes have differed widely in their intensity of exercise, and many have been too mild to be effective.

Does the available evidence justify the large-scale application of physical conditioning in the prevention and therapy of CHD? Here I can do no better than quote from the closing remarks of Professor A. Tybjerg Hansen of the University Hospital in Copenhagen, at a Symposium in Copenhagen in September 1970 on 'Coronary heart disease and physical activity':[14]

'Strictly speaking, in the traditional exact scientific sense, the answer is no, and our conference has not changed that situation in spite of many fine scientific communications. The answer is no because the pure irrefutable laboratory type of experiment in which exercise can be shown to prevent CHD either by itself or by influencing other risk factors, does not exist and probably never will.

'In a way I am not quite satisfied with the answer nor am I satisfied with the question. I shall therefore allow myself to rephrase the question in such a way that it becomes pertinent to our present situation and the way which it will actually be asked.

'I would like to word the question as follows: Should the medical profession encourage the public—patients and others—to exercise more than they do? If so, should it do so for the purpose of preventing heart attacks, should it say so, and finally should it advise as to which kind of exercise people should engage in? To all 3 sections of the question I think the answer is yes, but further elaboration is necessary.

'First, there is no doubt that modern life can be lived without much exercise. It is also true that we think the increase in coronary heart disease has something to do with modern life as compared with life in former times. Lack of exercise, therefore, falls into the same group of habits of modern life that are under suspicion, namely, our food and smoking habits, which we would like to change if it is possible without incurring other ill effects. The experience, expressed both in the literature and at our conference, tends to show beyond doubt that certain types of exercise may be advocated without risk.

'As regards normal people, there are no reservations apart from the usual caution for avoiding sore legs, etc. In respect of patients who have suffered heart attacks, their condition must be individually considered, and their work capacity tested at the beginning of training, and be medically supervised for some time in such a manner that necessary steps can be taken in the case of serious symptoms, particularly serious arrhythmias.

'The kind of exercise I am talking about is the type in which aerobic work is performed by exercising to the limit or almost to the limit of the capacity for short periods, but repeated relatively often. The exercise should include large muscle masses. I think we can agree that there is no better fulfilment of these requirements than walking, jogging and running. These activities can be executed without any paraphernalia, and can actually be included in the pattern of everyday life. Dramatic and large scale arrangements are not necessary, but it might be a good idea to organise training sessions, for instance, in schools after school hours, to teach interested people the elementary rules of training so that they can continue on their own. For the future generation it is advisable for schools to teach the principles of physical training, dealt with here, in the hope of establishing lifelong habits in agreement with the contention that any possible beneficial preventative effect of training is only obtained if physical exercise is maintained.

'As regards the amount of exercise, there seems to be agreement about the minimum at the present time: half an hour of exercise at the submaximal level or a little less, 2 or 3 times a week, supplemented with running or jogging, using the stairs instead of the elevator, etc. Any other equivalent forms of exercise than the ones mentioned may of course be substituted. The recommendation from the medical profession of including exercise in everyday life may restore running to a respectable place in our behaviour pattern.'

I entirely agree with Professor Hansen that walking and running or jogging are ideal forms of exercise because they can be so easily controlled and regulated, they can be carried out almost anywhere, and they require no elaborate apparatus, merely a running place and a stopwatch. I disagree with his proposals with regard to how often and how much. The weekend game of golf is neither sufficiently frequent nor sufficiently vigorous to stress the cardiorespiratory system. In my view, regular, daily exercise is needed.

The question of how much exercise, and how strenuous it must be, is a much more vexatious one. In my experience of exercise physiology I am certain that endurance training, i.e. at 70 - 80% of maximum oxygen intake (which raises the heart rate to about 150 beats/min), cannot be achieved in less than 30 min of exercise at least 4 times per week. There should also be short bursts of running at close to maximum oxygen intake (which should raise heart rate to close to 180 - 190 beats/min). Maximum effort of this sort should only be done by subjects who have had a month or more of regular endurance training. It will be clear that the drawing up of a physical conditioning programme can only be done on an individual basis and should only be done by some skilled in exercise physiology, if the programme is to be both safe and satisfactory. I must stress the fact that few physical education graduates, on the present-day training in South Africa, qualify in this regard.

