REFERENCES

1. Doménech M, Berruezo A, Molina I, Mont L, Coca A. La presión arterial ambulatoria nocturna se asocia al remodelado auricular y la activación neurohormonal en pacientes con fibrilación auricular idiopática. Rev Esp Cardiol. 2013;66:458–63.
2. Dominguez-Rodriguez A, Abreu-Gonzalez P, Kaski JC. Disruption of normal circadian rhythms and cardiovascular events. Heart Metab. 2009;44:11–5.
3. Domínguez-Rodríguez A, Abreu-González P, Reiter RJ. Melatonina y enfermedad cardiovascular: ¿mito o realidad? Rev Esp Cardiol. 2012;65:215–8.
4. Enjuanes-Grau C, Domínguez-Rodríguez A, Abreu-González P, Jiménez-Sosa A, Avanzas P. Niveles de presión arterial y el patrón de secreción de la melatonina en una población de médicos internos residentes de guardia. Rev Esp Cardiol. 2012;65:576–7.
5. Baltatu O, Lippoldt A, Hansson A, Ganten D, Bader M. Local renin-angiotensin system in the pineal gland. Brain Res Mol Brain Res. 1998;54:237–42.
6. Baltatu O, Afeche SC, José dos Santos SH, Campos LA, Barbosa R, Michelini LC, et al. Locally synthesized angiotensin modulates pineal melatonin generation. J Neurochem. 2002;80:328–34.
7. Campos LA, Cipolla-Neto J, Amaral FG, Michelini LC, Bader M, Baltatu OC. The Angiotensin-melatonin axis. Int J Hypertens. 2013;2013:521783. http://dx.doi.org/10.1155/2013/521783.
8. Tengattini S, Reiter RJ, Tan DX, Terron MP, Rodella LF, Rezzani R. Cardiovascular diseases: protective effects of melatonin. J Pineal Res. 2008;44:16–25.
9. Jonas M, Garfinkel D, Zisapel N, Laudon M, Grossman E. Impaired nocturnal melatonin secretion in non-dipper hypertensive patients. Blood Press. 2003;12:19–24.

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Relationship Between Nighttime Blood Pressure, the Renin-angiotensin System and Melatonin. Response

Relación entre la presión arterial nocturna, el sistema renina-angiotensina y la melatonina. Respuesta

To the Editor,

We appreciate the interest of Drs. Dominguez-Rodriguez and Abreu-Gonzalez in our article, as well as their comments, given their extensive experience in the role of melatonin in arterial blood pressure.

Although numerous epidemiological studies have demonstrated that the arterial blood pressure is the most important risk factor for atrial fibrillation,[1,2] the underlying pathophysiological mechanisms are still unknown. As we indicate in our article, it has been suggested that, among other effects, the activation of the renin-angiotensin system induces changes in cardiac structure, such as left ventricular hypertrophy and/or increase in left atrial size, that act as an ideal substrate for the neurohormonal activation implicated in atrial fibrillation.[3–5]

The objective of our study was to evaluate the hypothesis that the arterial blood pressure values obtained by means of 24-h blood pressure monitoring are associated with structural changes in left atrium and with the neurohormonal markers involved in the development of idiopathic atrial fibrillation (IAF). Thus, said results should be encompassed in the context in which the study was carried out, in the attempt to search for new pathophysiological mechanisms that help to shed light on as yet unidentified causes of IAF.

In this respect, the contributions of Dominguez-Rodriguez and Abreu-Gonzalez are highly interesting, as is the recent review by Campos et al.,[6] which summarizes the accumulated evidence on the important role of angiotensin II and melatonin in the modulation of circadian rhythm, and their implication in cardiovascular disease. Undoubtedly, and in view of the results of our study, the analysis of the possible interaction of the renin-angiotensin system and the melatonin concentration with nocturnal arterial blood pressure values in individuals with IAF could contribute to the elucidation of another new pathophysiological mechanism implicated in the development of IAF. Unfortunately, in the design of the pilot study and the subsequent case-control study,[7] we did not consider analyzing the melatonin concentration. Thus, with the present data, we are unable to respond to this unquestionably new and interesting pathophysiological hypothesis concerning the genesis of IAF.

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Available online 12 August 2013

REFERENCES

1. Nabauer M, Gerth A, Limbourg T, Schneider S, Oeff M, Kirchhof P, et al. The registry of the German competence NETwork on atrial fibrillation: patient characteristics and initial management. Europace. 2009;11:423–34.
2. Le Heuzey JY, Breithardt G, Camm J, Crijns H, Dorian P, Kowey PR, et al. The RecordAF study: design, baseline data, and profile of patients according to chosen treatment strategy for atrial fibrillation. Am J Cardiol. 2010;105:687–93.
3. Maisel AS, Koon J, Krishnaswamy P, Kazenegra R, Clopton P, Gardeto N, et al. Utility of B-natriuretic peptide as a rapid, point-of-care test for screening patients undergoing echocardiography to determine left ventricular dysfunction. Am Heart J. 2001;141:367–74.
4. Jourdain P, Bellorini M, Funck, Fulla Y, Guillard N, Loiret J, et al. Short-term effects of sinus rhythm restoration in patients with lone atrial fibrillation: a hormonal study. Eur J Heart Fail. 2002;4:263–7.
5. Dixen U, Ravn L, Soeby-Rasmussen C, Paulsen AW, Parner J, Frandsen E, et al. Raised plasma aldosterone and natriuretic peptides in atrial fibrillation. Cardiology. 2007;108:35–9.
6. Campos L, Cipolla-Neto J, Amaral F, Michelini L, Bader M, Baltatu C. The angiotensin-melatonin axis. Int J Hypertens. 2013;2013:521–783.
7. Domenech M, Calvo N, Ramos P, Berruezo A, Camafort M, Coca A, et al. Nocturnal hypertension as one of posible physiopathological mechanism of lone AF. J Hypertens. 2013;31 Suppl A:e32.

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