tive pulmonary disease," published online in Anatolian Journal of Cardiology 2016 Feb 10 (1). In their Letter-to-Editor, the authors emphasize that epicardial fat is an endocrine organ secreting various pro-inflammatory cytokines and is associated with inflammatory processes, including subclinical atherosclerosis. Previous studies have shown a relation between epicardial fat thickness (EFT) and visceral adiposity, metabolic syndrome, cardiovascular (CV) disease, and pro-inflammatory activity. Because a vast majority of our COPD patients had emphysematous-type disease, there were few obese (BMI >30) patients.

As the authors stated, magnetic resonance imaging (MRI) or computed tomography (CT) provide best images to assess the amount of epicardial fat; however we used transthoracic echocardiography (TTE) because it is a cheap, easily available, reproducible, and radiation-free imaging technique that we used in concordance with the description of Iacobellis et al. (2). Poor echogenicity was the reason for exclusion from the study in eighty patients. Lack of MRI/CT data regarding EFT in our patient population should be mentioned as a limitation of our study. We were unable to calculate the intra- and interobserver variabilities for EFT measurement; this was included in the study limitations. We also excluded patients with hypothyroidism, either apparent or subclinical.

As the authors stated, epicardial fat thickness has recently been shown to be associated with subclinical atherosclerosis in patients with inflammatory processes, including psoriasis, hypothyroidism, etc. Chronic obstructive pulmonary disease (COPD) is one of those diseases in which inflammation plays a key role in the pathogenesis and disease progression. Thus, our study supports the assertion that CV risk may increase as the EFT values increase in COPD patients.

In conclusion, further studies on EFT in COPD patients should consider the abovementioned concerns and limitations.

Melike Demir, Murat Yüksel
Department of Chest Disease, *Cardiology, Faculty of Medicine, Dicle University; Diyarbakır-*Turkey*

References

1. Demir M, Acet H, Kaya H, Taylan M, Yüksel M, Yılmaz S, et al. Relationship between metabolic syndrome and epicardial fat tissue thickness in patients with chronic obstructive pulmonary disease. Anatol J Cardiol 2016 Feb 10. Epub ahead of print.
2. Iacobellis G, Assael F, Ribaudo MC, Zappaterreno A, Alessi G, Di Mario U, et al. Epicardial fat from echocardiography: a new method for visceral adipose tissue prediction. Obes Res 2003; 11: 304-10.

Address for Correspondence: Dr. Melike Demir
Dicle Üniversitesi Tıp Fakültesi, Göğüs Hastalıkları Bölümü
Sur, Diyarbakır-*Türkiye*
Phone: +90 412 248 80 01 Fax: +90 412 248 85 23
E-mail: melikedoktor@hotmail.com

Erectile dysfunction and heart rate recovery. Is it autonomic nervous system?

To the Editor,

Ulucan et al. (1) observed in their valuable and interesting study that heart rate recovery (HRR) indices were attenuated in patients with erectile dysfunction when compared with healthy controls entitled " Deterioration of heart rate recovery index in patients with erectile dysfunction" published Anatol J Cardiol 2016; 16: 264-9. In addition, they observed in their analysis that HRR at 1 min (HRR1) and 3 min were independently associated with the presence of erectile dysfunction. Exercise testing can be abruptly terminated (cessation of exercise) with the patient in the standing or sitting positon (no "cool-down" period) or when the patient keeps walking in a predetermined speed and inclination (cool-down period), which can be a 2-min cool-down at 1.5 mph at 2.5 grade or 1-min cool-down at 1 mph at 0% inclination (2, 3). In protocols using cool-down, HRR1 is calculated by taking the difference between the heart rate at peak exercise and heart rate 1 min later, which is 1 min after the beginning of the cool-down period (2). Similarly, in exercise tests that stop abruptly, HRR1 is calculated by taking the difference between the heart rate at peak exercise and heart rate 1 min later at which time the patient is in complete rest in the supine or sitting positon. Abnormal HRR1 is usually defined as heart rate that declines to ≤12 beats/min in the first minute after exercise for protocols that use a post-exercise cool-down or ≤18 beats/min in the first minute post exercise for protocols that abruptly stop exercise (2, 4). Because the authors used post-exercise cool-down protocol, HRR1 ≤12 beats/min might be assumed to be abnormal in this case. HRR1 was 34.8± 1.2 in patients with erectile dysfunction in the authors' study. Thus, one should be very careful in interpreting their results. Approximately 95% of HRR1 was, statistically, between 12.4 and 57.2 beats/min in patients with erectile dysfunction. Therefore, we can assume that virtually all patients with erectile dysfunction had a normal HRR1. Hence, it might be misleading to suggest that patients with erectile dysfunction have impairment in autonomic nervous system. Looking carefully at the data, maximal heart rate was 158.2±18.7 beats/min in patients with erectile dysfunction and 167.2±16 beats/min in controls. Accordingly, most of the differences between patients with erectile dysfunction and controls with regard to HRR indices were due to lower heart rate attained at peak exercise in patients with erectile dysfunction. Namely, chronotropic incompetence, which might be due poor physical fitness, could be responsible for differences in HRR in this study population as demonstrated before (5). I believe that caregivers should be familiar with parameters gleaned from a standard exercise test and interpreting the results gained from it.

