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Review Article

Prevention of Cardiovascular Disease by Psychological Methods

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It is generally recognised that much cardiovascular disease is the result of voluntary behaviour such as smoking cigarettes, and the pursuit of stress-prone lifestyles. Since these risks are primarily behavioural, it is appropriate to attempt to alter them, and hence reduce the risk of cardiovascular disease, using psychological methods, and such methods can reduce both biological and psychological stress-related factors. Studies of healthy populations, of those at increased risk, and of patients with clear cardiovascular disease have all shown that risk-related behaviour can be altered and, in some cases, the incidence of cardiovascular disease reduced. Future research will have to extend these findings, which were often on atypical populations, and confirm reduction of cardiovascular disease.

Epidemiological evidence shows that myocardial infarction and cerebrovascular stroke are more likely in people who smoke, who are overweight, who have high blood pressure or high serum cholesterol (e.g. Pooling Project, 1978), who do not exercise (Paffenbarger *et al*, 1983), who drink too much alcohol (Marmot, 1984), or who are aggressive and over-busy or depressed (Booth-Kewley & Friedman, 1987) or phobic (Haines *et al*, 1987). Cardiovascular disease, like other diseases, may also be more likely in those with poor social support (Berkman & Syme, 1979; Seeman & Syme, 1987). These risks can be powerful in themselves and, since they are believed to operate multiplicatively rather than additively (Truett *et al*, 1967), become increasingly so the more that are present. Furthermore, it is becoming clear that, at least in animal models, stressful environments interact with various predisposing factors, such as the animal's genetic make-up or a diet rich in saturated fats or salt, to increase markedly the degree of risk, or even the development of coronary atherosclerosis (see Manuck *et al*, 1986). Many of these factors are either directly or indirectly linked to modifiable behaviour, and expert bodies now frequently recommend that changing such behaviour should be the first step in reducing the risk of cardiovascular disease (Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure, 1986; Shepherd *et al*, 1987).

Since much of the risk relates to behaviour, no special justification is needed for maintaining that the proper approach to risk reduction is by changing that behaviour. Nevertheless, it may be helpful to comment on the pharmacological approach to the prevention of cardiovascular disease. There is no doubt that medication which, for example, is designed to reduce blood pressure or serum cholesterol, is highly effective in achieving the desired risk factor reduction and can in some instances lower the incidence of cardiovascular disease (Veterans Administration study, 1967; Lipid Research Clinics, 1984; Frick *et al*, 1987). Such findings are obviously important clinically. They also test the fundamental assumption underlying cardiovascular disease prevention, that altering a risk factor actually reduces the risk. At least under some circumstances this is clearly true.

However, medication cannot be the complete answer. The reasons are fourfold: medication is rarely appropriate in the primary prevention of disease (see below); even in those at heightened risk, the numbers of people involved make medication financially and practically unattractive; medication has been ineffective in the reduction of the effects of some of the commonest risks; and medication may produce an unacceptable level of side-effects. The operation of these factors can be seen, for example, in the pharmacological reduction of raised blood pressure. It would clearly be inappropriate to attempt to prevent high blood pressure by giving medication to a healthy normotensive population. It is doubtful if medication is appropriate for the large numbers of adults (perhaps 20% of the population) with mildly raised pressures. Treatment of this group by medication has proved disappointing and the reduction in risk has not matched the drop in pressure. For example, the recent exemplary MRC trial of the treatment of mild hypertension (Medical Research Council, 1985) failed to show reliable effects of adequate pharmacological treatment of mild hypertension on the rate of coronary heart disease in a controlled study of approximately 18 000

middle-aged hypertensives; there was a reduction in strokes, although they are comparatively infrequent in this age group. Over 15% of patients reported side-effects that required a change or withdrawal of medication (Medical Research Council, 1981). Many of these symptoms were apparently minor inconveniences such as a dry mouth or runny nose, but 20% of men, for example, reported impotence, which was presumably at least temporarily disturbing. There was also evidence of an increase in total mortality in women in the treated group. There is therefore a definite and well recognised need for effective methods of reducing those types of behaviour which increase the likelihood of cardiovascular disease, and increasing those that diminish it.

In considering the literature on altering the behavioural aspects of the risk for cardiovascular disease, I shall adopt a tripartite categorisation of preventative programmes: primary, secondary and tertiary. These terms are used in various ways in the literature. By primary I mean the altering of risk in those not known to be at increased risk, secondary refers to lowering the risk of those at raised risk and tertiary doing the same with those with clear evidence of cardiovascular disease. While it can be argued that one or other form of prevention is most important or likely to be most effective, it is probably most appropriate to regard them as complementary.

