Post-traumatic stress disorder: breaking hearts

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This editorial refers to ‘Stiffness of large arteries and cardiovascular risk in patients with post-traumatic stress disorder’†, by J. Walczewska et al., on page 730

Worry and stress affects the circulation, the heart, the glands, the whole nervous system and profoundly affects heart action.

Charles William Mayo 1889–1968

When Charles William Mayo, the son of the founder of the Mayo Clinic, made this statement linking stress to cardiac disease, post-traumatic stress disorder (PTSD) was not a recognized entity. However, his words have since proved prophetic.

The modern world is not a peaceful place, with a number of long-term ongoing conflicts exposing ever more young men and women to the horrors of modern warfare. Inevitably this has led to an increasing number of individuals being diagnosed with PTSD. What effects this will inflict on sufferers when they reach older age are largely unknown. However, given the likelihood of ever-increasing numbers, this is an important question.

Although there is now considerable epidemiological evidence demonstrating an association between PTSD and cardiovascular (CV) disease,¹ the mechanisms remain unclear. Aortic stiffness has emerged as a major independent risk factor for CV disease in a number of population-based studies,² and a recent meta-analysis has confirmed that aortic pulse wave velocity (aPWV), the ‘gold standard’ for assessing aortic stiffness, is a strong predictor of CV events.³ The recent study of Walczewska et al. assesses aPWV in a cohort of subjects in their seventh and eighth decades of age who experienced PTSD following deportation to Siberia in their childhood.⁴ These individuals had greater aPWV, blood pressure, and incidence of CV disease compared with a matched group of subjects with no history of deportation and PTSD.

Studies on the association between PTSD and CV disease can be difficult to interpret due to the overlapping associations with psychological and physical illnesses as well as adverse health behaviours and substance misuse (Figure 1). However, if we assume that the association is directly causal, what are the potential mechanisms? The authors discuss the role of chronic activation of the sympathetic nervous system (SNS) associated with PTSD. Acute activation of the SNS does not alter aortic compliance,⁵ but there is evidence that long-term SNS stimulation, particularly in association with anxiety, results in long-term increases in arterial stiffness.⁶ Enhanced SNS activation has been demonstrated in people with PTSD through increased blood pressure and heart rate variability during ambulatory blood pressure monitoring⁷ and through loss of nocturnal dipping.⁸ There is also evidence to suggest that psychiatric diseases such as depression may be directly related to increased arterial stiffness⁹ and abnormal endothelial function even when compared with blood pressure-matched controls.¹⁰ Despite the role the authors suggest for the importance of chronic SNS activation in the aetiology of CV disease in the PTSD group, data for heart rate are not reported. This is a potentially important omission as heart rate is not only a useful marker of sympathetic activation but may also influence measured aPWV directly.¹¹ Blood pressure measurement with 24 h ambulatory recording would also have provided additional useful data regarding SNS activation. Another potential mechanism linking PTSD to arterial stiffness and cardiovascular disease is inflammation, which has been shown to promote both conditions. Evidence suggests that PTSD may induce chronic low-level inflammation, and a recent population-based study has demonstrated that inflammation is positively associated with an increased aPWV 20 years later.¹² Again, it is therefore unfortunate that the authors provide no data on markers of inflammation in their cohort.

Although this study is to be commended for focusing attention on the important issue of CV risk among survivors of traumatic events, there are a number of methodological issues that need to be considered in the interpretation of the study as a whole.

First, (estimated) mean blood pressure (MAP) is >10 mmHg higher in the PSTD group compared with the control group. Since aPWV is influenced by distending pressure,¹³ the difference between the groups will be smaller after correction for the difference in MAP. Indeed, since the difference in aPWV between the PTSD group and controls was already small, and only just significant (P = 0.042), it may have disappeared on correction for MAP. A similar argument applies to the graded relationship of increasing aPWV seen across the PTSD severity groups. Information regarding additional confounding influences such as customary alcohol intake and smoking is limited, both of which may be important mediating factors between PTSD and CV disease. Increased rates of alcohol dependence are well recognized

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among survivors of PTSD, and alcohol use is also associated with increased blood pressure and cerebrovascular disease. Similarly, higher smoking rates are associated with PTSD and result in increased cardiovascular risk. Although in this study current smoking rates are higher in the placebo group, no data regarding lifelong tobacco use are available and the rate in the PTSD group may be influenced by a higher rate of cessation following diagnosed CV disease.

Finally, the results of the study may be influenced by the ‘healthy volunteer effect’ since the PTSD group is being compared with a group of volunteers from a general practice database who are likely to exhibit better health behaviours and risk profiles.

Perhaps most importantly, this study raises another key issue: access to optimal medical care for high-risk and deprived groups across Europe. While the PTSD group may face many barriers to care, these results suggest that despite the high rates of vascular disease and vascular risk factors there is suboptimal treatment with statins and anti-hypertensives, and high levels of obesity and smoking. While physicians of our generation cannot right the wrongs of the past, we can do our best with the care of the survivors in the future.

Conflict of interest: none declared.

References