Whilst contributing to our understanding of the pathophysiology of chronic heart failure, the study by Schillinger and colleagues also highlights the increasingly recognized heterogeneous nature of this disorder. Improved insights into the molecular mechanisms involved in, for example, the genesis of lethal arrhythmias or the detrimental effects of sympathetic activation is essential and may facilitate the development of novel therapies. With the increasing complexity of available interventions in the management of chronic heart failure it is, however, likely that not all therapies will be applicable to each individual patient. In this respect patient profiling and targeted therapy is an appealing goal. Whilst this might begin with neurohormonal and cytokine assessment, more detailed evaluation at the cellular/molecular level may also be required.

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References


Exercise-related syncope: are athletes different from sedentary subjects?

See doi:10.1053/euhj.2001.3042 for the article to which this Editorial refers

Syncope seldom occurs during effort. For example, in one recent study, syncope during effort was reported in only 17 (5%) of 341 patients referred to three Syncope Units for evaluation of unexplained syncope. In the patients with structural heart disease, syncope during effort was a significant predictor of a cardiac cause of syncope (with an odds ratio of 3·1) with a specificity of 96\%\(^{11}\). In other words, in the patients with structural heart disease the occurrence
of syncope during effort almost invariably predicts a cardiac cause of syncope. In particular, in patients with bundle branch block a tachycardia-related (phase 3) exercise-induced second- and third-degree atrioventricular block has been shown to be invariably located distal to the atrioventricular node and is an ominous finding of progression to stable chronic atrioventricular block[2].

Conversely, syncope during effort was not associated with a cardiac cause of syncope in patients without structural heart abnormalities[1]. In these cases the syncope mechanism may be a manifestation of exaggerated reflex vasodilatation. Reflex syncope occurring during exercise is caused by marked hypotension without bradycardia[3].

Finally, syncope can occur immediately after exercise. Post-exertional syncope is almost invariably due to autonomic failure[4] or to a neurally-mediated mechanism[5-7] and is characterized by hypotension which can be associated with marked bradycardia or asystole; it typically occurs in subjects without heart disease. Tilt testing has been used to diagnose neurally-mediated syncope, which may manifest as post-exertional syncope[8]. A failure of reflex vasoconstriction during exercise in splanchnic capacitance vessels and in forearm resistance vessels has been shown in patients with vasovagal syncope[9].

In this issue, Colivicchi et al.[10] studied 33 competitive athletes who had one or more syncope episodes related to exercise. Syncope had occurred during exercise in seven cases, immediately after cessation of exercise in 16 cases and both during and after exercise in 10 cases. No athlete had structural heart disease. Therefore, the studied population belong to the group with syncope during effort without heart disease or the group with post-exertional syncope described above. Thus not surprisingly, the final diagnosis was neurally-mediated in most cases, no patient had a diagnosis of cardiac syncope and the outcome was excellent in all. The syncope-related syncope rate was 9% and 24% at 1 and 3 years, respectively, and was even lower than that observed in the general population of patients affected by multiple syncopes[11,12]. Thus, the authors conclude that exercise-related syncope is not associated with an adverse outcome in athletes without heart disease. No patient had syncope due to tachyarrhythmia, for example some forms of supraventricular tachycardia are not infrequent in young healthy people. This probably means that tachycardia-related syncope is unlikely to remain undiagnosed.

Two-thirds of the subjects had a positive response to tilt testing; their outcome was not different from those who had a negative response. This is in accordance with recent findings in the literature[12,13] showing that the patients with isolated unexplained syncope and those with a positive response to tilt testing have similar clinical characteristics, mechanism of syncope and outcome with a low recurrence rate for at least 1 year and a low risk of injury and adverse events. These findings strongly suggest that these patients may be part of the same population and, in both groups, the cause of syncope is probably neurally-mediated[12,13].

In the study by Colivicchi et al.[10], athletes underwent an extensive evaluation including Holter monitoring, exercise testing, electrophysiological study, electroencephalography, and CT scanning which was inconclusive. This was despite the fact that spontaneous syncope had occurred during physical exercise, an exercise test was unable to reproduce syncope and only in four cases was pre-syncope induced immediately after the end of exercise. The extensive work-up was justified by the fact that, in general practice, syncope in competitive athletes is regarded by many as a potentially ominous finding and its occurrence has a significant impact on the lay and medical community. The study by Colivicchi et al.[10] will have a great impact on sport physicians and physicians involved in qualification for competitive sports and will help to change their approach to the athletes with syncope. Hopefully, less diagnostic investigations should be required and the current guidelines should be modified.

In conclusion, in the absence of structural heart disease, syncope occurring during or immediately after exercise is invariably a benign condition, either in athletes or in the sedentary population; its aetiology is likely to be neurally-mediated irrespective of the result of tilt testing. There is no reason to consider athletes different from sedentary subjects. The absence of structural heart disease is the strongest predictor of good outcome. No other test seems useful for risk stratification as no test adds more to the prognostic significance given by the absence of heart disease; thus several investigations could be safely avoided. The study by Colivicchi et al.[10] suggests an important change to the current management of athletes with syncope.

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References