Author`s Reply

To the Editor,

We would like to thank the authors for their scientific comments related to our recently published article (1) entitled "Vagal denervation in atrial fibrillation ablation: A comprehensive review." published in Anatol J Cardiol 2017; 18:142-8. As mentioned by the authors, the inferior vena cava-left atrium fat pad (namely also ganglion C) located around the coronary sinus mainly provides vagal innervations and selectively innervates the atrio-ventricular node in humans (2, 3). Furthermore, this ganglion contains much more neurons than other ganglia (4). On the basis of this anatomical background, we only targeted ganglion C and performed the procedure using selective right atrial approach in patients with functional atrio-ventricular block (5, 6). Our selective right atrial approach was successful in six of seven patients. Considering the clinical features of failed case, this patient was the oldest patient in the study population. Therefore, we speculated that fibrosis of the conduction system due to advanced age may be the reason of unsuccessful ablation.

In their study, Xhaet et al. (3) tried to reveal the importance of fat pads in the vagal control of the atrio-ventricular node. The study demonstrated that parasympathetic innervations of atrio-ventricular node were mainly provided by the integrity of the vagal ganglia but not directly by the right vagus nerve. This study may be a starting point for well-designed studies to clarify selective or integrative innervations principles of cardiac parasympathetic system.

We thoroughly agree with the authors' comments that there are some difficult questions that should be answered:

(1) Which is the best method to define the exact location of vagal ganglia?

(2) How can we achieve complete and permanent ablation?

(3) Is there any importance of long-term effect of unrequited sympathetic activity after denervation?

Future studies are needed to clarify all these dilemmas in patients with atrial fibrillation.

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Ibuprofen-induced Kounis syndrome with diffuse ST segment depression and atrial fibrillation

To the Editor,

Kounis syndrome is defined as acute coronary events associated with allergy, anaphylaxis, or anaphylactoid reactions (1). In this syndrome, many inflammatory mediators, such as histamine, chymase, tryptase, thromboxane, prostaglandins, leukotrienes and its derivatives, as well as different cytokines and chemokines play a major role together with the activation of mast cells, lymphocytes, macrophages, and eosinophils (1, 2). Many factors, such as foods, different drugs, environmental exposures, and coronary stents, may trigger allergic reactions (1, 2). We presented a case of ibuprofen-induced Kounis syndrome with diffuse ST segment depression with ST segment elevation in aVR lead and atrial fibrillation.

A 57-year-old man presented with complaints of nausea, vomiting, itching, dyspnea, and retrosternal chest pain after taking ibuprofen+pseudoephedrine combination cold medication. He had no known systemic disease or prior drug use. He had taken two tablets of the same drug 15 days ago. Physical examination findings were as follows: cold, sweaty , blood pressure of 80/50 mm Hg, and pulse rate of 120 bpm. Isotonic sodium chloride infusion, dexamethasone, and pheniramine were administered, and subsequently, blood pressure increased. In the first 10 min of the ongoing chest pain, electrocardiography was performed, which showed diffuse ST depression with ST elevation in aVR lead. Cardiac troponin-I levels were 19.5 (normal range, 0.0–0.1)