LEADING ARTICLE

EXERCISE AND LIVE LONGER?

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Readers of this journal will almost all be sport and therefore exercise devotees. As such most of you will be believers in 'Loafers heart' (that physical sloth and inertia encourage heart disease) and would no doubt like to believe that regular exercise prolongs life by preventing or delaying the onset of coronary artery disease, the single most important cause of death in civilised man..

The purpose of this article, leading as it does the enthusiasts to follow, is to question some of the assumptions on which this belief is based, not because I think that it is wrong, but because more evidence is needed and widespread naive acceptance of this belief could be counter productive in the long run.

Prevention of Ischaemic Heart Disease

The prevention of ischaemic heart disease can be divided into primary prevention (preventing the first coronary event) and secondary prevention (delaying or preventing a recurrence). It can also involve the investigation of the multiple aetiological factors that place a person at risk from coronary artery disease.

Risk factors

These risk factors, such as smoking, hypertension, diabetes, hyperlipidaemias (excess of certain fats in the blood) have been shown to act independently of each other. Each, if operative, increases the individual's chances of suffering from a coronary event. Thus a heavy smoker with hypertension is more likely to have a coronary attack than someone who is only a heavy smoker and has no other risk factors operating. The risk factors can be scaled and added so that each individual's risk can be assessed on a statistical basis. Thus each individual's risk of suffering from a coronary event (infarct, angina or sudden death) over the next few years can be calculated as a ratio of the normal risk in the population. The absolute cause of ischaemic heart disease is not known. Very high risk subjects may defy the odds for years and many patients with myocardial infarcts have very low risk factors, but this is in the nature of the statistics of large numbers.

Since there are many risk factors, including sex, age, blood lipid levels, blood pressure, glucose tolerance, as well as racial, geographical, and dietary differences in the incidence of coronary events, isolation of additional factors, independent of the established ones, involves the study of very large numbers of subjects over many years. The reason for this is that even in high risk subjects the incidence rate is only about 2% per annum so that demonstrating even a 50% reduction from this level involves comparing 2% with 1%.

Exercise as a risk factor

Where does exercise and physical sloth fit into this scheme? Much of the evidence for the benefits of exercise is on the anecdotal level. "Mr Marathon's" post mortem (Currens and White) showing large clean coronary arteries, studies of 102 year olds who run 3 miles a day, studies of middle aged skiers and runners and of remote Indian tribes who run half the day and never have coronary artery disease, all suffer from lack of adequate control data, which in this branch of epidemiology requires investigation of the other risk factors. Most of these studies are on self-selected groups or individuals, who may well have extremely low risk. They may well be nonsmokers, have low levels of lipids in the blood as a result of diet fads and a small amount of body fat, and since success in endurance sports may well be related to a hereditary component to the maximum oxygen uptake, they may well belong to a low risk sub-group whose risk is low for a yet undiscovered, independent reason.

These studies on elite groups may be valuable in disproving the idea that damage to the heart results from excessive athletic competition, but cannot be used to claim that exercise would have a preventative effect on the population as a whole.

Population studies by Morris and his associates have shown that occupational physical exertion is associated with decreased risk of coronary events. There is some dispute about this as Finnish lumberjacks have a relatively poor record for ischaemic heart disease, and it has been suggested that people move to a less active job as they become less fit. However Morris has followed his work with a study of civil servants whose occupational energy expenditure is low (presumably involving the shifting of small piles of paper from one pending tray to another). Those who in a selected and previously unannounced period of 48 hours took part in a leisure activity requiring quite a severe degree of exertion were subsequently found to have a reduced incidence of

coronary events compared with their colleagues who in replying to the questionnaire on how they had spent this 48 hours had not exerted themselves strenuously. This questionnaire was thought to be the best unbiased way of trying to separate people who customarily exert themselves from the 'loafers'. Subsequent study of matched groups of these civil servants has shown a lower number of abnormal ECGs in the 'non loafers', other risk factors being evenly distributed between the two groups.

This study like the famous Framingham epidemiological study appears to suggest that exercise acts independently of other risk factors.

