Cardiac autonomic imbalance in an overtrained athlete

RIKARD HEDELIN, URBAN WIKLUND, PER BJERLE, and KARIN HENRIKSSON-LARSEN
Sports Medicine, and Clinical Physiology, Department of Surgical and Perioperative Science, Umeå University, Umeå, SWEDEN; Centre for Musculoskeletal Research, National Institute for Working Life, Umeå, SWEDEN; and Department of Biomedical Engineering, Umeå University Hospital, Umeå, SWEDEN

ABSTRACT

HEDELIN, R., U. WIKLUND, P. BJERLE, and K. HENRIKSSON-LARSEN. Cardiac autonomic imbalance in an overtrained athlete. Med. Sci. Sports Exerc., Vol. 32, No. 9, pp. 1531-1533, 2000. Purpose: In order to investigate overtraining-related adaptations in the autonomic nervous system, cardiac autonomic activity was examined in a junior cross-country skier who presented with reduced performance in competitions, early breathlessness during training sessions, and accumulated central fatigue. Methods: Power spectral analysis of heart rate variability (HRV) was performed before, when overtrained (OT), and after recovery (Rec). Results: In the overtrained state, high frequency (HF) and total powers in the lying position were higher compared with before and after. In normalized units, the increased HF in OT was even more prominent and clearly higher than in any control subject, and it was reversed in Rec. Resting heart rate was slightly reduced in OT and returned to baseline in Rec. Conclusions: The shift toward increased heart rate variability, particularly in the HF range, together with a reduced resting heart rate suggest a cardiac autonomic imbalance with extensive parasympathetic modulation in this athlete when overtrained. Key Words: OVERTRAINING, HEART RATE VARIABILITY, STAINESS, PARASYMPATHETIC

The overtraining syndrome is a result of an excessive training load together with low quality recovery and is characterized mainly by reduced performance and pronounced fatigue. Other symptoms are numerous and the lack of uniform features has made the diagnosis difficult. A relative parasympathetic dominance has been proposed to be associated with the overtraining syndrome in endurance athletes, although this has not been further investigated (6). By examining fast (high frequency; HF) and slow (low frequency; LF) modulators of heart rate, it may be possible to detect alterations in autonomic balance on the heart. The high frequency, or fast-acting, component of beat-to-beat, heart rate variability (HRV) is mediated through respiration and is generally believed to represent vagal activity (1). Although sympathetic activity appears to influence low frequency HRV, there is no consensus in the literature regarding a reliable marker of sympathetic activity. The high and low frequency components of HRV can be quantified by means of power spectral analysis. The aim of this study was to investigate whether spectral analysis of HRV can confirm the increased parasympathetic activity suggested in the overtraining syndrome.

METHODS, HISTORY, AND RESULTS

After several months of intensive training, peaking at 20 h of training-wk⁻¹, this cross-country skier (age: 16; VO₂max: 71 mL·kg⁻¹·min⁻¹) reported accumulated central fatigue, reduced performance at standardized bicycle work, and early breathlessness during training sessions. He also performed worse in competitions. Testing for infections (cytomegalovirus, Epstein-Barr, and enterovirus), pulmonary function, and exercise ECG (myocarditis) were all normal/negative. Hemoglobin, white blood cells, ferritin, and cortisol were in the normal range but luteinizing hormone (LH) was reduced and below normal when overtrained (1.4 IU·L⁻¹ when overtrained vs 2.3 IU·L⁻¹ after recovery). Profile of mood states (11) changed from an “iceberg” shape with a global score (G) of 110 to a G-score of 132 which was due mainly to increases in tension (7→17, healthy vs overtrained), anger (8→14), and in depression-scores (3→14) despite a relatively unchanged vigor score. He was exhorted to rest and after a recovery period of almost two months he regained previous work capacity.
Electrocardiographic recordings were performed in the lying position at rest during controlled breathing (12 breaths-min⁻¹), and after a tilt to a 70° upright position. Power spectral analysis (frequency domain) of cubic spline interpolated heart rate data was performed by autoregressive modelling as previously described (2). The spectral power in the low frequency (LF; 0.04–0.15 Hz) and high frequency (HF; 0.15–0.45 Hz) bands were calculated and log-transformed. Normalized units (nu) was obtained by dividing the actual measure (e.g., HF-power) with the sum of LF and HF powers. The HRV data was also compared with data from seven normally responding male XC-skiers of the same age examined before and after the same training period.