Primary prevention

Primary prevention is appealing since, as Rose (1981) and others (e.g. Lewis *et al*, 1986) have cogently argued, even a tiny shift in a risk factor in the community can substantially reduce the total number of cases of a disease, since it will affect the large number of people at slight risk who make the greatest contribution to the total number of cases. Rose has calculated, for example, that people with cholesterol levels not usually considered of clinical relevance in fact, because they are so numerous, account for three times as many cholesterol-related deaths as those with markedly elevated serum cholesterol. The disadvantage of primary prevention is that many at little or no actual risk are required to change their behaviour to save the unidentifiable few. However, it can be argued that only by altering community attitudes and behaviour can one realistically alter risky behaviour, since so many of these types of behaviour are maintained by the physical and social environment. If this is unaltered then interventions, even if directed at those at high risk, will founder under all the other pressures that lead to the high-risk behaviour.

Although primary prevention programmes are concerned almost exclusively with modifying behaviour, the formal impact of psychological theory and practice has, in some instances, been slight. For example, the approach to behaviour change adopted in the well known WHO-sponsored campaign (World Health Organization, 1986) to reduce coronary heart disease risk in over 60 000 factory workers in four countries was very unsophisticated in its use of adequate behavioural methods. It is, then, unsurprising that the results showed disappointing changes in risk factors and consequently unimpressive reductions in coronary heart disease.

The psychological input is much more explicit in the various community intervention projects, the earliest and best known example of which is the North Karelia Project (Puska *et al*, 1985). Almost two decades ago North Karelia had the highest rate of cardiovascular disease in Finland, and public and political pressure led to a substantial research project to reduce the classical risk factors. This very influential study showed that information from local medical practitioners and others, combined with limited media campaigns, could produce changes in smoking, diet (and hence serum cholesterol) and blood pressure. It has also been argued that there was a larger reduction in coronary heart disease in North Karelia between 1969 and 1982 than could be expected by chance (Tuomilehto *et al*, 1986). However, the initially high level of coronary heart disease in North Karelia and the steady decline in coronary heart disease in most of Finland in the same period make these results hard to interpret. The benefits may have been due to more widespread changes in behaviour or to the specific North Karelia programme.

The more recent American community-based interventions probably indicate the future direction of such programmes more accurately than the North Karelia study. These sophisticated projects attempt to change behaviour in many ways and at many levels, and it may be helpful to consider their procedures and results in terms of a simple model of risk factor reduction. Most of these programmes are primarily based on the provision of information, and the process through which this leads to a reduction in disease may be seen as consisting of the following sequence of changes:

(a) increased knowledge
(b) attitude change
(c) behaviour change
(d) risk factor reduction
(e) reduction in cardiovascular disease.

This is obviously an oversimplification, as attitude and behaviour change can occur without an increase in knowledge (e.g. when something becomes fashionable), but it does indicate that the fundamental premise of such interventions is that knowledge about risk and its modification is needed before people can change their attitudes to the risky behaviour and decide to change it. If they succeed this may lead to a reduction in the risk factor, which may in turn lead to a reduction in disease. It can be seen that the chain is long and may fail at any stage, so that only very modest reductions in disease might occur even if each stage was even 50% effective. These calculations may be a little pessimistic, since most programmes are directed at several risks, and, if the effects multiply as hoped, the whole may be more than the sum of the parts. Perhaps even more importantly, the numbers of people involved mean that even modest reductions in risk could greatly reduce the number of people killed or disabled by cardiovascular disease.

This approach can be seen in an elegantly designed field demonstration in three small California communities near Stanford (Farquhar *et al*, 1977). One town received no intervention, while in the other two, media campaigns were directed at smoking cessation, exercise, dietary change and blood pressure measurement and control. In addition, in one town the media interventions were supplemented with face-to-face interviews with participants at high risk. The media campaigns increased the population's knowledge about the risk factors for coronary heart disease, and led to changes in diet and reductions in plasma cholesterol and in overall cardiovascular disease risk as assessed by an epidemiologically-derived risk equation. Reducing smoking, however, appeared to require additional personal contact and behavioural interventions. The greatest increase in knowledge on some risk factors was in the community that had the face-to-face intervention with high-risk participants, but low-risk participants who were not seen face-to-face also showed increased knowledge. This suggests that, as one might suspect, the effects of interventions disseminate between members of the community, blurring the distinction between primary and other forms of intervention. This study also emphasised the limitations of media campaigns alone, a finding that has greatly influenced studies currently under way in Stanford and elsewhere.