Abdullah Tekin
Department of Cardiology, Faculty of Medicine, Başkent University; Ankara-*Turkey*

References

1. Ulucan Ş, Kaya Z, Keser A, Katlandur H, Karanfil M, Ateş İ. Deterioration of heart rate recovery index in patients with erectile dysfunction. Anatol J Cardiol 2016; 16: 264-9.
2. Cole CR, Blackstone EH, Pashkow F, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. N Engl J Med 1999; 341: 1351-7.
3. Maddox TM, Ross C, Ho PM, Masoudi FA, Magid D, Daugherty SL, et al. The prognostic importance of abnormal heart rate recovery and chronotropic response among exercise treadmill test patients. Am Heart J 2008; 156: 736-44.
4. Lauer MS, Mehta R, Pashkow FJ, Okin PM, Lee K, Marwick TH. Association of chronotropic incompetence with echocardiographic ischemia and prognosis. J Am Coll Cardiol 1998;32:1280–6.
5. Desai MY, De la Peña-Almaguer E, Mannting F. Abnormal heart rate recovery after exercise as a reflection of an abnormal chronotropic response. Am J Cardiol 2001; 87: 1164-9.

Address for Correspondence: Dr. Abdullah Tekin
Başkent Üniversitesi Tıp Fakültesi
Kardiyoloji Anabilim Dalı, Yüreğir, Adana-*Türkiye*
Phone: +90 322 327 27 27 Fax:+90 322 327 12 86
E-mail: tekincardio@yahoo.com
©Copyright 2016 by Turkish Society of Cardiology - Available online at www.anatoljcardiol.com
DOI:10.14744/AnatolJCardiol.2016.7231

Author`s Reply

To the Editor,

We read the letter about our article entitled "Deterioration of heart rate recovery index in patients with erectile dysfunction" published in the April issue of Anatolian Journal of Cardiology 2016; 16: 264-9 (1). The authors evaluated only the HRR1 parameter and stated "It would be too generous to claim that patients with erectile dysfunction (ED) have impairment in autonomic nervous system (ANS)."

First, we analyzed all heart rate recovery (HRR) indices (HRR1, HRR2, HRR3, and HRR5) and not only HRR1 to determine the ANS dysfunction in ED patients. In our study, we found significant differences in all parameters between control and patient groups. Although the author's comment about HRR1 is acceptable, other HRR indices, especially HRR2, had an abnormal range in ED patients, whereas it had a normal range in the healthy group. Furthermore, we found that HRR1 and HRR3 were independent risk factors for ED in linear multivariate regression analysis.

When all these results are evaluated together, the significant differences in HRR indices between the two groups are thought to be associated with both low exercise capacity and ANS dysfunction in ED patients.

Zeynettin Kaya
Department of Cardiology, Faculty of Medicine, Mevlana University; Konya-*Turkey*

Reference

1. Ulucan Ş, Kaya Z, Keser A, Katlandur H, Karanfil M, Ateş İ. Deterioration of heart rate recovery index in patients with erectile dysfunction. Anatol J Cardiol 2016; 16: 264-9.

Address for Correspondence: Dr. Zeynettin Kaya
Mevlana Üniversitesi Tıp Fakültesi, Kardiyoloji Bölümü
Yeni İstanbul Cad. No: 235
42003 Selçuklu/Konya-*Türkiye*
E-mail: zeynettinkaya@yahoo.com

Heart rate recovery, cardiac rehabilitation, and erectile dysfunction in males with ischemic heart disease

To the Editor,

I have read the article entitled "Heart rate recovery, cardiac rehabilitation, and erectile dysfunction in males with ischemic heart disease" by Kałka et al. (1) with great interest, which was recently published in Anatolian Journal of Cardiology 2016; 16: 256-63. The investigators reported that in patients with ischemic heart disease (IHD) and erectile dysfunction (ED) subjected to cardiac rehabilitation, enhancement of autonomic balance assessed using heart rate recovery (HRR) plays a significant role in the mechanism of improvement in erection quality (1). Authors have reported that there was no significant difference with regard to beta-blocker therapy (1).

Beta-blockers are one of the most commonly used and cornerstone therapy in the treatment of ischemic heart disease (2). Nebivolol is a third-generation beta-blocker, and has a vasodilating effect that is attributed to the generation of endothelial nitric oxide, in addition to β1-adrenoceptor selectivity (3).

It is well known that beta-blocker therapy effect might be different with regard to ED depending on sort of it in patients with IHD (4). Aldemir et al. (4) have reported that although ED in males undergoing CABG surgery decreases when metoprolol is used, nebivolol had a protective effect on the sexual activity of men undergoing coronary artery bypass surgery with cardiopulmonary bypass. In addition, Brixius et al. (5) have reported beneficial effects of nebivolol on the erectile function in hypertensive men.

I would like to emphasize one important point to clarify in this article. Kind of beta-blocker therapy is very important to evaluate ED in patients with IHD (3–5). Therefore, authors should mention kind of beta-blocker therapy used in this study group.

In conclusion, ED is more common in men with IHD. Nebivolol, a third-generation beta-blocker, seems to have beneficial effects on ED compared with metoprolol (3–5). Kind of beta-blocker therapy might affect ED in patients with IHD.