

keynote report

Emotions and the heart: psychological risk factors for cardiovascular disease

by Professor Derek W. Johnston

The past

The idea that emotions contribute to heart disease has a long history. In the 18th century John Hunter, the Scottish surgeon and anatomist, and a famously quick tempered man with angina, reportedly said “My life is in hands of any rascal who chooses to tease or annoy me”. He died of a heart attack after losing his temper at a committee meeting in his medical school. Over 100 years ago William Osler, a Canadian physician with a dominant position in Anglo Saxon medicine described the typical heart disease patient as “a keen and ambitious man, the indicator of whose engine is always at ‘full speed ahead’”, i.e., an early recognition of the Type A personality. Today if you ask survivors of myocardial infarction (MI) what they think caused their heart attack, 70% believe that stress is involved (Gudmundsdottir, Johnston, Johnston, & Foulkes, 2001). Despite this long history and the current lay acceptance of the link between emotion and heart disease, the scientific study of the association is comparatively recent. Only 30 years ago, Weiner’s (1977) massive text book on psychosomatics “Psychobiology and Disease” did not include a chapter on heart disease and myocardial infarction (MI) had only two entries in the index. Dorothy Levenson’s (1994) splendidly gossipy history of the American Psychosomatic Society hardly mentioned cardiovascular disorders (CVD) until Type A personality is first mentioned in the 1960s. Perhaps most surprisingly no paper on CVD was included in “Classics from Psychosomatic Medicine, 1959-1979”. There were exceptions to this apparent lack of interest in heart disease. Friedman and Rosenman started their highly influential work in the late 50’s that culminated in the report of the Western Collaborative Group study in 1975 and in 1977, Jim Henry summarised his extensive studies of the effects of stress on the cardiovascular systems of mice and placed it in a wider social and cultural context in his wonderful monograph “Stress, Health and the Social Environment” (Henry & Stevens, 1977). However it is broadly true that the scientific study of the role of emotion as a risk factor for cardiovascular disease is a product of the last forty years.

How far have we gone?

In attempting to establish the importance and possible causal significance of a risk factor epidemiologists still rely on Bradwell Hill’s famous eight criteria. These are strength of association,



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consistency, specificity, temporality, biological gradient, coherence (perhaps more usually seen as biological plausibility), experiment and the little considered category of analogy. When we apply these criteria to emotion as a risk factor in cardiovascular disease it can be argued that the greatest advances have been made on the psychobiological basis of cardiovascular disease (biological plausibility); prospective epidemiological studies of emotion and CVD (temporality) and the greatest disappointment has been in the comparative failure of interventions designed to alter emotion to reduce subsequent CVD (experiment).

Biological Plausibility

The dominant psychobiological model is some variant of the reactivity hypothesis first proposed by David Krantz and Steve Manuck in 1984. This deceptively simple model proposes that stress (and by implication negative emotions) lead to altered physiological response in some people and that these responses are harmful to the arteries. The model usually incorporates a diathesis (vulnerability) component and asserts that specific individuals are, because of inherited or environmental influences, susceptible to the effects of stress or emotion, and hence if exposed to stress will suffer health damaging consequences. The reactivity hypothesis is usually applied to the effects of chronic stress (or repeated acute stress) on arterial deterioration but it is now also appreciated that stress and emotion play an important role in the acute process that trigger acute coronary events, such as a myocardial infarction (Johnston, 2002). Understanding of the pathophysiology of arterial deterioration and acute coronary events has changed dramatically in the last

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decade and it is now appreciated that it is a very active, highly complex process in which inflammatory mechanisms are critically involved. The reactivity hypothesis was originally proposed in a period when CVD was seen as a rather simple passive process and there was a danger that emotion would be seen as less and less important as our physiological understanding increased. The opposite has in fact happened and the last few years have seen the emergence of several essentially similar well documented and plausible models of the role of stress, emotion and related processes in cardiovascular disease (Black & Garbutt, 2003; Kop, 1999; Steptoe & Brydon, 2005).

My colleagues and I have for many years been examining the relationship between cardiovascular reactivity to laboratory stressors and CV reactivity to stressors in real life. If the reactivity hypothesis is valid then those individuals who produce the largest CV response in the laboratory must react more frequently or more intensely to stressful events and emotions in everyday life. The picture that emerges from studies we have carried out over the last 15 years suggests that laboratory reactivity does indeed generalise to real life and that when individuals prone to CV hyper-reactivity encounter stressful events in real life they show increased CV reactions. In our most recent study (Johnston, Tuomisto & Patching, in press) participants who showed the largest increase in heart rate to a variety of laboratory stressors also showed much larger increase in heart rate when speaking in public and when they reported feeling tense and aroused during the day. See also Johnston (1992) and Jain, Schmidt, Johnston, Brabant, & von zur Muhlen (1998).