In drawing up the physical conditioning programme two facts are essential. One is an estimate of the individual's capacity for endurance exercise. This is measured by the physiologist in terms of the maximum oxygen intake or maximum aerobic power. The second fact needed is an estimate of the rate of oxygen consumption when walking or running at different speeds. With these two facts available, speeds of walking or running can be chosen in relation to the individual's maximum oxygen intake, so that he can carry out 30 min of exercise which stresses his cardiorespiratory system adequately, but does not overstress it. The importance of this assessment in planning an exercise programme was brought home to me at a symposium I attended a few years ago in Israel on this subject. The USA delegate reported a number of deaths in men over 50 while jogging, and attributed their deaths to the fact that they had not sought prior expert advice on the level of exercise which they could safely undertake.

A further safeguard would therefore be wise in the over 40-year-old male taking up jogging for the first time or after a long inactive period. It is an exercise ECG at the same level of oxygen consumption as he would be advised to jog at. If any evidence of cardiac ischaemia is revealed, then the level of exercise would need to be drastically revised.

MEASUREMENT OF CAPACITY FOR ENDURANCE EXERCISE

The physiologist measures the capacity for endurance exercise in terms of the individual's maximum oxygen intake or maximum aerobic power.

In the laboratory this measurement is made by having the subject either run on a treadmill or pedal a bicycle ergometer at a number of different workloads of increasing intensity. The rate of oxygen consumption is measured at each of these workloads, and it is found that at submaxi-

mal effort oxygen consumption increases as a linear function of work rate. However, as the work load is increased towards maximum effort, a point is reached at which the individual is able to work a little harder on the bicycle ergometer (or run a little faster on the treadmill), but he is unable to increase the rate of oxygen intake because he has reached the maximum rate of oxygen intake. The extra physical effort above this point is carried out on anaerobic metabolism and this is indicated by a sharp increase in the serum concentrations of the products of anaerobic metabolism, lactic and pyruvic acids.

There are three main physiological parameters which set the limit to the maximum oxygen intake. They are, firstly, the capacity of the heart as a pump to circulate blood from the lungs, where oxygen is taken up by the blood, to muscle capillaries where the oxygen is transferred to working muscle. Secondly, the lungs also play a part in terms of the maximum volume of air which can be ventilated through the lungs and the barrier, due to the alveoli walls, to the diffusion of oxygen from alveoli to blood in lung capillaries. The third factor is rate of diffusion of oxygen from capillaries into muscle cells. It is thought that at sea-level the most important parameter limiting maximum oxygen intake is the capacity of the heart to pump blood from lungs to muscle capillaries. In athletes cardiac outputs of 30 litres/min have been recorded at maximum effort. However, as we ascend in altitude it is the ability of the lungs to transfer oxygen into lung capillaries which is the limiting factor until at the top of Mt Everest, even with maximum respiratory ventilation, only enough oxygen gets to working muscles for very slow climbing indeed.

The direct measurement of maximum oxygen intake by the above procedures is time-consuming and requires costly apparatus and skilled technical staff. It is clearly not a procedure which can be used by the medical practitioner. There has in consequence been a search for a shorter and simpler method. Fortunately the maximum oxygen intake can be assessed by indirect methods which are not as accurate but can be carried out with simple apparatus. One is a simple step-test and the only equipment needed is an adjustable height bench, a bathroom scale, a metronome and a stop-watch. The metronome is set to 24 beats/min, the subject is weighed, and the height of the bench is set so that at 24 step-ups/min the subject works at 2 400 ft lb/min. Thus a 200-lb man would step a height of 6 inches and a 100-lb man a height of 12 inches. The rate of oxygen consumption for this work rate is 1,32 litres/min. The heart rate is measured at 10 - 15 min from the start of exercise. This indirect method of assessing maximum oxygen intake is dependent upon the fortunate physiological finding that heart rate and oxygen consumption both increase with increasing effort and both reach maximum values at approximately the same workload. Thus heart rate and oxygen consumption are linearly related up to maximum effort.[15,16] Because of this fortunate relationship heart rate can be measured at one work rate of which the oxygen consumption is known, and a straight line can be drawn to join this heart rate with a common heart rate of 70 beats/min for a resting oxygen consumption of 0,25 litres/min. Extrapolation of this line, as in Fig. 5, to 185 beats/min, the population mean maximum heart rate, gives a rough estimate of maximum oxygen intake. In a recent comparison of maximum oxygen intakes, measured on 25 members of a laboratory staff by the treadmill test and the step test, a correlation coefficient of 0,88 was found.[17]

The other indirect method which has come into prominence is that put forward by Dr Kenneth Cooper, the author of the popular publication entitled *Aerobics*.[18,19] It is the distance a man can walk or run in 12 min. Cooper found a correlation coefficient of 0,89 between

