The current status of the coronary prone behaviour pattern

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Introduction

The belief that a pattern of aggressive or irascible behaviour is associated with coronary heart disease holds a peculiar and persistent fascination for both the lay public and physicians and psychologists. While the history of the idea can be traced back for at least several centuries, the scientific study of the possible behavioural basis of coronary heart disease (CHD) was laid by the pioneering work of Friedman and Rosenman who, over 30 years ago, described what they termed the Type A or coronary prone behaviour pattern[1,2]. This pattern was characterized by competitiveness, time urgency and aggression, and was measured using a mildly challenging structured interview. Type A behaviour was determined both from the content of the subject's answers to the interview questions and also from the style in which they responded. In the Western Collaborative Group Study, a prospective study of approximately 3000 healthy middle aged men, Rosenman and colleagues found that, over a 8.5 year period, Type A men were twice as likely to experience a myocardial infarction as Type B (the opposite of Type As) even if allowance was made for other risk factors, such as blood pressure and serum cholesterol[3]. They suggested that Type A behaviour was an independent risk factor of approximately the same predictive power as the classic risk factors for coronary heart disease of cigarette smoking, raised blood pressure, and raised serum cholesterol. If this is generally true then the understanding and modification of Type A behaviour is an important part of the understanding, prevention and treatment of CHD.

Since Friedman and Rosenman's initial studies research on coronary prone behaviour has taken five main forms: (1) prospective studies of the Type A behaviour pattern; (2) studies of Type A behaviour and coronary artery disease; (3) attempts to isolate the important components of Type A behaviour; (4) studies of the modification of Type A behaviour; and (5) examination of the possible physiological mechanisms relating Type A behaviour to CHD.

Prospective studies of Type A behaviour and coronary heart disease

There are now over 20 prospective studies of Type A behaviour with very mixed results[4,5]. While some studies have replicated the initial findings from the Western Collaborative Group Study[6,7] many have not, including two substantial studies conducted in this country[8,9]. Some of the differences are, in all likelihood, due to inadequacies of measurement, such as using simple questionnaires[10] rather than the structured interview, which is generally considered the only adequate measure of Type A behaviour. However, this is not a convincing explanation of the overall pattern, nor does it explain a number of very influential studies that conspicuously failed to confirm the predicted relationship. Shekelle and others attempted to predict CHD using a very carefully standardized and monitored version of the structured interview in 3110 men at high risk for CHD admitted to the MRFIT randomized control trial of coronary risk reduction[11]. Over 7.1 years the percentage Type A men experiencing a coronary event was 4.06, and for Type Bs a virtually identical 4.40. Even more troubling is two long-term follow up studies of the original Western Collaborative Group Study sample. Ragland and Brand showed that over a 22-year-period mortality from coronary heart disease was not related to Type A status, although such deaths were predicted by the tradition risk factors[12]. In a separate study they also showed that coronary mortality in survivors of a first myocardial infarction was significantly *higher* in Type B individuals[13].

Post hoc explanations for all these discrepant findings are possible, and have been offered. It is possible that Type A behaviour is only predictive in a healthy low risk sample and not the high risk sample of, for example, the MRFIT study. It is also quite likely that Type A is not a stable characteristic and a classification carried out in middle age may not apply to the elderly. Equally it can plausibly be argued that Type Bs who experience a myocardial infarction must be at increased risk on some other risk factors (since they are clearly low on at least one) and therefore at different risk from Type A survivors. However plausible each individual explanation, serious doubts about the importance of Type A behaviour pattern must be raised by the need for so many unique, *post hoc* explanations of aberrant findings. The traditional risk factors of cigarette smoking, raised blood pressure, and raised serum cholesterol have been shown to be powerful and consistent predictors of CHD in numerous studies in many countries and with many different populations. Type A behaviour is clearly not a risk factor of the same power and generality.

Type A behaviour and coronary artery disease

The prospective follow up study of a substantial cohort of individuals on whom appropriate measures have been made is a powerful technique. It is also slow, costly, and critically dependent on the adequacy of the initial measurement. As we have seen many of the prospective studies of Type A behaviour relied on inadequate measures of behaviour. A more flexible interim strategy may be to relate behaviour to the concurrent state of the patients coronary arteries, as

revealed by coronary angiography. This approach does not have the power of the prospective study since: (1) cause and effect relationships are even harder to determine; (2) it only relates to one possible mechanism linking Type A and CHD; and (3) finally and most critically, there are substantial sampling biases in the sample of patients who are available for study, since patients without some indication of CHD rarely undergo angiography. Despite these defects the study of patients undergoing angiography has proved instructive.

