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Letters

TO THE EDITOR

Sympathetic and Autonomic Effects of Renal Denervation on Atrial Remodeling and Atrial Arrhythmias

I read the article by Schirmer et al. (1) with great interest and congratulate the authors on their excellent work. As the authors correctly state, there is a direct, partly blood pressure (BP)-independent effect of renal denervation (RDN) on cardiac remodeling and the occurrence of premature atrial contractions. However, I would like to call attention to a point that needs further clarification. RDN reduces renal efferent and possibly also afferent sympathetic activity, but compared with beta-blocker treatment, RDN results in an even more pronounced attenuation of atrial effective refractory period (AERP) shortening during negative thoracic pressure in obstructive sleep apnea (2). Moreover, both increased sympathetic and vagal nerve discharges before the onset of atrial arrhythmias (3) and increased sympathetic and parasympathetic nerve growth in the left atrium are present in pacing-induced congestive heart failure (4). Both acute autonomic activation-induced electrophysiological changes and autonomic remodeling-associated atrial structural changes may play a significant role in the creation of atrial ectopies and atrial fibrillation substrates (4). RDN reduces atrial sympathetic nerve sprouting, structural alterations, and AF complexity in persistent AF, independent of changes in blood pressure (5), but RDN might reduce arrhythmogenic atrial autonomic signaling and structural alterations (4). Modulation of the autonomic system by RDN already shows promising results (2). I would like to point out that the authors reported no relationships with modulation of the autonomic system.

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Please note: The author has reported that he has no relationships relevant to the contents of this paper to disclose.

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REPLY: Sympathetic and Autonomic Effects of Renal Denervation on Atrial Remodeling and Atrial Arrhythmias

We thank Dr. Patanè for his interest and his valuable comments on our paper (1). He argues that, despite the attenuation of atrial arrhythmogenic structural remodeling after renal denervation (RDN) shown in our paper (1), sympathetic modulation by RDN may also affect atrial electrophysiology and arrhythmogenesis. This point is interesting but, unfortunately, beyond the scope of our paper. A combined sympatho-vagal activation rather than sympathetic or parasympathetic activation alone plays a relevant role for the initiation and progression of atrial fibrillation (AF). RDN affects nerves along the renal arteries, thus reducing efferent and afferent sympathetic nerve activity. However, the parasympathetic nervous system is not directly modulated by RDN because parasympathetic nerves do not run along the renal arteries. Recently, we showed that RDN reduces atrial sympathetic but not vagal nerve sprouting, which resulted in lower complexity of AF in goats (2). Dr. Patanè mentions our recent observations that RDN can suppress vagally mediated shortening in