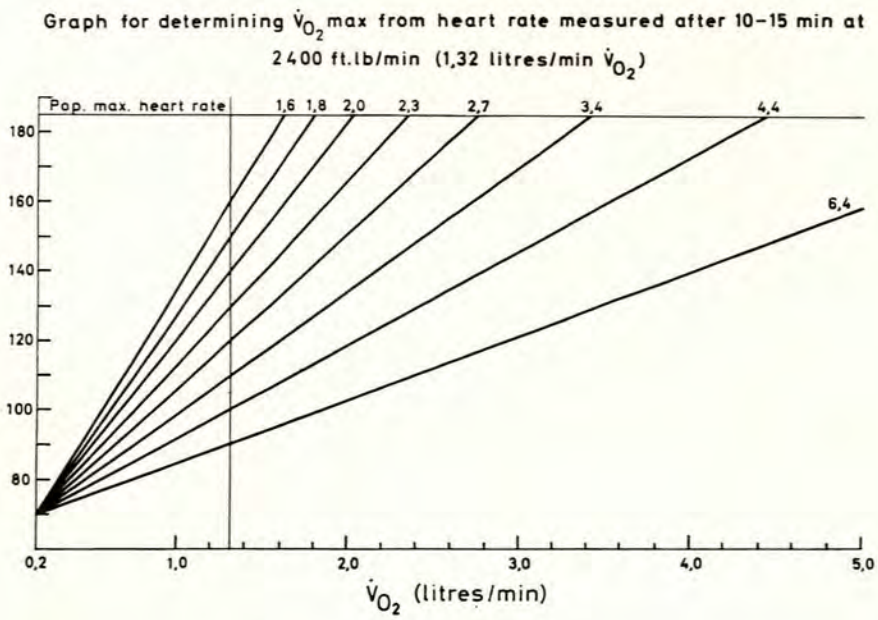

Fig. 5. See text.

the distance run and maximum oxygen intake measured on the treadmill. He had 115 subjects between the ages of 17 and 52 years, and weighing between 52 and 122 kg. We made a similar comparison on 25 members of my laboratory staff, who varied in age between 17 and 54 years and weighed between 60 and 110 kg. The correlation coefficient was even higher than Cooper's, being 0,94. I am confident, therefore, that provided the subjects are well motivated, the Cooper 12-min run gives a very good estimate of the maximum oxygen intake. Values of maximum oxygen intake can be determined roughly from Cooper's Table as follows:

TABLE III. VO_{2max} AND FITNESS LEVEL DETERMINED FROM DISTANCE RUN IN 12 MIN

Distance run in 12 min (miles)	Maximum oxygen intake ml/kg/min	Fitness level
Less than 1,0	**Less than 25,0**	**Very poor**
1,0 - 1,24	**25,0 - 33,7**	**Poor**
1,25 - 1,49	**33,8 - 42,5**	**Fair**
1,50 - 1,74	**42,6 - 51,5**	**Good**
1,75 & more	**51,6 & more**	**Excellent**

Cooper's 12-min walk or run test can be used safely in the under 30s to predict maximum oxygen intake. Its use in the over 40s should be conditional upon the subject being passed as fit by a medical practitioner, especially the overweight and unfit individual starting an exercise programme after a long period of physical inactivity. Where possible an exercise ECG should be done. It should be obligatory in the over 50s. If there is any doubt about the cardiorespiratory fitness of the subject it is preferable to use the step test. The work load in the step test is relatively mild and the heart rate and ECG can be monitored from the start of the exercise.

In interpreting the results of the Cooper test, two reservations should be borne in mind. One is that at our altitude of 1 876 m above sea-level, 10% should be added to the values obtained. The second is that an adjustment must be made for age. The various class intervals for the various states of physical fitness given in Cooper's Table should be adjusted downwards by 10% for every 10 years above 40 years. Thus a man of 55 years who lives in Johannesburg would be judged to be in a good state of physical fitness if he could run between 1,25 and 1,50 miles in 12 min and his maximum oxygen intake was therefore between 34 and 42 ml/kg/min.