In terms of the model of the link between information and coronary heart disease endpoints, this study documents most links in the chain up to, but not including, the final and vital one of a demonstrable reduction in morbidity and mortality. A very much larger study which seeks to do this is now nearing completion, the Five Cities Project (Farquhar *et al*, 1984). This study is broadly similar to the earlier one but provides more direct face-to-face interventions through the use of community resources as well as media campaigns. At least two other major studies are also running, in Pawtucket (Lasater *et al*, 1984) and Minnesota (Blackburn *et al*, 1984), which share many of the features of the Stanford studies but differ in their emphasis on such factors as the use of specially trained lay volunteers, the active co-operation of local supermarkets and restaurants, and programmes directed at schoolchildren. When these report it may be possible to gauge whether the alteration of these risk factors does indeed confer the expected advantages in health. The techniques used in these studies, in which there may be no contact between the change agents and the recipients, is very different from the usual clinical situation. However, if it is an effective way of producing change in large numbers of people, these studies may have a much wider relevance than simply the reduction of cardiovascular disease, important though that is. With advances in epidemiology we are increasingly aware of high-risk behaviour and at-risk groups. The various community projects provide a powerful model for altering such behaviour.

Secondary prevention

Psychological approaches to secondary prevention rely mainly on face-to-face contact between health professionals and the individual at risk. The risks that have been the subject of most psychological interventions are primary hypertension, Type A behaviour, various diet-related risks and smoking.

Primary hypertension: stress management

The psychological approaches to hypertension have taken two forms: the altering of stress-related processes and the reduction of the known risk factors for hypertension – obesity and the excessive consumption of alcohol and salt. Most psychological effort has been directed at stress-related processes. The proposition that stress has a causal role in the production or maintenance of primary hypertension draws support from two lines of research. Firstly, there is a developing body of evidence suggesting that lower animals when placed in stressful situations show elevations of pressure that eventually persist even after the animal is returned to the original benign environment. For example, mice reared in isolation show elevations in pressure when forced to interact with other mice, and with sufficient exposure

sustained high blood pressure and cardiac damage are seen (Henry *et al*, 1975). Similar studies also show that stress combines with other physical risk factors such as a genetic predisposition to hypertension or increased salt in the diet to produce high blood pressure (Anderson *et al*, 1983). With careful selection of the parameters, it is possible to demonstrate that two factors, say heredity and a stressful environment, can be required to produce elevations in pressure (Lawler *et al*, 1981). Secondly, studies with humans show that challenging cognitive tasks produce marked blood pressure rises in many normotensive subjects, and even larger reactions are seen in hypertensive patients. The offspring of hypertensives also show unusually large responses, even if their blood pressure at rest is apparently normal (Falkner *et al*, 1979). These findings point to the critical role of the interaction of psychological and biological risk factors.

Biofeedback was once used quite extensively as a stress reduction method but this, in part, reflected a confusion about the process involved in learning to control a visceral response. The most popular model of biofeedback was the motor skills model, which, in essence, asserted that using feedback to control a visceral response was very similar to using naturally occurring feedback to control a motor skill, such as playing darts. From this perspective, biofeedback does not enable one to control stress and hence lower pressure but to control pressure directly and bypass the factors that are causing the problem. This technological approach to a complex problem is attractive, perhaps seductively so, but almost two decades of research have undermined it more or less completely. The motor skills model failed even the most elementary tests, since biofeedback was not needed to produce most of the autonomic changes observed when subjects voluntarily controlled functions such as heart rate (Johnston, 1976) or blood pressure (Steptoe, 1976). Subjects can achieve similar control with simple instructions. Furthermore, changes in automatically innervated functions were not directly mediated but the result of changes in muscle or respiratory activity.

As well as failing at the theoretical level, cardiovascular biofeedback has proved entirely unnecessary clinically. There is no convincing evidence that biofeedback of blood pressure leads to a greater reduction in pressure than much simpler procedures that do not involve feedback (e.g. Blanchard *et al*, 1979; Frankel *et al*, 1978). As a result, the use of blood-pressure feedback has almost entirely ceased. There has been a recent interest in the use of finger-temperature feedback, presumably to reduce sympathetic arousal, in the treatment of hypertension (Blanchard *et al*, 1986). However, this has not been studied rigorously.

Stress management has withstood well over a decade of research, much better than biofeedback. In a recent review, I calculated that stress management was more effective than experimental control procedures in lowering pressure in mild primary hypertension in 50% of the 25 controlled studies then available (Johnston, 1987). Relaxation-based stress management was better than simple attendance at a clinic (Patel & North, 1975), non-directive psychotherapy (Taylor *et al*, 1977), elaborate physiological measurement (Bali, 1979), skin conductance biofeedback (Zurawski *et al*, 1987) and a carefully constructed placebo based on mild mobility exercises (Irvine *et al*, 1986). Not surprisingly, pharmacological treatments were slightly more effective than stress management (Goldstein *et al*, 1982; Jacob *et al*, 1986), but, as we have seen, their use in mild hypertension is under severe challenge. The effects of stress management have been shown to last for at least four years (Patel *et al*, 1985), although in some recent studies it appears that blood pressure also drops in control patients, given enough time (Agras *et al*, 1987; Chesney *et al*, 1987). A number of different procedures have been used in these various studies and there is no strong empirical basis for choosing any one over another. In general it appears that the best results have been obtained in studies in which the relaxation training is live rather than taped, the patients are encouraged to practise regularly at home, and there is an explicit effort to teach the patient to apply the techniques to manage stress in daily life. While it has not been studied in hypertension, relaxation training given by health professionals has been found to be superior to that provided by specially trained community volunteers in the treatment of the aversive effects of chemotherapy (Carey & Burish, 1987).

