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Stress and the heart

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ABSTRACT The role of stress in the genesis of coronary heart disease has been difficult to resolve because stress is a nebulous concept that is difficult to define and virtually impossible to measure. Although there are links between psycho-social and emotional stress and cardiovascular disease there is little prospect of reducing coronary heart disease mortality by reducing stress. We should instead concentrate our efforts on identifyting, preventing, and eliminating the factors that create the underlying atheromatous coronary plaques that make us susceptible to the effects of stress.

KEYWORDS Catecholamines, Coronary heart disease, myocardial infarction, plaque rupture, stress

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INTRODUCTION

The role of stress in the causation of coronary heart disease is one of the most controversial and hotly debated issues in cardiology. Opinion surveys have consistently shown that the general public believes stress is an important cause of heart disease. Stress is also widely held to be the major cause of heart attack by the survivors of myocardial infarction (heart attack) and their close relatives. The directors of Human Resource departments and senior managers are therefore often reluctant to allow coronary heart disease patients to return to work for fear of worsening their condition. In marked contrast to these views, cardiologists and cardiovascular epidemiologists continue to argue that smoking, hypertension, hyperlipidaemia, diabetes, lack of exercise and obesity are much more important risk factors for the development of coronary heart disease than emotional or psycho-social stress.

PROBLEMS OF STUDYING STRESS IN HEART DISEASE

This controversy has been difficult to resolve because stress is a nebulous concept that is difficult to define and virtually impossible to measure. Moreover, there are often potentially confounding links between psycho-social stress and conventional risk factors such as smoking and a sedentary lifestyle. In biological terms, stress is perhaps best considered as a source of excessive sympathetic nervous (adrenergic) stimulation, leading to the release of catecholamines into the blood, which may be acute or chronic. High catecholamine concentrations increase cardiac work by increasing heart rate, blood pressure and myocardial contractility; it is, therefore, easy to construct biologically plausible hypotheses linking stress to the pathogenesis of heart disease. This concept has been given further credibility by a recent paper describing significant but reversible left ventricular dysfunction induced by acute emotional stress.

DUAL NATURE OF CORONARY ARTERY DISEASE

Coronary heart disease is a complex condition that is characterised by a progressive inflammatory disorder of the coronary arteries. Focal lipid-rich deposits of atheroma (fatty streaks) often appear in early adult life, but clinical manifestations such as angina on effort (stable coronary disease) or an acute coronary event (myocardial infarction, unstable angina, sudden death and other arrhythmia) seldom present before middle life. There are essentially two components of the disease; atheroma and thrombosis. Atheroma is a slow indolent and to some extent predictable process that leads to progressive narrowing of the coronary arteries. Thrombosis (an occlusive blood clot) is a sudden and typically unpredictable event that is thought to be responsible for the vast majority of acute coronary syndromes and cardiac deaths.

EVIDENCE FOR STRESS IN HEART DISEASE

Although it is easy to argue that chronic stimulation of the central nervous system might promote atheroma by inducing low-grade hypertension and systemic inflammation there is very little, if any, evidence to support the view that psycho-social stress causes atheroma. Nevertheless, an intriguing study of coronary heart disease mortality among Whitehall civil servants in the UK has produced compelling evidence of a link between 'job-strain' and heart disease. In this study, occupations characterised by low control over one's life were independently associated with increased coronary heart

disease mortality (relative risk 1·5:1) but those occupations characterised by high demand were not. In other words working hard did not seem to cause heart disease but being unable to control one's pattern of work might have done. Low control is usually associated with low pay and these findings may go someway to explaining the well-publicised link between social deprivation and coronary heart disease. On the other hand, much of this difference could easily be explained by social differentials in smoking, plasma cholesterol, blood pressure and obesity).

MYOCARDIAL INFARCTION

Coronary thrombosis usually follows a breach in the integrity (ulceration or fissuring) of an existing atheromatous plaque. Such 'plaque events' may be triggered by a sudden increase in mechanical wall or sheer stress due to a surge in blood pressure. 'Vulnerable' or dangerous plaques are characterised by a lipid-rich core, a thin fibrous cellular cap, an increase in the inflammatory cells, and release of specific cytokines that degrade matrix proteins. In contrast, stable or safe plaques are typified by a small lipid pool, a thick fibrous cap and plentiful collagen cross struts.

Physical exertion, a form of biological stress, is a well-recognised trigger for acute myocardial infarction. There is also strong evidence to support the view that other forms of stress can trigger plaque events. For example, there is an increase in myocardial infarctions early in the morning when stress hormones (cortisone, adrenalin etc) and blood pressure reach their diurnal peak; interestingly, there is also an increase in myocardial infarctions on Monday mornings compared to other days of the week. In addition, case control studies have shown links between emotional outbursts of anger and the onset of myocardial infarction.

In the best-known study, patients with acute myocardial infarction were twice as likely to have experienced an outburst of anger in the two hours preceding the onset of their symptoms than case controls. Moreover, Israeli cardiologists observed a four to five-fold increase in the number of patients with proven myocardial infarction admitted to Tel Aviv's coronary care units on days that SCUD missile attacks were launched against Israel during the Gulf War.

Similar data have come from other communities exposed to widespread stress as a result of natural disasters such as earthquake. A statistically significant increase in the numbers of acute myocardial infarction was even observed in England's hospitals on the day that England lost to Argentina in a penalty shoot out at the World Cup (30 June 1998)!

These observations are intriguing and fit well with the nineteenth century romantic novelists' view of sudden cardiac death. Most heart attack victims would have no difficulty in identifying a stressful event in the days or hours preceding their heart attack. Indeed, some form of triggering event can be identified in at least 80% of cases.

However it is desperately important to appreciate that such triggers may only bring forward by a few hours what is an inevitable event. A broken shock absorber on an old car is a good analogy. The driver may claim that the shock absorber was broken by driving over a bump in the road with good reason. However, one cannot ignore the fact that the shock absorber was badly worn because the car was old, and perhaps poorly maintained, and had driven many miles before hitting the bump.

CONCLUSIONS

Unravelling the role of acute stress in promoting heart disease and triggering myocardial infarction has been a fascinating process and has undoubtedly enhanced our understanding of the pathogenesis of coronary disease. However the reader will appreciate that there is little prospect of reducing coronary heart disease mortality by reducing psycho-social or emotional stress. We should instead concentrate our efforts on identifying, preventing, and eliminating the factors that create the underlying atheromatous coronary plaques that make us susceptible to the effects of stress.

KEYPOINTS

- Most patients and their relatives identify stress as an important factor after myocardial infarction (heart attack).
- Stress is difficult to define and impossible to measure, but is associated with increased catecholamines in the blood which increase functional demand on the heart.
- Myocardial infarction is associated with episodes of physical and emotional stress, and with a lack of control over one's own life.
- While stress or other forms of triggering event can be identified in at least 80% of myocardial infarctions, such triggers may only bring forward by a few hours what is an inevitable event.
- Preventing coronary artery disease currently depends on promoting weight control and exercise, stopping smoking, and controlling high blood fat concentrations, hypertension and diabetes mellitus.

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