Parameters of VO_{2max}

Body size and composition, age, sex, state of health and nutrition, state of physical activity and athletic ability, and altitude are all parameters of VO_{2max}. They have been dealt with in detail in a recent publication from this laboratory.[20]

In brief summary, a study from this laboratory of 80 fit, young army recruits, showed that gross body mass, fat-free mass and stature were all significantly correlated with VO_{2max}. However, when we separated the effects of these parameters, by the use of partial correlation coefficients, gross body mass was shown to be the main determinant. Multiple variance analysis indicated that 70% of the differences between the 80 army recruits in VO_{2max} was due to differences in body mass. Differences in height only accounted for a further 1% of the differences in VO_{2max}. Studies in the USA on samples of men with a much wider range of fat-free mass show that fat-free mass in such populations is a more important determinant of VO_{2max} than gross body mass. The important implication of this result for our present purposes, however, is that comparison of VO_{2max} of individuals, or of groups of men, of different body mass should be made in terms of VO_{2max} per unit body mass, i.e. ml/kg/min.

Age has a significant effect on VO_{2max}. It is highest in the 20 - 30 years age group, and falls off at roughly 10% per decade after the age of 30 years. Sex is another important determinant. VO_{2max} is approximately 20% lower in females than males in the same age group. Altitude also affects VO_{2max}, there being a 10% decrease in VO_{2max} per 2 000 m increase in altitude above sea-level up to about 6 000 m.

Athletic ability and physical activity markedly affect VO_{2max}. As Astrand, one of the world's foremost exercise physiologists, puts it, 'if you want to be an Olympic athlete you must choose your parents carefully'. World-class endurance athletes have VO_{2max} of 70 - 80 ml/kg/min, measured at sea-level (the values of South African international athletes measured in Johannesburg, at 2 000 m above sea-level, ranged from 62 - 70 ml/kg/min). Active men have VO_{2max} of 50 - 60 ml/kg/min and sedentary men are around 40 ml/kg/min.

Prolonged bedrest of 2 - 3 weeks causes a fall in VO_{2max} of about 25%. Chronic diseases of the cardiorespiratory system decrease VO_{2max} markedly. The state of nutrition also affects VO_{2max}. A sample of Black mine workers showed an increase in gross body mass from 55,2 to 58,4 kg over the period of a month on the mine compound diet of 16 000 kJ/man/day. VO_{2max} increased from 2,32 to 2,79 litres/min, or 42,1 to 49,0 ml/kg/min, indicating that not only had there been an increase in fat-free mass but cardiorespiratory fitness had also improved. Physical conditioning programmes cannot be expected to effect an increase in VO_{2max} of more than 10 - 20%. An ordinary individual with a VO_{2max} of 48 ml/kg/min cannot be made into a world-class athlete, no matter how hard he trains.

Energy Costs of Walking and Running at Different Speeds on the Flat

It is just as important to know the energy costs of walking and running at different speeds as it is to know the maximum oxygen intake, in advising upon the speeds at which an individual can walk or jog with safety, but also stress the cardiorespiratory system adequately. It is well established in exercise physiology that gross body mass is a most important determinant of the rate of energy expenditure of individuals walking at different

speeds, but there is no easily available information in the medicophysiological literature with which a medical practitioner can predict the oxygen intakes of individuals of different mass when they either walk or run at different speeds.

Recently this laboratory published[21] the results of a study on 25 members of the laboratory staff, in which oxygen intakes were measured when they walked at 3, 4 and 5 mph (4,8, 6,3 and 8,0 km/h) and when they ran at 6, 7 and 8 mph (9,7, 11,3 and 12,9 km/h). The subjects varied in mass from 132 to 247 lb. (60 to 110 kg). Because some of the heavy men were very unfit it seems probable that they reached the limit of their maximum oxygen intakes at the higher speeds of running, and that the oxygen intakes of the heavier men were probably underestimated. A further, more detailed study was therefore undertaken on 6 men, varying in mass from 62,8 to 102,1 kg, in which 3 oxygen intakes were measured at 2, 3, 4 and 5 mph (3,2 - 8,0 km/h) while walking, and at 5, 6, 7 and 8 mph (8,0 to 12,9 km/h) while running.[22]