Much of the treatment of stress in hypertensives uses very much simpler procedures than those used in the current cognitive/behavioural treatments of psychological problems such as anxiety or depression. It is probably appropriate that a psychological therapy offered to a large group of people united by a physical and not a psychological problem should be simple and readily offered by many professions. Furthermore, it should not be assumed that simple treatments only have simple effects (nor indeed should the converse be assumed). I have recently argued that complicated changes in behaviour and thinking can follow even from apparently simple procedures such as relaxation training (Johnston, 1986). At the experimental level,

we have shown that relaxation training can alter cognitive processes so that subjects recall more positive than negative words in an incidental recall task after they have relaxed (Peveler & Johnston, 1986). Despite the apparent power of such simple procedures, it is likely that particular hypertensives with more complex problems will benefit from specific cognitive or behavioural interventions directed at these problems. While little evidence on this point is available, it is probable that interventions that reduce subjective arousal in hypertensives (whether it be anxiety, anger or some other emotion) will also reduce their blood pressure.

Blood pressure is highly variable and very responsive to environmental factors. It therefore cannot be assumed that the pressures obtained in the clinic are representative of pressures at other times and in other places. A minority of studies of stress management have included either home or ambulatory measurement, with mixed findings. Some have found that stress management lowers pressure in the home and workplace (Southam *et al*, 1982; Chesney *et al*, 1987), while others have failed to do so, even in studies in which pressure was reliably reduced in the clinic (Irvine *et al*, 1986). This may well be because blood pressure is often much lower at home, even without treatment. Nevertheless, this is a critical issue which must be resolved.

The other critical issue is, of course, the effect of stress management on health. Part of the impetus for using stress management is that the pharmacological solutions have not affected coronary heart disease convincingly, at least in the mildly hypertensive. The effects of stress management on mortality and morbidity has not been examined, and the scale of the enterprise is daunting, but Patel *et al* (1985) have shown encouraging, and statistically reliable, reductions in coronary heart disease in a study of only 200 men at increased risk of coronary heart disease. A similar trend was found in a very recent study (Patel & Marmot, 1988).

Primary hypertension: weight, salt and alcohol

An alternative behavioural route to the lowering of blood pressure is to alter the physical factors which contribute to raised pressure. It is generally held that there is incontrovertible evidence that *obesity* leads to heightened pressure in some people (Chiang *et al*, 1969), and that substantial weight reduction leads to profound drops in pressure in the grossly obese (Maxwell *et al*, 1984). More modest weight reductions allow mildly obese patients to cease medication for long periods (Langford *et al*, 1985), and can lower pressure as effectively as medication (MacMahon *et al*, 1985) and reduce left ventricular hypertrophy (MacMahon *et al*, 1986). Contrary results have been reported (Haynes *et al*, 1984), but the weight reductions obtained in these studies were slight and the hypertension borderline. Surprisingly, the psychological input in many of these studies is modest, most studies relying simply on dietary advice from physicians and dietitians, although the more recent studies appear to be using behavioural programmes (e.g. Langford *et al*, 1985). The evidence with obese normotensives suggests that such programmes will be more consistently effective than advice alone (Brownell & Wadden, 1986), thus increasing the potential for cardiovascular benefit.

The consumption of *salt* has long been seen by interested members of the general public as a major factor in hypertension. This message may have been oversold, since there is a continuing lively debate between hypertension specialists on the exact role of sodium and its importance in hypertension (McCarron, 1985; MacGregor, 1985). It is almost certainly true that the virtual elimination of salt from the diet will lower pressure, but few would find the resulting diet either palatable or practical. The effects of more manageable levels of salt restriction are contentious and at best appear to be slight, even when salt is reduced to the desired level (Grobbee & Hofman, 1986). It has been suggested that there is a subclass of 'salt-sensitive' hypertensives, but they have proved difficult to identify in advance of salt restriction.

The evidence linking *alcohol* and blood pressure is steadily growing (MacMahon, 1987), and it is now clear that high levels of alcohol consumption are associated with heightened pressure (Shaper *et al*, 1981). Alcoholics show a reduction in pressure when they cease drinking, which reverses on relapse (Saunders *et al*, 1981), and blood pressure drops with the cessation of alcohol consumption in both hypertensives (Potter & Beevers, 1984) and normotensives (Puddey *et al*, 1985). There does not as yet appear to be a comprehensive controlled trial of the effects of an alcohol restriction programme in hypertensives.

