EDITORIAL

PSYCHOLOGICAL MANIFESTATIONS AND HEART DISEASE

The effect of psychosocial and behavioural factors on cardiovascular disease continue to attract a lot of interest and have been the subject of epidemiological and psychosomatic medicine research for the past 20 years. What is difficult to find are studies conducted among black populations linking psychiatric morbidity with heart disease. In this issue, Njenga et al(1) conclude that African scientists must continue to concentrate on the urgent medical priorities of today (AIDS, malaria, measles, etc) while taking cognisance of the other emerging epidemic.

That psychological factors are tied to the pathogenesis of heart diseases has been a closely studied subject and some of the postulated mechanisms link them with coronary heart disease and hypertension. These factors have been categorised as 'pressure reactivity' on the one hand and on the other as "personality/behavioural" factors. In many reviews that have examined physiological hyper reactivity to environmental stimuli, there is relatively strong evidence that there exist subsets of individuals who have greater blood pressure reactivity to a variety of stressors than do others, ranging from experimental stress induced in the laboratory to stressful social conditions such as racism. However, the evidence linking reactivity in normotensive individuals with the eventual development of hypertension is equivocal. Perhaps most importantly, pressure reactivity in patients who have already developed hypertension may exacerbate and even accelerate their disease process. Further, both inhibited anger expression and excessive anger expression, may be precursors to hypertension.

Stress as a behavioural risk factor in coronary artery disease has been shown to operate through causing a sympathetic-adrenomedullary alarm reaction characterised by excess catecholamine secretion. It is believed that catecholamine-mediated cardiac effects such as increased heart rate, contractility, and conduction velocity, as well as a shorter atrioventricular refractory period, may be pathogenically related to adverse cardiac events.

Studies examining the temporal relationship between sudden death from arrhythmias and stressful events suggest uncertainty and fear of loss of control leading to a 'giving up state', a state akin to depression leading to vasodepressor syncope, arrhythmias and sudden death in patients predisposed to myocardial disease(2). Myocardial perfusion abnormalities occur during mental stress such as mental arithmetic stressor in patients with typical angina and coronary heart disease(3,4). Coronary artery disease and type A behaviour pattern suggests morbidity related to cynicism, hostile affect, and aggressive responding(5). Type A behaviour patterns have been shown to display larger episodic increases

in blood pressure, heart rate and catecholamine levels when confronted by a stressful task. Evidence from primate studies have linked development of atherosclerosis to sympathetic nervous system activation. These findings suggest a link between psychological states, physiological reactivity and subsequent cardiovascular morbidity(6).

The role of mood states and cardiovascular morbidity and mortality has been studied. One study showed major depression to predict cardiac morbidity up to 12 months post-cardiac catheterisation(7). Sociological factors such as work overload and life stress in addition to lack of social support have been shown to enhance coronary risk. Non-married status, tack of social support and reduced socioeconomic resources are linked to increased mortality in coronary artery disease(8-11).

Risk factors for cardiovascular disease include family history, smoking, poor diet, high cholesterol, obesity, lack of exercise and stress levels. The 1997 National Survey of Mental Health and Wellbeing found that 43% of Western Australians with a diagnosable mental health condition were current smokers, compared with 24% among people without a mental disorder(12). The prevalence of smoking was even higher among patients with psychosis, at 66%. Cigarette smoking is a powerful independent contributor to the occurrence of myocardial infarction, sudden death, peripheral vascular disease and stroke. It has been shown to act synergistically with other traditional risk factors such as hypertension and high blood cholesterol to increase the risk of coronary artery disease. Fifteen years after smoking cessation, the risk factor for coronary heart disease approaches the risk of individuals who never smoked. Rosenberg et al(13) have reported that the risk of myocardial infarction for cigarette smokers decreases within two years of quitting to a level similar to that in men who have never stopped.

Obesity is known to be a problem among people with chronic mental illness and has been linked to overeating, under activity and ignorance of correct dietary principles. Weight gain is also a known side-effect of some antipsychotic medications. Lower levels of physical fitness have also been found in mental illness, linked to physical inactivity. There is a strong association between obesity and hypertension, hypercholesterolemia and diabetes mellitus as risk factors for cardiovascular disease.

The foregoing suggests that psychological interventions may be possible. Thus individuals with type A behaviour with coronary artery disease may benefit from interventions such as stress management, biofeedback and relaxation therapy. Whereas patients

with co-morbid depression post cardiac catheterisation and myocardial infarction require aggressive antidepressant treatment. Smoking cessation is a difficult intervention however nicotine dependence can now be treated effectively using behavioral and pharmacological approaches. Behavioural treatments for obesity involve strategies combining exercise, dietary restriction, longitudinal monitoring and professionally-led skills training have demonstrated efficacy, especially when they have focused on maintenance of behaviour change.

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