The effect of exercise training on autonomic cardiovascular regulation: from cardiac patients to athletes
Ferdinando Iellamo
Dipartimento Medicina Interna -IRCCS San Raffaele, Università “Tor Vergata”, Roma, Italy

Regular exercise training induces adaptational changes in the neural cardiovascular regulation, which can be easily assessed by employing non-invasive markers obtained through the analysis of HR and BP variability signals. Moderate intensity endurance exercise training has been shown to reduce sympathetic while concomitantly enhancing vagal modulation HR, as reflected by a decrease in low-frequency (LF) and an increase in high-frequency (HF) spectral components of HR variability (HRV), respectively, in hypertensive patients. This was accompanied by an increase in baroreflex sensitivity (BRS), that is typically depressed in these patients, thus confirming the enhanced vagal contribution to HR regulation induced by training. Similarly, a shift away from sympathetic toward enhanced vagal activity after training has also been reported in patients with chronic heart failure, implying an improvement of the abnormalities in the autonomic control of the circulation known to occur in this syndrome. Finally, exercise training positively affects cardiac autonomic function in patients with coronary artery disease (CAD), as indicated by the increase in HRV and BRS (1). The relevance of this finding is outlined by a recent study showing that the mortality rate during a 10-year follow-up was strikingly lower in post-myocardial infarction patients in which training had induced a significant increase in BRS (2).

It thus appears that exercise training is an effective nonpharmacological tool to improve neurovegetative control of the heart in patients with cardiovascular diseases, being associated with cardioprotective effects. However, the effects of training on neural cardiovascular regulation appear to be strikingly modulated by the exercise training load. This is exemplified by the findings obtained in athletes.

In high performance world class athletes, such as rowers, increasing training load up to 75% of maximum was associated with a progressive resting bradycardia, increased HF and reduced LF component of HRV with a concomitant increase in BRS, similar to cardiac patients. However, at 100% training load these effects were reversed, with increases in resting HR, BP, LF component of both HR and BP variability and decreases in HF component of HRV and BRS (up to 50%) in comparison to pre-training levels in longitudinal assessments performed during a whole training season culminating with the Rowing World Championship (3). Three out of seven athletes won medals in the World Championship performed 20 days after assessment at 100 % training load, thus excluding an "overtraining" effect. In a subsequent study with a similar design, the increase in LF component of HRV with the increase in training load was associated with a simultaneous decrease in T-wave voltage on surface ECG, another indicator of training status related to sympathetic nerves activity (4). The continuous link between training status and changes in T-wave voltage and HR variability indices is further supported by the finding of the reversal of sympathetic and vagal markers of sino-atrial node regulation and T-wave amplitude in the recordings made during the World Championships, when the the training load had been markedly reduced.

Analysis of HRV can be combined with other non-invasive and minimally time-consuming indicators, serves as a simple and valuable tool to assess neurovegetative adaptations not only to exercise training of cardiac patients, but also to follow the time course of neurovegetative response to competitive training, providing additional information useful to individually tailor exercise training programs in elite athletes.

REFERENCES