Rather like the prospective studies, both positive[14] and negative[15,16] findings have been reported. The largest and most convincing study, of over 2000 patients undergoing coronary angiography[17], showed that Type A behaviour was associated with increased likelihood of coronary artery occlusion in younger patients but not in those in late middle age or above. The authors suggest this is due to the survival of those in whom Type A behaviour is less of a personal risk, since more of those susceptible to this risk factor will have experienced a myocardial infarction and died. However, the main importance of the studies of coronary artery disease (CAD) is the impetus they gave to the examination of the components of Type A behaviour.

The components of Type A behaviour

Type A behaviour, as originally described by Friedman and Rosenman, was a potentially complex mixture of various components including competitiveness, feeling under time pressure and aggression. Any, all, or a particular mixture of these components might best define coronary prone behaviour. In a series of very influential studies Dembroski and colleagues[18,19] developed a rating scheme to obtain component scores from tapes of the structured interview and applied these to existing data sets from studies of Type A behaviour and CAD. They proposed that coronary prone behaviour could be characterized in terms of speech style and various content and style measures the most important of which was what they termed 'potential for hostility', a readiness to get angry and assume the worst of people. Potential for hostility related to the extent of coronary occlusion, even in studies in which Type A behaviour did not, suggesting that it was the important component defining coronary prone behaviour.

In a particularly neat study Siegman and colleagues[20] showed that while expressive hostility (which is akin to a lay view of aggression and includes the expression of anger) related as expected to the extent of coronary artery occlusion, neurotic hostility (which reflects the experience of anger) related negatively, i.e., the more hostile the less coronary occlusion. This presumably reflects the tendency of neurotic patients to experience more pain of a non-organic origin and to be more likely to be referred for angiography in the absence of CAD.

Re-analyses of data from prospective studies showed that subjects high in potential for hostility were more likely to experience a subsequent myocardial infarction. This was true both in studies in which Type A behaviour also related to a myocardial infarction[21] and more importantly in studies, such as MRFIT in which Type A behaviour was not related to myocardial infarction[22].

In a related series of studies Williams and colleagues showed that hostility measured by the Cook-Medley questionnaire[23] related to the extent of coronary occlusion. In various opportunistic prospective studies using convenience samples, such as medical students who had completed the Cook-Medley on admission to medical school, Williams and others showed that hostility predicted subsequent myocardial infarction[24-26]. Negative prospective studies have also been reported[27,28].

The current consensus view is probably that some aspect of hostility relates to CAD[29]. However, it should be acknowledged that there are many inconsistencies in the available data and many of the positive findings come not from carefully set up studies designed to investigate the hypothesized link between hostility and heart disease but from opportunistic examinations of existing data sets or follow up studies of highly selected samples. These findings should be regarded as suggestive rather than conclusive and we await clear cut findings from studies specifically designed to investigate the hostility hypothesis.

The modification of coronary prone behaviour

If there is an identifiable pattern of coronary prone behaviour then the reduction of such behaviour becomes of obvious practical importance. Additionally, the effects of the modification of a risk factor on subsequent disease is an important step in establishing the causal role of the factor in the disease process. Rather surprisingly, given the complexities of the epidemiological evidence on coronary prone behaviour, attempts at modification of coronary prone behaviour have been spectacularly successful.

There is a consistent literature showing that the various components of Type A behaviour can be modified by fairly simple procedures. Among the most persuasive of such studies is a comparison of stress management and either aerobic or weight training in healthy Type A managers[30]. A package of stress management involving relaxation training and the identification of Type A behaviours and situations was much more effective in modifying both Type A behaviour and hostility that either of the exercise regimes, which were ineffective.