A recent major study attempted to reduce weight, salt consumption and alcohol consumption in hypertensives taken off medication (Stamler *et al*, 1987). The major outcome criterion was return to medication in the subsequent four years. The programme was generally successful, since 39% of the treated group remained off medication as compared with 5% of the untreated. Weight was reduced, as was the consumption of very large amounts of salt, but there was little effect on alcohol consumption. The authors do not appear to have

offered a specific behavioural programme. This is an important line of enquiry and there is an obvious need for studies of the effects of systematic behavioural programmes to reduce alcohol consumption in hypertensives.

Type A behaviour

The most researched, discussed and, on occasions, attacked behavioural risk factor is Type A, or coronary-prone, behaviour. This complex behaviour pattern, comprising excessive competitiveness, sense of being under time pressure, and easily provoked hostility, was first shown to be an independent risk factor for coronary heart disease in 1975 (Rosenman *et al*, 1975) and achieved widespread acceptance, in North America at least, over the next few years. Since then the picture has grown increasingly confused, with technically satisfactory studies both confirming (Haynes *et al*, 1980), and disconfirming the original findings (Johnston *et al*, 1987). Similarly, studies relating Type A to the extent of occlusion of the coronary arteries of patients with suspected coronary artery disease have had very mixed results (Dimsdale *et al*, 1979; Williams *et al*, 1980). A recent meta-analysis suggests that Type A does relate, albeit rather weakly, to coronary heart disease (Booth-Kewley & Friedman, 1987), particularly if the Structured Interview method of determining Type A is used. More detailed studies of the components of Type A suggest that the hostility component is particularly powerful in predicting coronary heart disease (Matthews *et al*, 1977; MacDougall *et al*, 1985).

There is no consensus on how Type A causes coronary heart disease. Many investigators operate on the working hypothesis that it leads to harmful increases in sympathetic arousal and cardiovascular reactivity. It is quite easy to find contrary evidence to this position (Myrtek & Greenlee, 1984), but it prevails, primarily because of the absence of a plausible alternative.

The current uncertainties about the mechanism of Type A risk obviously pose problems in designing interventions. Ideally, the intervention should concentrate on those components that carry the risk for coronary heart disease. Present evidence suggests that overall ('Global') Type A and Hostility should be altered by any intervention thought likely to be of value in reducing coronary heart disease. Most of the efforts to alter Type A behaviour in the otherwise healthy have been little more than feasibility demonstrations, with the best work done in patients following a myocardial infarction. There are exceptions, however. Roskies and her colleagues have reported a careful study of Type A executives, and Gill and co-workers an intriguing study of senior army officers.

Roskies *et al* (1986) regard Type A behaviour as a form of excessive behavioural and physiological reactivity and have employed an elaborate form of stress management to reduce this behaviour. This procedure was more effective than either aerobic exercise or weight training in altering both Global Type A and Hostility, but all procedures were equally ineffective in altering the cardiovascular response to challenging laboratory tasks. This may not be serious, since, as we have seen, it is not clear that such responses relate to Type A behaviour in any important way. Gill *et al*'s (1985) study is an offshoot of a study of Type A change following myocardial infarction which will be discussed in the section on tertiary prevention. Briefly they were concerned to see if a programme that altered Type A in post-myocardial infarction patients also did so in healthy middle-aged Type A men. In addition, they sought to determine if Type A reduction had any deleterious side-effects since, informally at least, some have maintained that Type A behaviour is necessary for effective survival in modern society. Both Global Type A and Hostility were altered in healthy middle-aged officers of the rank of lieutenant colonel and colonel, and there was no deterioration in their effectiveness, as determined by their brother officers.

The alteration of aspects of Type A is therefore possible. So far, Type A has been assessed using the Structured Interview, the first, and arguably best, measure of Type A, and also by various questionnaires filled in by the participant, or occasionally, an informant. Most of these measures might well be susceptible to falsification by participants who know that their behaviour is being measured, and future studies will have to establish that Type A behaviour changes reliably in a wide range of real life situations. There is at present no evidence that Type A change alters the risk of coronary heart disease in otherwise healthy Type A subjects, and such evidence will be difficult to acquire until we have a clearer understanding of the mechanisms through which Type A affects the heart, and of which components of the behaviour pattern predict coronary heart disease and under what circumstances.

