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EDITORIAL COMMENT

Cardioneuroablation: A clinically useful vagal ablation

Cardioneuroablação: uma ablação vagal clinicamente útil?

Jose Carlos Pachon-M[a,b,c,*], Enrique I. Pachon-M[a,b,c], Carlos Thiene C. Pachon[b]

[a] *Sao Paulo University, Brazil*
[b] *Sao Paulo Heart Hospital, Brazil*
[c] *Sao Paulo Dante Pazzanese Cardiology Institute, Brazil*

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We congratulate Neto et al., whose case report on treating a marathonist by cardiac vagal denervation is published in this issue of the *Journal*,[1] for their commitment to treating the patient with maximum rationality and for the excellent final result obtained with cardioneuroablation (CNA).

CNA is a radiofrequency catheter ablation technique applied in the atrial endocardium with the aim of attenuating the vagal response. It was first developed in the 1990s, and the first series was published in 2005.[2] In the first study, highly positive results were observed in cardioinhibitory neurocardiogenic syncope, functional atrioventricular block (AVB), sinus node dysfunction and atrial fibrillation.

Indications for CNA are functional bradyarrhythmias due to vagal hypertonia with symptoms refractory to medical treatment without significant heart disease and with good response to atropine.[2]

Invasive electrophysiological study is highly recommended before CNA to exclude organic disease of the conduction system. It is essential in AVB to rule out His-Purkinje lesions, together with ajmaline testing for detecting difficult subclinical His-Purkinje lesions.

An atropine test is highly desirable before CNA to assess the degree of vagal tone in sinus bradycardia and AVB.[2] Atropine may have a long residual effect, so it can be used no less than 48 hours before CNA. It is performed by infusion of 0.04 mg/kg of intravenous atropine up to 2 mg. A normal response is characterized by a $\geq$50% increase in sinus rate and $\geq$30% increase in Wenckebach's point ending with $\geq$120 bpm. An inappropriate response suggests intrinsic disease of the conduction system. Atropine or any other vagolytic drug is contraindicated at the beginning of CNA.

Extracardiac vagal stimulation (ECVS) aims to study the vagal response through endovenous stimulation of the vagus nerve without direct contact at a frequency of 30 Hz for 50 μs at 1 V/kg up to 70 V.[3] It is of considerable value in CNA and in electrophysiology to study vagal activity in arrhythmias and anomalous bundles.

Several techniques of vagal denervation have been tried,[4] however, regardless of the technique, the most important goal is to eliminate the vagal effect at the end of the CNA.[3] This can be easily verified during the procedure through repeated ECVS. At the beginning of CNA, ECVS is performed, recording the baseline vagal response of the sinus and AV nodes. Vagal activity in the sinus node causes immediate sinus arrest, while in the AV node it causes high-grade AV block with ECVS during atrial pacing.[3] CNA should be ended after elimination of vagal effects on the sinus and AV nodes. In this study, an episode of Wenckebach-type AVB occurred in long-term follow-up, suggesting incomplete AV node denervation. However, this assessment is hampered as, unfortunately, EVCS was not performed to confirm the efficacy of the CNA in this case.

Ganglionated plexuses cannot not visualized but can be accessed anatomically based on spectral maps from initial studies. However, when anatomical CNA does not eliminate

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* Corresponding author.
E-mail address: pachon@usp.br (J.C. Pachon-M).

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vagal activity, denervation should be expanded by ablating AF nests.[5] For this purpose, we developed fractionation mapping in 2005 to label AF nests in the electroanatomical model.

Conflicts of interest

The authors have no conflicts of interest to declare.

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