The most important question is whether such behaviour modification is accompanied by a reduction in CHD. Friedman, Thoresen and others examined this in a study of re-infarction in almost 900 patients who had experienced at least one myocardial infarction[31,32]. In a randomized trial two thirds of the patients were allocated to a group programme designed to reduce all aspects of coronary prone behaviour while the remainder met in groups led by cardiologists and received general counselling about recovery from an infarct plus standard cardiological care. The behaviour modification groups received a programme directed at altering the environmental, behavioural and cognitive processes thought to underlie coronary prone behaviour. The procedures used included relaxation training, role play of Type B behaviour and various forms of cognitive restructuring in which the patients identified and modified hostile cognitions and the setting of inappropriate, over ambitious or unrealistic personal goals.

Over a 4-year-period there was significantly greater reduction in both Type A behaviour and hostility in the behaviour modification programme and, critically, the re-infarction rate was reduced by almost 50% in

this condition compared to the control group. There was no detectable difference in medical or surgical treatment between the two conditions and there was no difference in the severity of the initial infarction, although the benefits of the behaviour change programme were largely confined to the patients with less severe infarctions. No harmful side effects of changing coronary prone behaviour were reported and in a subsequent study of senior army officers it was shown that Type A behaviour change was not associated with any reduction in the officers' military efficiency[33].

Therefore, it has been reasonably clearly demonstrated that many aspects of coronary prone behaviour can be changed and one substantial study has shown that such change has the predicted effect on CHD. However many questions remain unanswered, including the long-term maintenance of the behaviour change, the effective components of the group programme and, of course, the success of the programme in reducing CHD in other samples of patients.

Mechanisms linking coronary prone behaviour and CHD

Attempts to determine the physiological processes through which coronary prone behaviour might produce CHD have been rather limited and disappointing. In part this stems from the difficulty in determining what has to be explained, since the basic findings on Type A and related behaviours have been so contradictory. There are two fundamental questions to consider, the pathological process to be explained and the intervening physiological mechanism.

While it is seldom made explicit, most investigators appear to assume that coronary prone behaviour acts to increase the likelihood of the patient developing CAD through the development of atheroma. Coronary prone behaviour is therefore seen as operating over a long time to increase the patient's risk. Clearly the studies of coronary occlusion, in as far as they are positive, support this view but it should be recognized that coronary prone behaviour could equally well relate to the acute processes that lead to a myocardial infarction such as plaque rupture and thrombus formation or to the occurrence of fatal arrhythmias. There is little positive evidence on this but the sudden explosive nature of some of the components of Type A behaviour could plausibly relate to these acute processes where the more long-term chronic components, such as potential for hostility, may relate to the presumably slower processes that lead to the build up of atheroma.

Investigations of intervening mechanisms are largely confined to investigating whether Type A or coronary prone individuals show increased cardiovascular responsiveness to various forms of psychological challenge. This potential mechanism appears to have received much attention largely because cardiovascular reactivity was already under extensive scrutiny since some consider it a risk factor in its own right. It is compatible with coronary prone behaviour relating either to CAD or to the acute process preceding myocardial infarction or cardiac death. The research findings are complex and some reviewers have concluded that there is no reliable difference between Type A and Type B individuals in their response to a wide range of tasks[34] but a recent statistical analysis of over 70 published studies[35] suggests Type A men show greater increases in blood pressure and heart rate than Type B men during challenging tasks such as video games. The effect is not seen in women and is very dependent on the task used and the measure of Type A behaviour. This conclusion should be viewed with some caution since it is likely that many failures to find the predicted relationship fail to be published and hence do not enter literature based summary analyses.

Summary

Despite over 30 years of increasingly vigorous research it is still not possible to claim with acceptable certainty that there is an identifiable pattern of coronary prone behaviour nor to say with any confidence that the idea is misguided. The scientific process that leads from initial tentative findings through generation of a hypothesis, to rigorous and cumulative tests of that hypothesis has not happened. Instead there has been a rather erratic series of positive and negative studies, and the generation and modification of essentially rather similar hypotheses. As a result we are still in the position of claiming that there may be a pattern of behaviour that predicts CHD and that it is probable that hostility is involved. It is not clear why the idea is so persistent but it may well lie in the combination of a widely held lay belief that heart disease relates to stress and personality, with tantalizing positive findings occurring every few years. While there has been little increase in understanding of the role of behavioural factors in CHD as a result of this 30 years of endeavour there have been clinical benefits. It has clearly been shown that what are regarded as coronary prone behaviours can readily be modified and that their modification appears to confer some health benefits and no detectable health hazards.

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