Although HF and total powers in the lying position were initially high, both increased in OT and then returned to within the control group range. The reduction of resting heart rate, although small, also supports the possibility of an increased parasympathetic activity. If the response in parasympathetic activity only reflects a change in training load or truly is associated with changes in performance is, however, difficult to say. Although some studies have shown a relationship between parasympathetic activity and physical fitness, this relationship is a controversial issue (4,7).

Signs of an adaptation in sympathetic activity are more difficult to interpret from these data. Although the reduced resting heart rate also could result from reduced sympathetic activity, there is no HRV measure that for certain can confirm such a mechanism. A current issue is to what extent sympathetic activity really can be interpreted from LF-power of HRV. The origin of LF variability has been attributed to fluctuations in blood pressure caused by sympathetic modulation of vascular resistance, which result in parallel, vagally mediated changes in heart rate by feedback through the baroreflex (3,5). The high HF (nu) in the lying position could thus be the result of a reduced LF-power of sympathetic origin. On the other hand, a reduced sympathetic activity should be in that case appear in the LF band after tilt. After tilt in OT, LF in absolute units was slightly reduced but LF (nu) was unchanged. According to this, we cannot exclude the possibility that the LF reduction, seen in

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DISCUSSION

The reported symptoms of physical inability in view of the training history together with regained performance only after several weeks of rest supports the diagnosis of an overtraining syndrome (staleness) in this athlete (8). In the search for responsible mechanisms of overtraining-related symptoms, one area of interest has been changes in autonomic function. Altered catecholamine release (nocturnal urinary and exercise-induced plasma) has been shown in overtrained athletes indicating an adaptation of the sympathetic nervous system (9). Further, in the overtraining syndrome, an increase in parasympathetic relative to sympathetic activity has been suggested but not confirmed (6). Since the 1980s, spectral analysis of HRV has offered a new way of evaluating autonomic balance. From HRV recordings, HF and total powers are generally accepted to reflect parasympathetic activity in the supine position (1,12). The remarkably high HF- and total powers in the lying position at the time of overtraining syndrome, together with a considerable HF dominance, suggest an increased parasympathetic activity in this athlete when overtrained. After recovery, these values returned to within the control group range. The reduction of resting heart rate, although small, also supports the possibility of an increased parasympathetic activity. If the response in parasympathetic activity only reflects a change in training load or truly is associated with changes in performance is, however, difficult to say. Although some studies have shown a relationship between parasympathetic activity and physical fitness, this relationship is a controversial issue (4,7).

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Signs of an adaptation in sympathetic activity are more difficult to interpret from these data. Although the reduced resting heart rate also could result from reduced sympathetic activity, there is no HRV measure that for certain can confirm such a mechanism. A current issue is to what extent sympathetic activity really can be interpreted from LF-power of HRV. The origin of LF variability has been attributed to fluctuations in blood pressure caused by sympathetic modulation of vascular resistance, which result in parallel, vagally mediated changes in heart rate by feedback through the baroreflex (3,5). The high HF (nu) in the lying position could thus be the result of a reduced LF-power of sympathetic origin. On the other hand, a reduced sympathetic activity should in that case appear in the LF band after tilt. After tilt in OT, LF in absolute units was slightly reduced but LF (nu) was unchanged. According to this, we cannot exclude the possibility that the LF reduction, seen in
absolute units, reflects an attenuated sympathetic cardiovascular modulation in the overtrained state. However, because HF in absolute units was very high in the lying position, the high HF in normalized units is likely an effect of increased parasympathetic activity. Besides, the most striking findings were seen during parasympathetic stimulation (controlled breathing). A sympathetic adaptation has been described in overtrained athletes, but that finding was based on the secretion of catecholamines (10).

Despite the limitations of a one-subject case study, our data give some support to the theories of Israel (6) of a relative parasympathetic dominance in the endurance-type overtraining syndrome. Cases of the full-blown, spontaneously developed overtraining syndrome are not easy to obtain to make a basis of statistical conclusions, but, of course, findings like ours need to be confirmed in more subjects.

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Address for correspondence: Karin Henriksson-Larsén, M.D., Ph.D., Sports Medicine, Umeå University, S-901 87 Umeå, Sweden; E-mail: karin.larsen@idrott.umu.se.

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