This issue of *Heart* focuses on exercise and cardiovascular (CV) health featuring a state-of-the-art review on the basic science behind the CV effects of exercise by Prof Matthew Wilson (see page 758), jointly published with the *British Journal of Sport Medicine*. The effects of a prolonged period (>6 months) of regular intensive exercise include a decrease in resting heart rate by 5 to 20 beats per minute, an increase in stroke volume by about 20% and increased myocardial contractility. Myocardial mass increases with a small increase in wall thickness along with increased chamber volumes (Figure 1).

The physiological adaptation to exercise differs from pathological cardiac remodeling at the tissue level both in terms of activation of signaling pathways and upregulation of growth factors. Exercise induced adaptations of the endothelium and blood vessels also contribute to the beneficial effect of exercise. Prof Wilson concludes: “Since aerobic capacity is a prognostic marker of CV disease and mortality more than any other established risk factor, clinicians should promote the expansive benefits of exercise in all spectrums of society, be it the casual exerciser, the sedentary individual or those with established CV disease.”

In an editorial, Drs LaGerche and McMullen (see page 742) challenge us to look critically at the association between exercise and CV health. “As enthusiastic pro-exercise scientific clinicians/researchers, we share the optimism of our colleagues and seek to extol the virtues of exercise but we also take the opportunity here to critically appraise some of the limitations in the exercise science translational model”. They suggest several areas for future inquiry: (1) identification of impediments to exercise and explanations for individual variability in keenness for exercise, (2) delineation of the exercise dose-response curve, (3) avoidance of the simplistic hypothesis that the exercise response is binary (physiologic versus pathologic), (4) discovery of signaling pathways that might yield therapeutic targets, (5) recognition that laboratory results may not always translate to the clinical setting and (6) attention to differences in risks within a population allowing individualized exercise recommendations.

Cardiorespiratory fitness in children and adolescents is associated with lower rates of CV disease in adulthood. In an original research paper in this issue of *Heart*, Dr Ruiz and colleagues (see page 766) hypothesized that cardiorespiratory fitness in adolescents is associated with a more favorable CV health profile. In a multicenter cross-sectional study of 510 European teenagers, cardiorespiratory fitness was measured using the 20 meter shuttle test with maximum oxygen consumption (VO₂max) estimated using a standard equation. Ideal CV health was defined based on 7 factors – 4 behaviors (smoking, body mass index, physical activity and diet) and 3 measurements (total cholesterol, blood pressure and glucose). At least 4 factors were present in 87% of boys and 84% of girls, although only 1 girl had all 7 factors, and cardiorespiratory fitness was associated with increasing numbers of ideal CV health components (Figure 2).

Commenting on this article, Prof Siegel and colleagues (see page 745) summarize the connection between genetics, personal health behaviours and development of CV disease.
disease (Figure 3) and raise the concern that fitness and levels of physical activity are partly related to heritable factors which “may exert their influence through modulation of motivational drive towards exercise behaviour, or through sheer physical capacity such as innate muscle size, strength and endurance, and untrained peak VO₂.” They conclude: “it is clear that physically fit children are more likely to be physically active adults and have a lower risk of CV disease most likely by lowering risk factors such obesity, dyslipidaemia, inflammatory mediators and hypertension.” and that “greater efforts are needed in understanding how to improve physical activity and fitness in children, to reduce their risk of preventable disease and to address the problem globally.”

Despite the beneficial CV effects of exercise in most patients, there are concerns about the possible risk of exercise in some patient groups, for example those with connective tissue disorders, such as Marfan syndrome. Clinical recommendations are primarily based on the theory that increased aortic wall mechanical stress due to the physiological effects of exercise might lead to adverse events; however our evidence base is weak with little clinical outcomes data. Drs Cheng and Owens (see page 752) provide a comprehensive and thoughtful review of the effects of exercise on aortic remodeling, the current published data on exercise related outcomes and current guidelines for exercise in Marfan syndrome patients.

The Education in Heart article in this issue builds on the theme of exercise and heart disease with a review of the effects of cold and exercise on the cardiovascular system by Dr Manou-Stathopoulou and colleagues (see page 808). This article reviews coronary physiology and then shows how physiologic changes are associated with clinical events in response to cold or exercise stress. In patients with underlying atherosclerotic coronary disease, cold stress is associated with increased angina symptoms and a higher incidence of acute myocardial infarction, most likely due to a relative reduction in coronary blood flow and increase in myocardial work (Figure 4). Further, the combination of exposure to cold and exercise...
may result in ischemia at a lower workload than during warm weather.

The Image Challenge case (see page 793) in this issue shows an interesting imaging finding in a 23 year old man with ventricular tachycardia after aortic valve replacement surgery for infective endocarditis. See if you can make the diagnosis based on the continuous wave Doppler tracing!

Figure 4  Vasomotor response of atherosclerotic coronary artery to cold stress. Vasodilatory impulses arise from (A) direct \( \beta_2 \)-adrenoceptor-mediated response to increased adrenaline concentration and (B) metabolic hyperaemia resulting from increased cardiac work (as per figure 4). Note there is minimal contribution from NO and EDHF due to endothelial dysfunction. Vasoconstrictive impulses are the same as in healthy coronary arteries. Due to a relative lack of NO and EDHF, vasoconstriction impulses dominate on coronary arterial vessel tone. In addition to the direct effects of a dominant vasoconstrictive vasomotor response, coronary blood flow will be further impeded by the presence of significant coronary artery stenosis. Ach, acetylcholine; Ad, adrenaline; CO, cardiac output; EDHF, endothelium-derived hyperpolarising factor; M3, muscarinic receptor; NA, noradrenaline; NO, nitric oxide; \( \alpha_1 \), \( \alpha_1 \) adrenergic receptor; \( \beta_2 \), \( \beta_2 \) adrenergic receptor.