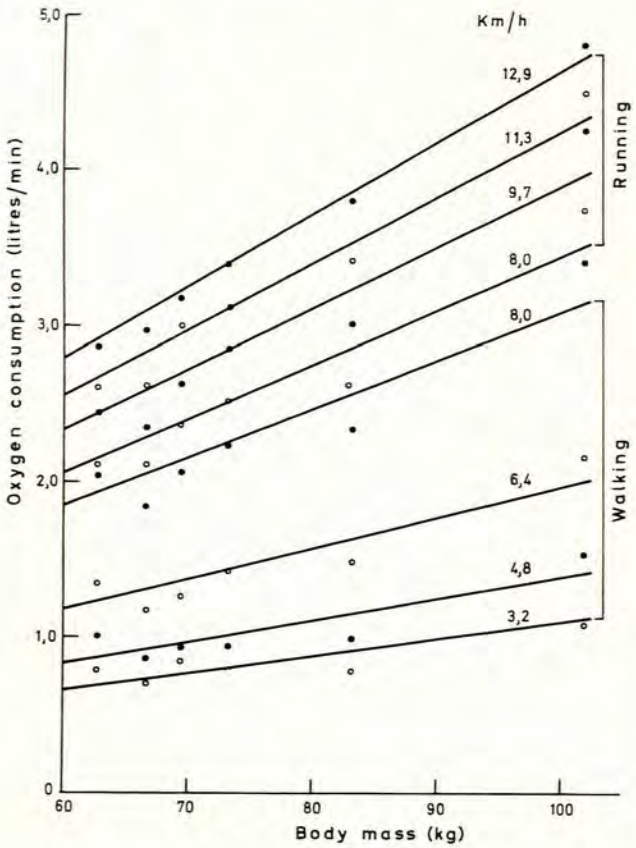

Fig. 6. See text.

The regression lines fitted to plots of oxygen consumption against gross body mass of the 6 subjects are given in Fig. 6 for each of the 4 speeds of walking and the 4 speeds of running. The correlation coefficient for walking ranged from 0,85 to 0,95 and from 0,98 to 0,99 for running. This shows that under carefully controlled experimental conditions body mass accounts for 90% of the variance in oxygen intake during walking, and for 98% during running. Further analysis shows that stature also has a small but significant effect, accounting for 2% of the variance. This indicates that, for two men of the same mass, the taller one has the lower oxygen intake.

Another figure was drawn from these data which is probably easier for medical practitioners to use for predicting the oxygen intake of men of different mass either walking or running at different speeds. Fig. 7 gives the curves of oxygen intake against speed of walking or running for men of different body mass in 10-kg class intervals, from 60 to 110 kg.

Fig. 7 shows that for walking, oxygen intake increases in a highly non-linear fashion over the range of speeds of walking investigated. For running, oxygen intake increases linearly with increase in speed. The shape of the curves for walking are much steeper than for running. It can also be noted from Fig. 7 that the oxygen consumption for walking at 5 mph is lower than for running, but extrapolation of the walking curves to 6 mph indicates that the predicted oxygen intake for walking at 6 mph would be much higher than for running. The speed of 5 mph is apparently a critical one, and men should be advised to jog rather than walk if they wish to exceed 5 mph in a physical conditioning programme.

Oxygen consumption can also be calculated, for those who prefer to use mathematical equations rather than graphs, from equations which my laboratory published recently.[22] They are based upon the curves given above.

Walking: $VO_2 = 0{,}00599V + 0{,}000366MV^2$

Running: $VO_2 = -0{,}419 + 0{,}03257M + 0{,}000117MV^2$

where VO_2 is oxygen consumption in litres/min,
M is mass in kilograms, and
V is speed of walking or running in kilometres per hour.

Safe Speeds for Walking or Running for Daily Exercise

The aim is to encourage the subject to exercise at about 80% of maximum oxygen intake for 30 min each day. This level of exercise is strenuous enough to strain the cardiorespiratory organs, and to produce a mild degree of anaerobic metabolism in most subjects. The heart rate should not be below 140 or above 160 beats/min if the safe speed of walking or running has been estimated correctly.

The procedure for determining the correct speed of walking or running is to take the measurement of the individual's maximum oxygen intake (estimated either from the step test or from Cooper's 12-min walk or run) and determine the oxygen consumption at 80% of maximum. With this estimate of the safe level of oxygen consumption, one looks up the rate of walking or running which will give this safe rate of oxygen consumption for the individual's body mass in Fig. 7. Examples are taken from the data on the 25 subjects, in the experiment referred to earlier, to illustrate how the maximum oxygen intake and Fig. 7 should be used in deciding upon the correct speed for walking or jogging (Table IV).

