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Opinion Article

Heart Rate Recovery in Adults with Chronic Obstructive Pulmonary Disease

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DESCRIPTION

Exercise causes a decrease in vagal tone, which contributes to an increase in Heart Rate (HR), and vagal reactivation, which causes the heart rate to rise again after exercise (HRR). Slow HRR is the decrease in heart rate from the peak heart rate (HR peak) one minute after the conclusion of activity that is less than 12 or 14 bpm. In those with and without cardiovascular or respiratory conditions, a sluggish HRR is a predictor of overall mortality. An increased incidence of acute exacerbations was seen in people with Chronic Obstructive Pulmonary Disease (COPD) who had a low HRR following the Six Minute Walking Test (6MWT). It has been demonstrated that therapeutic measures like exercise training or oxygen supplementation can enhance Autonomic Nervous System (ANS) function and HRR in people with COPD.

When compared to their healthy peers, children with asthma had a considerably slower HRR, according to a recent study. The ability to predict and identify the danger of a less HRR following exercise may enhance clinical management (e.g. exercise training programs). Therefore, the purpose of this study was to compare adults with COPD to those with asthma in order to determine the prevalence of slow HRR (defined as post-exercise decline in HR 12 bpm). We assessed the traits of persons with slow HRR as secondary goals. This is the first study to look at how common slow HRR is in adults with asthma; it was shown that at least 30% of them had the condition, which is substantially less common than COPD patients. Only HR for the recruited populations was a reliable indicator of delayed HRR. Asthma has been linked to ANS imbalance, as evidenced by the fact that children and teenagers with asthma had slower HRR following a field test than their peers. In those with cardiac vascular illnesses, a sluggish HRR may be a sign of autonomic dysfunction brought on by an unbalanced sympathovagal system. The examination of HR variability revealed a sympathetico-vagal imbalance in people with asthma as well. However, HRR was not reported in these investigations. According to studies, children with asthma who have delayed HRR are more likely to have poor exercise tolerance. Participants in our study who had slow HRR walked much less than those who had normal HRR.

It has been proposed that the parasympathetic branch of the Autonomic Nervous System (ANS) may have a role in the etiology of asthma. Asthmatic people may have different changes in ANS function after exercise than do non-asthmatic people, according to a number of studies. The cardiac response to different autonomic function tests has shown that there is actually an increase in cardiac vagal responsiveness in asthma.

The idea of system-independent ANS control is supported by additional investigations that found no correlation between bronchial and cardiac vagal tone. After a 6MWT, the prevalence of HRR in our study was assessed. Maximum exercise is not necessary for HRR to be useful in clinical settings. The predictive value of HRR in patients with heart failure following the 6MWT and symptom-limited cardiopulmonary exercise test was investigated by the authors. The outcomes demonstrated that HRR following the 6MWT is a potent predictor that functions similarly to HRR following maximal activity. Other authors also employed the 6MWT to evaluate the HRR in people with chronic respiratory illnesses, demonstrating the utility of this field test. At least 30% of these persons have sluggish HRR, which is a much lower prevalence than in COPD sufferers.

After a 6MWT, no baseline feature can forecast this outcome. It will be useful to assess prospective studies to determine the best type of exercise (walking *vs.* cycling, incremental *vs.* endurance) to provoke this event in adult asthmatics.

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