Ganglionated plexi interactions with atrio-ventricular node and right vagus nerve

To the Editor,

We read with great interest the article titled "Vagal denervation in atrial fibrillation ablation: A comprehensive review" by Aksu et al. (1) published in Anatol J Cardiol 2017; 18: 142-8. It is an excellent narrative review about ganglionated plexi (GP), vagal denervation, and atrial fibrillation. We have some commentaries about GP interactions with atrio-ventricular node (AVN) and right vagus nerve.

There is a large interactive network among different GP; this network serves as an "integrated center" of the cardiac autonomic innervation (2). Vagus nerve exerts its influence on the AVN through the epicardial fat pads that are primarily located on the posterior wall of the left atrium (2). The inferior vena cava-left atrium fat pad (namely also right inferior GP) located around the coronary sinus mainly provides vagal innervations and selectively innervates the AVN in humans (2, 3). It was shown that high-frequency stimulation of the right anterior (or superior right atrial vagal GP) and left superior GP (or superior left atrial vagal GP) could also influence the AVN (3). In addition, the influence of the right anterior GP on the AVN appears to be more important than its influence on the left superior GP (2, 3).

A functional neural pathway between the right vagus nerve and the AVN was identified (2), and the integrity of the GP seems to represent a mandatory interconnected network (3). In this study by Xhaet et al. (3), the absence of any alteration in the ventricular rate in response to high-frequency stimulation of the right vagus nerve after the ablation of GP suggests that the right vagus nerve is not directly connected to the AVN and that the integrity of the GP is required to produce vagal effects on the AVN (3). Probably, there is no direct pathway between both the right and left vagus nerves and the AVN.

The long-term influence of GP ablation on the electrophysiology of the AVN is not known. However, the incomplete GP ablation can increase the vulnerability of the atria to atrial fibrillation and denervation is likely transient (4, 5). In addition, GP ablation that led to parasympathetic denervation of the AVN could play a role in the high ventricular rate response of atrial tachycardia after atrial fibrillation ablation. Therefore, GP interaction with the AVN and right vagus nerve could provide new insights on that particular mechanism.

Figure 1 is very interesting from the anatomical point of view, but there are two GC without any GB. The alignment of the GP nomenclature is also obviously required.

In conclusion, there are GP interactions with the AVN and right vagus nerve with possible important consequences on vagal denervation in atrial fibrillation ablation. However, the role and influence of the GP on the complicated vagal innervation of the heart still needs to be clarified.

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DOI:10.14744/AnatolJCardiol.2017.8087