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# Author`s Reply

## To the Editor,

We thank the author(s) for their constructive comments on our study entitled "Heart rate recovery, cardiac rehabilitation, and erectile dysfunction in males with ischemic heart disease" published in the Anatolian Journal of Cardiology 2016; 16: 256-63 (1). In our study, we aimed to assess the relationship between heart rate recovery and the severity of erectile dysfunction (ED) in patients with ischemic heart disease and ED who have undergone cardiac rehabilitation. In addition, we assessed the impact of pharmacotherapy on the severity of ED among others. We are glad to learn that pharmacotherapy of ED and concomitant diseases are interesting because this can improve the overall quality of life in patients with many coexisting disorders.

Indeed nebivolol has unique properties when compared with previous generation beta-blockers. Nebivolol is approximately 3.5 times more cardio selective than bisoprolol, which reduces the risk of side effects typical for other beta-blockers (2). Another advantage of nebivolol is its vasodilator effect due to the increase of endogenous nitric oxide release by the endothelial cells, which leads us to hypothesize about the potentially antiatherogenic effect of this drug and creates the premise that nebivolol could also be beneficial in patients with ischemic heart disease (3); however, at present, it is not approved for the treatment of ischemic heart disease without coexisting arterial hypertension or heart failure (4).

In the erection mechanisms, endothelium-dependent relaxation of the penile arteries is crucial because rapid increase of their capacity up to 80% allows for bringing sufficient volume of blood to initiate the corporal veno-occlusive mechanism and maintain erection (5). The unique effect of nebivolol on the endothelium improves vessel relaxation, and in contrast to other beta-adrenergic blocking agents, nebivolol does not impair sexual function. In males with hypertension and coronary artery disease invasively treated, nebivolol had a protective effect on sexual function (2, 6).

In our study, beta-blockers were taken by 84 (94.38%) patients. Their use had no significant influence on the initial IIEF-5 (EQ1) score, as well as their change ( $\Delta$  EQ) caused by cardiac training (1). We agree that the comparison of nebivolol with other beta-blockers could bring additional information, but the small percentage of patients on nebivolol vs. bisoprolol, metoprolol, and carvedilol would not guarantee reliable results. At the time of the study, patients used to choose other drugs because of economic reasons. This situation has changed as the introduction of generics improved the availability of nebivolol for more male patients than before and allowed them to benefit from the unique properties of this drug in terms of sexual function.

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# Is thyroid function associated with masked hypertension?

#### To the Editor,

Masked hypertension (MHT) was first defined by Pickering in 1992, and its importance is progressively increasing (1). MHT is a condition wherein blood pressure measured according to hypertension guidelines in office is normal, whereas the mean 24-h ambulatory blood pressure measurement or blood pressure measurement out of office is high (2). Studies relating to the etiology of MHT is limited, and possible etiological factors include work stress, smoking, alcohol use, male sex, and excessive physical activity (3, 4). The association between MHT and thyroid hormone, which has major effects on the cardiovascular system, is not known. This study aims to investigate the association between thyroid hormone and blood pressure in newly diagnosed MHT patients.

In total, 712 patients without a previous diagnosis of hypertension and who were admitted to the outpatient clinic with hypertensive symptoms were enrolled. Patients were categorized into three groups, MHT, primary hypertension (PHT), and normotensive, according to the blood pressures measured at home and at the hospital. The mean systolic blood pressure (SBP) measured in office was <140 mm Hg and the mean diastolic blood pressure (DBP) was <90 mm Hg, whereas the mean measurements made at home were >135 mm Hg and >85 mm Hg, respectively; with these values, a diagnosis of MHT was made (2). Thyroid stimulating hormone (TSH), free-thyroxine (fT4), and free-triiodothyronine (fT3) levels were evaluated in 73 MHT, 73 PHT, and 74 normotensive participants using electrochemiluminescence immunoassay. The measurement device (Omron-M3, Omron-Healthcare Co. Ltd., Tokyo, Japan) was given to the participants, and they were instructed to measure blood pressure for 7 days, twice a day (2).

Of the 712 participants included in study, PHT in 206 patients, MHT in 73 patients, and normotension in 433 patients were determined. The mean SBP and DBP of patients with MHT and PHT were similar, whereas the mean SBP and DBP of the normotensive group were lower than those of the hypertensive groups. The mean log (TSH) level was higher, whereas the mean fT4 level was lower in the PHT group as compared with the MHT and normotensive groups. Log (TSH) and fT4 levels were similar in the MHT and normotensive groups. The proportion of patients with hypothyroidism was higher in the PHT group as compared with the other groups (PHT: 17.8% vs MHT: 1.4% vs normotensive: 5.4%). Stepwise multiple regression analysis showed that mean SBP and DBP are associated with log (TSH), fT4, and presence of hypothyroidism in the PHT group. Such associations were not found in MHT and normotensive groups.

No associations were determined between patients with MHT and thyroid hormone. The finding that there was no association between MHT and thyroid hormone can be interpreted in two ways. First, the risk factors effective in the pathophysiology of MHT increase blood pressure independent of the levels of thyroid hormone. This hypothesis is supported by the fact that blood pressure increases during work stress and related etiological factors and is regulated during rest in patients with MHT (4). Second, thyroid hormone dysfunction may not cause MHT pattern (out of office) of high blood pressure and instead may lead to persistent hypertension pattern of high blood pressure.

This abstract was presented as a poster presentation in the European Society of Endocrinology Congress (Dublin, 2015).

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