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**Available Online Date:** 25.12.2014

## Heart rate recovery and methodological issues

To the Editor,

We read with great interest the article, entitled "Heart rate recovery may predict the presence of coronary artery disease" by Akyüz et al. (1) published in *Anatolian J Cardiol* 2014; 14: 351-6.

They observed in a retrospective analysis that abnormal heart rate recovery at 1 min (HRR1) was associated with the presence of angiographically proven coronary artery disease. This study strengthens previous research that the heart rate information gleaned from a standard exercise test can be used to supplement prognostic and diagnostic data. There are some methodological issues that need to be clarified in order to understand how these data were obtained. The authors' statement that "post-exercise HRR was measured in the sitting position during the cool-down period after the cessation of peak exercise" might lead to misunderstandings and is inappropriate with regard to terminology. Exercise testing can be terminated (cessation of exercise) abruptly with the patient in the standing or sitting position (no 'cool-down' period), or the patient keeps walking in a predetermined speed and incline (cool-down period), which can be a 2-minute cool-down at 1.5 mph on a 2.5° grade or a 1-minute cool-down at 1 mph at 0% incline (2, 3). In protocols using cool-down, heart rate recovery at 1 minute is calculated by taking the difference between the heart rate at peak exercise and heart rate 1 minute later, which is 1 minute after the beginning of the cool-down period (2). Similarly, in exercise tests that stop abruptly, heart rate recovery at 1 minute is calculated by taking the difference between the heart rate at peak exercise and heart rate 1 minute later, at which time the patient is at complete rest in the supine or sitting position. Abnormal HRR1 is usually defined as heart rate that declines  $\leq 12$  beats/min in the first minute after exercise for protocols that use a post-exercise cool-down or  $\leq 18$  beats/min in the first minute

postexercise for protocols that stop exercise abruptly (2, 4). Since the authors defined abnormal HRR1 as  $\leq 21$  beats, we assume that there was no cool-down period in their study. Although the authors mentioned heart rate reserve in the results section and tables, they did not define it in the methods. It is not clear whether heart rate reserve is in beats per minute or in percentages. Heart rate reserve in beats per minute is calculated as [(220-age in years) - resting heart rate in beats per min], while heart rate reserve in percentages is calculated as (peak heart rate- resting heart rate in beats per min)/[(220-age in years) - resting heart rate in beats per min] multiplied by 100 (2). Heart rate reserve in percentages is also an indicator of chronotropic response. Heart rate reserve below 80% is considered to be evidence of an impaired chronotropic response, which is a powerful indicator of mortality (5). We believe that caregivers should be familiar with these parameters and consider for routine incorporation into exercise test interpretation.

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**Available Online Date:** 25.12.2014

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DOI:10.5152/akd.2014.6021



## Author's Reply

To the Editor,

We would like to thank the authors for their comments and criticism of our original investigation (1), entitled "Heart rate recovery may predict the presence of coronary artery disease," published in *Anatolian J Cardiol* 2014; 14: 351-6. We wrote in the methodology section that "post-exercise HRR was measured in the sitting position during the cool-down period after the cessation of peak exercise." "Cooling down" commonly refers to easy exercise following strenuous exercise. In contrast, the "cool-down period" refers to the length of the warming-down time. In the

manuscript, the phrase "during the cool-down period after the cessation of peak exercise" means during the length of early recovery time after peak exercise. We retrospectively enrolled subjects in the sitting position during the recovery phase who had exercise testing abruptly terminated. The values of  $\leq 12$  beats/min in the first minute after exercise for protocols that use a post-exercise cool-down and of  $\leq 18$  beats/min in the first minute postexercise for protocols that stop exercise abruptly have prognostic value, especially in predicting mortality (2, 3). However, these two values were generally not accepted for determining the presence of coronary artery disease (CAD). Georgoulis et al. (4) used an HRR1 of  $\leq 21$  beats/min after abruptly stopping exercise for determining the presence of CAD. Hence, an HRR1 value of  $\leq 18$  beats/min might arguably determine the presence of CAD. After we used ROC analysis in Matlab software (Version 12.5.0, Ostend, Belgium) to determine the best HRR1 value, we obtained a value of  $\leq 21$  beats/min as the best specificity and sensitivity point for predicting CAD. The main aim of the study was to investigate an HRR1 value of  $\leq 21$  beats/min for determining the presence of CAD but not heart rate reserve. We mentioned heart rate reserve as an exercise testing parameter in the manuscript. We calculated heart rate reserve as  $220 - \text{age in years} - \text{resting heart rate in beats/min}$ . If we had defined heart rate reserve in the methods, it would have made a better manuscript.

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**Available Online Date:** 25.12.2014

## The first experiences with the lotus valve system in Turkey as an alternative valve system in TAVI

To the Editor,

Transcatheter aortic valve implantation (TAVI) is an alternative therapy to surgical aortic valve replacement (AVR) in inoperable patients with severe aortic stenosis (AS). Currently, new valve systems are being developed, and we experienced TAVI with the Boston Scientific Lotus Valve

System (Marlborough, Massachusetts, USA) with two patients for the first cases in Asia-Pacific countries and Turkey. The first patient was a 77-year-old woman with severe AS with an echocardiographic aortic valve area of  $0.8 \text{ cm}^2$  and a mean aortic pressure gradient of 52 mm Hg, and her left ventricular function (LVEF) was 35%. Her logistic EuroSCORE was 38%, and she had New York Heart Association (NYHA) functional class III dyspnea. The other patient was a 82-year-old woman with severe AS; in her echocardiographic examination, the aortic valve area was  $0.6 \text{ cm}^2$ , and the mean aortic pressure gradient was 62 mm Hg, with an LVEF of 52%. Her logistic EuroSCORE was 29%, and she had NYHA class III dyspnea. The Lotus valve system has some advantages, such as it does not require rapid pacing during valve system implantation and balloon pre-dilatation, and it has a specific pre-shaped guidewire that has two types varying the length and curve, designed according to the size of the left ventricular cavity diameter. This valve system supports an ability to change positions while opening the valve system at the aortic valve level. Likely, if the chosen aortic valve size and aortic roof size do not match, the valve system could be taken back through the sheath. The other important feature of the Lotus valve is success in the prevention of paravalvular leak (PVL), which is related with increased mortality rate (1). In the REPRİSE I trial, in which the safety and efficacy of the Lotus valve were studied, one patient had stroke, PVL was seen in 3 of 11 patients, and permanent pacemaker implantation was required due to complete heart block, left bundle branch block, or atrial fibrillation with slow ventricular rate in 4 of 11 patients, while the requirement of permanent pacemaker implantation varies between 3% and 40% with other valve systems (1, 2).

In our patients, the follow-up echocardiography showed a well-functioning prosthesis, with a mean gradient of 7 mm Hg and 9 mm Hg, respectively. There was no paravalvular leak or pacemaker implantation required in either patient. The patients were clinically stable at 30 days of follow-up after the procedure. In summary, the ability to change valve position to obtain optimal implantation placement and the decrease in PVL rate are the most important reasons for using the Lotus valve system.

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**Available Online Date:** 25.12.2014

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DOI:10.5152/akd.2014.5784