Multiple risk factor modification

Smoking and cholesterol reduction have been studied most persuasively in the context of multi-factor risk reduction studies. A good example of such is the widely known and very impressive Oslo study of

reductions in serum cholesterol and smoking (Hjerman *et al*, 1981). In this study 600 men with raised serum cholesterol and elevated scores on a composite risk score, based on cholesterol, smoking (almost 80% smoked) and blood pressure, were given individual advice by a specialist team of physicians and dietitians. Compared with a randomised control sample, there was a 13% reduction in serum cholesterol, plus other beneficial changes in blood lipids, and a 45% reduction in smoking. The incidence of fatal and non-fatal myocardial infarction and sudden death in the subsequent five years was reduced by almost 50%. Detailed analyses suggested that much of this reduction was due to the effects of the change in serum cholesterol, smoking having rather less effect than predicted. The effectiveness of this study may well illustrate the importance of using experienced specialist staff with a commitment to changing risk factors. The Oslo group appears to have used many of the procedures which are also used in the more obviously psychologically-based studies, such as direct specific advice given more than once, individual dietary plans, involvement of spouses, and feedback of serum cholesterol levels. It may be significant that the best results in the otherwise disappointing WHO study, mentioned previously, came from the Belgian centres, who also used specialist teams to reduce the risk factors (Kornitzer *et al*, 1983).

The positive results of the Oslo study contrast with the poor effects of the MRFIT project on reducing coronary heart disease (Multiple Risk Factor Intervention Trial, 1982). In this study over 12 000 men at increased risk of coronary heart disease were randomly allocated either to normal care or to programmes directed at reduction in smoking, dietary change to lower serum cholesterol, and medication to control high blood pressure. Complex behavioural programmes were used to alter smoking and diet. Despite the finding that the risk factors were successfully reduced, there was not a reliable reduction in mortality from coronary heart disease over the seven years of the study. On the face of it this might seem to suggest that behavioural methods of risk reduction, even when appropriately and apparently successfully applied, may not in fact reduce risk, a finding that casts a doubt over the whole endeavour. The authors suggest that the difficulty may, in part, be due to changes in the risk factors in the control group, which lead to a much lower than predicted mortality. The drop in risk may reflect changes in medical practice and public attitudes during the study. They also present evidence suggesting that the anti-hypertensive medication may have actually increased the risk of cardiac-related deaths in some patients and offset the beneficial effects of the substantial reductions in smoking and serum cholesterol.

The value of a systematic psychological input was very clear in a recent (Lovibond *et al*, 1986) study of men at high risk for coronary heart disease as assessed by a composite function based on weight, blood pressure, physical fitness and cigarette smoking. The 25% at highest risk of over 300 men received behavioural programmes of three levels of intensity. In the most intense programme the participants received regular feedback on their coronary risk status, detailed health educational material, and a detailed personalised behavioural programme for altering the risk factors, which was implemented under close supervision. The less intensive programme was similar but the implementation of the programme was not so closely directed. In the least intensive programme subjects received only a general description of the behavioural programme and did not keep detailed records of their own efforts nor were they given detailed feedback on their risk factors. Participants in all groups met frequently over a six-month period. In general, while all three interventions were effective, the most intensive produced the greatest change. The size of changes was impressive: overweight participants lost over 10 kg, those with high blood pressure decreased SBP by over 10 mm Hg, and there were marked increases in physical fitness. With respect to the function used to assess risk at the end of the intervention, the most intensive group had decreased their risk by 44% and the least by 23%. As the MRFIT study indicates, it is not prudent to assume that such reductions will actually turn into lives saved and disease avoided; nevertheless, this is an impressive demonstration of the power of a very vigorous and systematic behavioural programme in reducing the risk factors for coronary heart disease.

Tertiary prevention

From a psychological perspective the role of prevention after there is definite evidence of major coronary artery disease, such as following a myocardial infarction, presents an interesting and complex challenge. On the one hand after an infarction the major risk factors (except cigarette smoking) may be of much less importance in predicting recurrence than is the state of the myocardium, while on the other the patients have had a very dramatic experience that must be a highly motivating warning of the urgent need to change their behaviour. Many of the psychological interventions after myocardial infarction are concerned

with reducing the psychological sequelae of the infarction, such as anxiety, depression or undue restriction in activities. Such interventions (reviewed by Johnston, 1985) can be important in patients with demonstrable behavioural problems, but have, at least explicitly, little to do with prevention of a further myocardial infarction. However, stress, by activating sympathetic mechanisms, may increase the likelihood of myocardial infarction or sudden cardiac death in the diseased heart.