Prospective studies

Since the landmark Western Collaborative Group Study (Rosenman, Brand, Jenkins, Friedman, Strauss, & Wurm, 1975) there have been well over 100 substantial prospective studies of stress and emotion as possible risk factors for heart disease. Kuper, Marmot & Hemingway published an authoritative systematic review in 2002. In studies of initially healthy populations they find that depression was a reliable risk factor for heart disease in 15 out of 22 studies, as was stressful work characteristics in 10 out of 13 studies. Depression was also a reliable risk factor in populations with pre-existing cardiovascular disease (18 from 34 studies showed this). There were too few studies of work characteristics in the unhealthy group to support any conclusion. They find the evidence on anxiety to be less convincing and, in common with most other reviewers, they find no evidence that Type A or

Hostility is a risk factor in those with pre-existing heart disease and, rather more controversially, they also conclude that Type A or Hostility is not a risk factor in the healthy either. Incidentally they show that social support is a powerful protective factor in the healthy and unhealthy alike. Strike and Steptoe (2005) in a comprehensive review found that emotion and stress are important triggers for acute coronary syndromes.

Experiment (the effects of interventions)

By far the most convincing way of establishing causality is by experiment. In applied fields the experiment of choice is the randomised controlled trial in which the putative causal factor (stress, negative emotion etc) is reduced and, if the emotion is indeed a risk factor then the harmful consequence should also be reduced. Experiments of this type are expensive and difficult to mount and are consequently rare. I would like to discuss three RCTs, all with participants with pre-existing heart disease: Recurrent Coronary Prevention Program (RCPP), Enhanced Recovery in Coronary Heart Disease Patients (ENRICHED) and the exhaustion intervention trial (EXIT). RCPP (Friedman, Thorensen, Gill, Powell et al., 1984) was an attempt to reduce Type A Behaviour in survivors of a MI. It was wonderfully successful. Over a four-year period Type A behaviour was reliably reduced and recurrent MI fell by 50%. However it has not been influential, probably because of the consensus that Type A behaviour is not a risk factor in the group being studied. Scientifically the outcome is puzzling and practically, there is little appeal in altering a behaviour that appears to be unrelated to recurrent heart disease. Nevertheless something happened in the RCPP and we would benefit greatly from knowing what aspect of the complex intervention involved carried the therapeutic power, and through what mechanism. ENRICHED (Writing committee for ENRICHED investigators, 2003) has a much more secure epidemiological foundation. In this study post MI patients who were either depressed and/or perceived themselves as lacking social support (both well established risk factors) received interventions designed to deal with these risks. The results were disappointing. There was little specific effect of the intervention on depression or social support and no effect on recurrent MI. EXIT (Appels, Bar, van der Pol, Erdman, et al., 2005) was trial of a therapy designed to reduce vital exhaustion (a risk factor for recurrent MI that many see as akin to depression) in patients following angioplasty. It was equally disappointing. The intervention had little effect on exhaustion and absolutely no effect on reducing recurrent coronary events. These negative studies naturally lead one to ask if negative emotions can be

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usefully altered in patients following an acute coronary syndrome. Given the success of cognitive behavioural therapies in reducing emotional disorders it appears very unlikely that negative emotions cannot be helpfully reduced in patients with heart disease. Indeed we showed that a very simple intervention focusing on the patient's primary concerns reduced anxiety and depression in patients following an MI (Johnston, Foulkes, Johnston, Pollard & Gudmundsdottir, 1999).

Conclusion

- There has been an immense increase in the understanding of the mechanisms involved in heart disease. This new understanding is consistent with the view that emotional processes are risk factors for cardiovascular disease.
- There is evidence from laboratory and real life studies that stress and related emotions produce the physiological responses seen as critical in current biological models of coronary artery disease.
- There is epidemiological evidence that negative emotions, especially depression, work stress and acute stress and emotions are risk factors for Coronary Artery Disease and Acute Coronary Syndromes.
- There is not strong evidence that altering stress or emotion reduces heart disease.
- There is a need for studies that both reduce psychological risk factors and have enough power to detect effects of such reductions on heart disease

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