Editorial

The predictive value of cardiorespiratory fitness

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This editorial refers to “The predictive value of cardiorespiratory fitness for cardiovascular events in men with various risk profiles: a prospective population-based cohort study”† by J.A. Laukkanen et al. on page 1428

More than 2000 years ago our cultural roots point to a rather active role of the school system in promoting greater activity in play and competition. The old Greek gymnasia considered exercise and physical activity to be at least as important as intellectual education.

Today the myth of everlasting youth, beauty, happiness and freedom of disease is, more than ever, a part of the lifestyle and identity of the western world. Fitness is one of its artificial parts, being achieved mainly by training, through sportive indoor and outdoor activity and resembling the exercise capacity that was formerly acquired by activities necessary to cope with everyday life. Fitness training has become a well-established part of wellness and providing outfits for training is a growing industry. Thus, the importance of fitness in health and disease is also an important issue in our health system.

The role of exercise for health has been investigated in numerous studies and surveys and the association of coronary artery disease and sedentary lifestyle has stimulated recommendations from many societies including the American Heart Association,1 the American College of Cardiology,2 the centres for disease control3 and the NIH4 to recommend regular aerobic exercise. Without doubt, a more sedentary lifestyle is related to classical coronary risk factors, like elevated low density and reduced high density lipoprotein, cigarette smoking, hypertension and others. Exercise has antiatherogenic effects, mainly by influencing classical coronary risk factors, has antithrombotic effects, has favourable effects on endothelial function, modulates the sympathetic nerve activity, has anti-ischaemic effects particularly by lowering the resting heart rate, and decreases the risk of ventricular fibrillation.

So far, exercise capacity has been clinically estimated on the basis of speed and grade of treadmill or external work performed on the bicycle. In studies using this technique exercise capacity has been shown to be a more powerful predictor of mortality than other established risk factors for cardiovascular disease.5 However, these studies did not establish a causal relationship between exercise capacity and overall mortality. Furthermore, peak oxygen consumption was not measured directly, which is a more accurate and reproducible measure and has been shown to be an excellent predictor of outcome in various diseases. Recently, large scale investigations of exercise capacity derived from peak cardiorespiratory exercise data have been used to study the relationship between VO$_{2\text{max}}$ and risk of cardiac and all cause death6 and in heart failure cardiopulmonary exercise test results have been established as outstanding prognostic information.7

In this issue of the Journal, Laukkanen et al.8 report a large group of 2361 eastern Finnish men evaluated by cardiopulmonary exercise testing between 1984 and 1989 and followed for an average of almost 14 years. At baseline 1294 patients were apparently healthy, 13% of them died during the follow-up period, while 1057 had a history of coronary artery disease, typical angina, heart failure, claudication, stroke, cardiomyopathy, arrhythmia, chronic obstructive pulmonary disease, pulmonary tuberculosis, bronchial asthma or cancer, 23.7% of them died during follow-up. While earlier studies have established exercise capacity or fitness mainly as a dependent risk factor, especially for coronary artery disease morbidity and mortality, the study by Laukkanen

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established VO2peak as a risk predictor for cardiovascular disease providing prognostic information independent of traditional coronary risk factors, like cholesterol, smoking, hypertension. The relative risk for cardiovascular death in healthy subjects after adjustment for classical risk factors was 0.85 in otherwise healthy people and 0.71 in people with pre-existing disease (see above). The risk reductions for coronary heart disease death and overall death for an increase of exercise capacity equal to one metabolic equivalent (1 MET) were similar in various subgroups of patients. Derived from the presence or absence of classical risk factors, the reduction in coronary heart disease death relative risk was 28 to 51%. Given the weakness of the definition of non-fatal events (24 h survival after an event) a more in depth analysis of fatal events is interesting in this study: The risk of death increased in unhealthy subjects with VO2max below 21.2 ml O2/kg/min was 4.5-fold higher than in patients with a VO2max above 32.4 mlO2/kg/min. Similarly, the risk of death was again more than 4.5-fold higher in healthy subjects with a VO2max below 27.6 ml O2/kg/min compared to a VO2max above 37.1 ml O2/kg/min. This rise was even greater for unhealthy subjects if fatal cardiac events alone were evaluated.

In healthy men with and without risk factors, there was a marked increase in overall death rate with reduction in oxygen consumption but the number of risk factors markedly influenced the overall risk. However, while there was a marked influence of fitness in men unhealthy at study entry, the influence of classical risk factors became negligible in the subgroup of people who had an oxygen consumption below 21.2 ml O2/kg/min. Thus, the most important results for clinicians are that tors became negligible in the subgroup of people who were healthy at study entry, the influence of classical risk factors markedly influenced the overall risk. However, spontaneous VO2max and trained VO2max do not necessarily have the same prognostic impact; like the absence of ventricular arrhythmia during its treatment has a different prognostic impact as the absence of ventricular arrhythmia without medical intervention. The improvement through training of depressed oxygen consumption caused by pulmonary or cardiovascular disease might modify the outcome parameter without affecting outcome because the underlying disease itself is still present and might be the prognostic limiting factor. A more in-depth analysis of cardiopulmonary exercise test results than was possible in this study by Laukkanen may greatly enhance our knowledge of the mechanisms and the importance for the improvement of exercise capacity in training programmes. In specific cardiovascular diseases, like heart failure and pulmonary hypertension, the in-depth analysis of physiological information from cardiopulmonary exercise testing has already improved our knowledge of the pathophysiology of exercise capacity.9–11 The more we learn about the effects of training on overall physiology and pathophysiology the more we will understand to which degree a decreased maximal oxygen consumption result is a hereditary phenomenon or a modifiable risk factor.

References