Behavioural efforts have been directed primarily at Type A change, stress reduction and increasing physical fitness. Rather surprisingly, despite the importance of cigarette smoking in patients who have experienced a myocardial infarction, there does not appear to have been a randomised trial of cessation methods after an infarction. Burling *et al* (1984) suggest that up to 50% of smokers cease or reduce smoking after a myocardial infarction, and this is associated with a clear reduction in mortality and morbidity. Patients are more likely to stop with vigorous advice given more than once (Burt *et al*, 1974). There is evidence that patients with definite cardiovascular disease are more likely to give up smoking than people with other smoking-related conditions (British Thoracic Society, 1984). Adding a booklet or nicotine chewing gum to advice did not improve cessation in 1550 patients attending clinics for smoking-related illnesses, including cardiovascular diseases (British Thoracic Society, 1983).

Before considering the long-term effects of interventions after myocardial infarction, it is worth commenting on the psychological factors operating immediately after the infarction when the patient is still under intensive care. Lown and his colleagues have demonstrated very elegantly that stress can lower the threshold for fibrillation and induce potentially fatal arrhythmias in the ischaemic myocardium of the dog (Verrier & Lown, 1984). Such effects could occur at any time in the diseased heart but might be most likely in the period shortly after a myocardial infarction. There have been very few interventions during this period but Gruen (1975) reports that an eclectic form of psychotherapy led to patients experiencing less potentially dangerous arrhythmias during their time in coronary care than did standard hospital care. They also spent less time in hospital. This suggests that the use of simple stress management at this time is worth exploration.

Physical fitness programmes are a major part of most myocardial infarction rehabilitation schemes. This is based primarily on the relationship between physical fitness and coronary heart disease and the haemodynamic benefits of the physical training. Unfortunately, it has proved very difficult to show that such programmes actually reduce the risk of future myocardial infarction. Substantial studies, such as the National Exercise and Heart Disease Project, have failed to show any convincing reduction in myocardial infarction (Shaw, 1981). In part the problem may relate to the difficulty in maintaining physical fitness over a prolonged period of time.

Type A behaviour and stress reduction

We have already seen that much controversy surrounds the claim that Type A predicts the incidence of coronary heart disease in the apparently healthy. The evidence on Type A as a risk factor after a myocardial infarction is also contentious. Among recent studies, Case *et al* (1985) failed to find any evidence that Type A predicted the recurrence of a myocardial infarction, Powell & Thoresen (1985) found that some aspects of Type A did predict recurrence, and Ragland & Brand (1988) found that Type B individuals were more, rather than less, likely to die of a subsequent infarct. Nevertheless, a major study suggests strongly that an intervention designed to alter Type A behaviour reduces the recurrence of myocardial infarctions. Friedman, Thoresen and colleagues (Friedman *et al*, 1984, 1986) have developed a very complex package for altering all aspects of Type A behaviour. This package attempts, using a wide range of cognitive/behavioural techniques, to alter the patient's thinking, behaviour, environment and physiological responses. In a manner that will be familiar to those who use cognitive methods to treat depression and anxiety, the patient's underlying assumptions about personal worth and professional goals are examined, specific types of behaviour incompatible with Type A behaviour are encouraged, and role-playing, modelling etc. are used to modify competitive or aggressive behaviour.

In a study lasting over four years, this package was used with groups of up to ten patients who had experienced at least one myocardial infarction. The groups initially met weekly and then less frequently, but patients remained in therapy for the complete period of the study. Approximately 600 patients received this intervention, while a randomly selected group of approximately 300 met regularly to receive high-quality cardiovascular care and assistance with the psychological effects of the myocardial infarction, but not any systematic attempt to alter Type A behaviour. The intervention group met on average 38 times and the controls 33. The results were dramatic. Type A behaviour was assessed by a modified version of the Structured Interview, patient

self-report, and reports from the patient's spouse and an informant at work. Both Global Type A and Hostility decreased more in the intervention than the control group. When the recurrence rate in all patients who entered the study was examined, the annual percentage of cardiac events was 4.97% in the control group and 2.96% in the intervention group, nearly a 50% reduction. In addition, participants who showed substantial change in Type A by the end of one year were four times less likely to have a myocardial infarction in the subsequent two years. The primary effect was on non-fatal myocardial infarction, although in a subsidiary analysis Friedman *et al* (1986) argue that mortality was also reduced after the patients had been in the programme for some time. The results were not due to differences between the groups in other risk factors or medication. When subjects were stratified on the severity of their myocardial infarction, it was clear that the main effect of treatment was on those with less severe myocardial damage.

This study clearly shows the benefits that may accrue from treatment that is directed at altering the Type A behaviour and thinking of patients at high risk for myocardial infarction. Obviously, very many issues remain unresolved by this one study. It is likely that a complex treatment lasting many hours over four years contains irrelevant aspects which could be pruned, perhaps with significant savings in time or with the substitution of more powerful procedures. The control group probably provided an adequate control for general attention, but is unlikely to have controlled for the many complex effects of the cognitive/behavioural package. It would, therefore, be premature to conclude that it was the changes in Type A that mediated the cardiovascular disease outcome. The data are supportive, but Type A was the main behaviour measured and so only Type A could be seen to change. It is at least plausible that the cognitive/behavioural intervention altered other factors such as mood, level of stress and perhaps even social support.