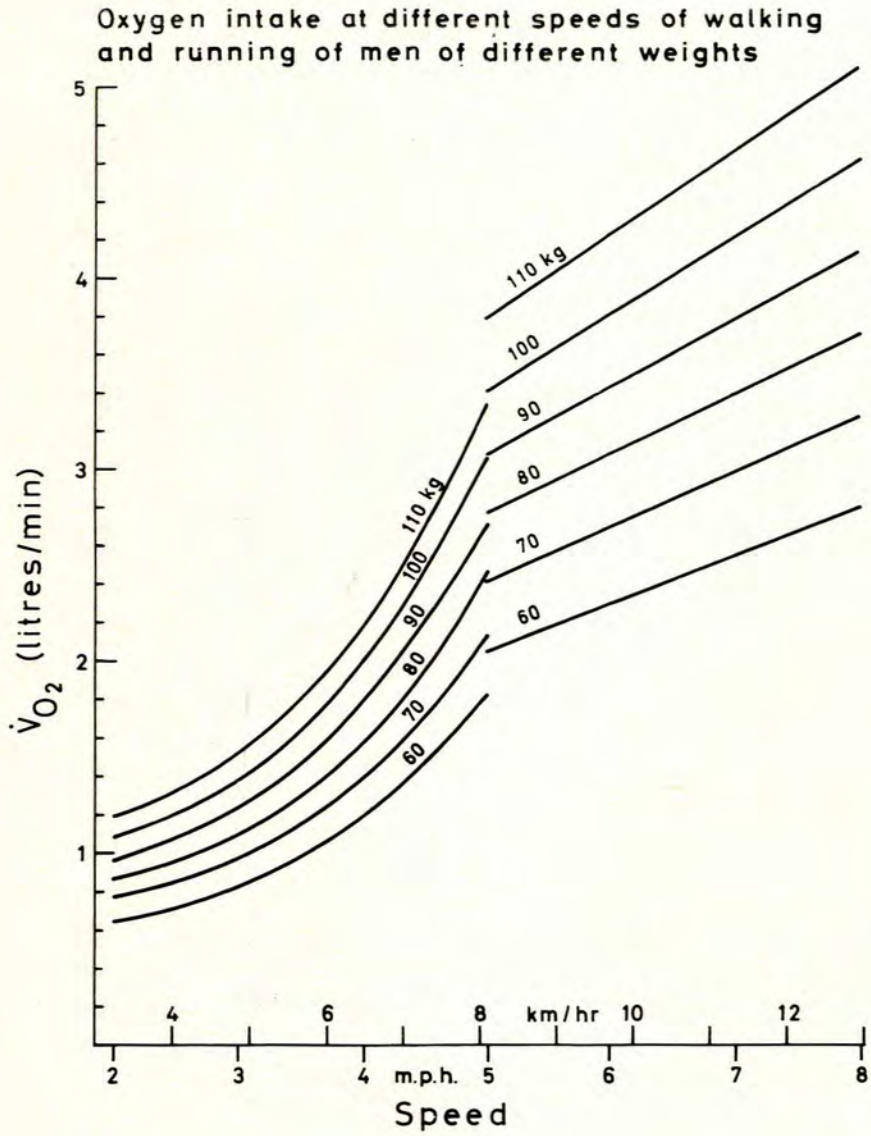

Fig. 7. See text.

TABLE IV. EXAMPLES OF SAFE SPEEDS FOR WALKING OR RUNNING AT 80% OF VO_{2max}

Age (yr)	Mass (kg)	State of fitness	Max. l/min	Oxygen ml/kg/min	80% max.	Speeds km/h	Speeds mph
24	85,9	Very unfit	2,35	27,5	1,9	7,1	4,5 (W)
32	111,7	Very unfit	3,50	31,2	2,8	7,1	4,5 (W)
31	110,6	Unfit	3,85	34,7	3,1	8,1	5,0 (W)
54	90,6	Fit	3,70	40,7	3,0	8,1	5,0 (W or R)
28	70,8	Very fit	3,30	46,5	2,7	9,6	6,0 (R)
27	61,7	Athlete	3,70	59,4	3,0	12,9	8,0 (R)

Table IV illustrates a point which is not generally realised. It is that overweight and very unfit young men should not, in the initial phase of a programme of physical conditioning, exceed a walking speed of 4,5 mph (7,1 km/h). On the other hand a relatively fit individual in his 50s can safely walk or jog at 5 mph (8,1 km/h), and even jog at 6 mph (9,7 km/h) without danger.

As regards physical conditioning programmes my advice is to use Kenneth Cooper's *New Aerobics*.[23] I do not think it can be improved upon. He is a medical physiologist and has built into his proposed programmes the necessary safeguards and checks which the physical educationalists are not able to carry out. I am very much in favour of his emphasis on jogging as the ideal physical conditioning exercise and of the careful programmes he gives for improving physical fitness.

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