If we therefore accept that it is established that those who habitually take exercise have a reduced incidence of coronary artery disease the mechanism poses several questions.

1. Does exercise act through the established risk factors? (This seems unlikely from the two studies mentioned above).
2. Does exercise act quite independently of the other risk factors? If so it would be a panacea which if universally adopted would reduce the incidence of ischaemic heart disease.
3. Do those who take regular exercise spontaneously have a certain vitality or *joie de vivre* or some other attribute that makes ischaemic heart disease less likely? If so universal prescription of compulsory exercise to those lacking this other factor might have no effect on the incidence of heart disease.

These questions can only be answered by studies of primary prevention programmes.

Primary prevention programmes

Studies so far seem to indicate that at the levels of exercise that can be tolerated by most participants in these programmes there is only a slight effect on the other risk factors from three or four exercise sessions lasting 15 to 30 minutes per week. More prolonged and intense exercise can appreciably effect serum triglycerides at the same time as reducing body fat. Thus the average jogger may be influencing his blood pressure and cholesterol relatively little whilst participants in the Boston marathon (3000 this year) may well be benefiting their serum lipids considerably. Primary prevention trials are obviously limited by the trade off between the intensity of exercise and the drop out rate of participants. Many participants may have a relatively low motivation, as trying to prevent something that may never happen has less appeal than stopping a second heart attack. Lack of results from primary prevention programmes can always be blamed by the enthusiasts on too low a level or too short a period of exercise, but in most series there is a high drop out rate from musculo skeletal problems and also from the difficulties and sometimes expense of attending supervised exercise sessions.

Secondary prevention

Secondary prevention studies have usually involved intense investigation and conditioning of dwindling numbers of post infarction patients. There is little doubt of the rehabilitative effects of these programmes in promoting confidence and a sense of well being in these patients, as well as improving the patients' ability to exercise by making their cardiovascular response to exercise more efficient.

The life saving or preventative effects of exercise programmes are still controversial, largely because many of the studies have been designed by enthusiasts who have incorporated inadequate controls in their trial design and have in some cases only managed to prove that the patients with the least myocardial damage take most readily to exercise, are less likely to drop out and have the best prognosis.

An ideal trial would have matched post infarction groups, with an even distribution of coronary artery lesions (found at angiography) as well as risk factors.

Many studies have shown that regular exercise appears to protect the post coronary patient from sudden death, but no study has shown any effect of the exercise on the diseased coronary arteries. Whilst the coronary artery disease itself exists it may well be that exercise delays rather than prevents further coronary events. This is suggested by a well designed study from Gothenburg (Sanne *et al.*) where the early lower mortality in the exercising group was cancelled out by a higher late mortality, which has been subsequently reported.

This rather depressing evidence must be seen against the real psychological benefits that patients get from feeling fitter and also feeling that they are contributing to their own health actively rather than passively swallowing tablets. On the other side, secondary prevention exercise programmes need very careful supervision, with individual testing and exercise prescription for each patient. Unfortunately there is evidence from Seattle that the risk of a potentially fatal dysrhythmia developing on exercise in post infarction patients does not diminish with time in the exercise programme. If substantiated there would continue to be risk in allowing any post infarction patient to exercise intensively on his own. However many do and some have become veteran marathon runners.

In conclusion there are many theoretical reasons to believe that exercise should prevent or lessen the impact of coronary artery disease (see Table I in Carruthers *et*

al. this journal and also Tunstall Pedoe and Thomason). However the evidence that exercise alone would make a significant impact on the terrifying incidence of ischaemic heart disease in this country is still lacking. Exercise should therefore be encouraged as one of several anti-coronary measures, including diet and the denigration of smoking. To be acceptable exercise should become a continuing habit from schooldays rather than a form of health insurance grabbed at in middle age.

Despite the lack of objective evidence I personally believe that relatively intense aerobic exercise must have some protective effect and have been running regularly (not only for this reason but because I also enjoy it) for more than 20 years. So as one of the self selected anecdotal pieces of evidence I am pleased to say that at 1800 kilopon metres on the bicycle ergometer my ECG shows no evidence of ischaemia.

REFERENCES (and further reading)

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