The likelihood that simpler, quicker and cheaper intervention can reduce the risk of recurrence of myocardial infarctions is supported by an interesting attempt to deal with stress in the year following an infarction (Frasure-Smith & Prince, 1985, 1987). In this study slightly over 200 patients in an intervention group were regularly contacted by phone, and a questionnaire about general health was administered. If symptoms of stress were detected the patients were visited by a nurse counsellor who took whatever action seemed appropriate, which could involve referral to a specialist agency. A control sample received normal care. In the following year the groups differed slightly in their stress symptoms, and the intervention group had a 50% reduction in mortality. It is not entirely clear that this was due to stress reduction. The randomisation procedure in this study was unsatisfactory and may have led to social class differences between the treatments, with unpredictable effects on outcome. In addition, the authors did not monitor medication use, which raises the suspicion that compliance with medication could have been altered by the intervention. However, one would not have expected what must surely have been a minor difference in medication to effect survival reliably in a small sample. These interpretative problems must be resolved in future studies, and detract from the value of this study, which does at the least draw attention to the advantages of directing interventions at those patients who are reporting problems, and then tailoring the intervention to that problem.

Comment

Before drawing conclusions from this overview of behavioural methods of reducing the risk of cardiovascular disease, it might be helpful to make some general comments.

There are three main problems with the behavioural approaches to altering high-risk behaviours. Firstly, many of the interventions appear costly and labour-intensive. While one might hope that future research will develop more efficient procedures, and ways of using less expensively trained personnel, nevertheless psychological interventions are likely to remain expensive if costed over the comparatively brief period during which they are delivered. However, it is more appropriate to cost them over the period during which they are effective. If the effects persist, as it appears they do, then the costs become trivial. Secondly, even the best cognitive/behavioural methods are far from perfect. For example, there are large numbers of people whom we are unsuccessful in training to manage their stress, alter their diet or cease smoking. Perhaps most critically we are not particularly good at motivating people appropriately who might initially be reluctant to change their behaviour, although there are procedures for increasing motivation for treatment (Miller, 1985). Unless the patient wishes to change, most behavioural programmes are ineffective. Obviously the community-based primary prevention programmes have a vital role to play in this respect. Finally, as with any programme of risk factor reduction, it cannot be assumed that even successful programmes of behavioural change will lead to actual reductions in cardiovascular disease. This has only

been demonstrated convincingly in a few rather special instances. It is unrealistic to hope that it will be possible to mount major trials of the effects of the modification of all the behavioural risk factors on cardiovascular disease. Nevertheless, more evidence of the real benefits of behavioural risk factor reduction carried out under realistic field conditions must be gathered. It would be unreasonable and ultimately counterproductive if it became accepted dogma, in the absence of empirical evidence, that reduction in the behavioural risk factors is beneficial. This is particularly important in primary prevention, where the aim is to change the behaviour of apparently healthy people at low risk of cardiovascular disease.

These possible restrictions in the potency or applicability of psychological interventions in this field are offset by powerful advantages. The most obvious advantage is the total inappropriateness of anything other than a psychological intervention to deal with some of the risk factors, such as smoking, Type A behaviour or poor social support. More subtly, it is a powerful advantage that the psychological interventions are usually much less specific than their pharmacological equivalents. Most weight reduction regimes, for example, will attempt to reduce the intake of fat, particularly animal fats, and increase fibre, and this can be expected to reduce the lipid-related risks. Even more importantly, stress-related treatments are not finely targeted with respect to physical endpoints or biological risk factors, and it is reasonable to assume that interventions that reduce stress in an individual or community will affect many stress-related risk factors and reduce the effect of the more biological risk factors, such as elevated serum cholesterol. In this case stress management may have more effect on coronary heart disease than would be predicted from the change in the target risk factor, as appears to have happened in the studies of blood pressure reduction by Patel *et al* (1985) and Patel & Marmot (1988). It may also affect other stress-related illnesses. Finally, behavioural interventions such as stress management are believed to have few negative side-effects and are therefore acceptable to patients. They are unlikely to increase one risk factor while diminishing others, as may happen with medication.

Conclusion

Much cardiovascular disease is associated with voluntary behaviour and hence people should change or avoid such behaviour to reduce their risk. There is evidence that they can do so when healthy and at low risk, when at high risk, and after they have manifest cardiovascular disease. It appears that both the classic biological risk factors and more obviously psychological factors can be altered by procedures based on the principles and methods of psychology, and there is limited, but encouraging, evidence that such alterations reduce the likelihood of cardiovascular disease.

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