Letters to the Editor

Comment on the Management of Resistant Hypertension in a Multidisciplinary Unit of Renal Denervation: Protocol and Results

Comentario al manejo de la hipertensión resistente en una unidad multidisciplinaria de denervación renal: protocolo y resultados

To the Editor,

I have read with great interest the article entitled “Management of Resistant Hypertension in a Multidisciplinary Unit of Renal Denervation: Protocol and Results” (“Manejo de la hipertensión resistente en una unidad multidisciplinaria de denervación renal: protocolo y resultados”), in which the authors report an improvement in arterial blood pressure similar to that observed in previous studies, as well as a more marked reduction in the use of antihypertensive drugs in patients who undergo renal denervation performed within a multidisciplinary program. The reported findings are highly interesting; however, I feel that certain observations could be clinically relevant.

First, the authors consider pseudoresistant hypertension (HT) to be present in patients with mean arterial blood pressure values of less than 140/90 mmHg coinciding with a period of activity occurring during ambulatory blood pressure monitoring (ABPM). However, in the current recommendations, HT is defined as arterial blood pressure values greater than 130-135/85 mmHg in an ABPM recording during the period of activity. Thus, HT in which the patients have a mean arterial blood pressure in ABPM greater than 130-135/85 mmHg cannot be considered pseudoresistant. This bias in the inclusion of patients with resistant HT may have affected the observed findings.

Secondly, it is noteworthy that, despite the definition of resistant HT as the condition in which the arterial blood pressure values exceed 140/90 mmHg even with the intake of 3 or more drugs, including a diuretic, 10% of the patients in the published report who underwent the procedure were not being treated with diuretics, and the percentage of subjects receiving diuretics after renal denervation is not disclosed. The pharmacological optimization of these patients in later visits may have altered the reported findings.

Finally, the authors administer aldosterone antagonists to counteract possible secondary hyperaldosteronism. However, my attention is drawn to the absence of staging of other secondary forms of HT, especially when it is known that 27% of the population that undergoes the procedure is diagnosed as having obstructive sleep apnea syndrome. In fact, as the authors point out, there could be a placebo effect in the response to the denervation, an occurrence that would not only be related to greater adherence to the treatment or to the low-sodium diet, but to an improvement in the obstructive sleep apnea syndrome with better dietary adherence. Likewise, drug-induced HT was not tested in the study population, a circumstance that could also influence the results obtained.

In agreement with the authors and the Symplicity HTN-2 trial, renal denervation results in a decrease in the arterial blood pressure and a reduction in the drug therapy. However, an exhaustive search for secondary forms of HT and an optimization of drug therapy could avoid the need for the renal denervation procedure which, although it has been shown to be feasible and safe, is not free of complications.

Juan C. Bonaque*

Servicio de Cardiología, Hospital Torrecárdenas, Almería, Spain

*Corresponding author:
E-mail address: jc_bonaque@hotmail.es

Available online 24 July 2013

REFERENCES


SEE RELATED ARTICLES:
http://dx.doi.org/10.1016/j.rec.2013.06.003
http://dx.doi.org/10.1016/j.rec.2012.09.006
http://dx.doi.org/10.1016/j.rec.2013.05.014
Relationship Between Nighttime Blood Pressure, the Renin-angiotensin System, and Melatonin

Relación entre la presión arterial nocturna, el sistema renina-angiotensinno y la melatonina

To the Editor,

We have read with great interest the article on nighttime blood pressure (BP) and neurohumoral activation in patients with idiopathic atrial fibrillation in the Revista Española de Cardiología.1 According to the authors, nighttime BP values are directly associated with left atrial size and atrial and brain natriuretic peptides in patients with idiopathic atrial fibrillation. We think it may be of interest to discuss a number of issues related to nighttime BP and neurohumoral activation.

First, the authors do not mention the effect of another neurohormone, melatonin, on BP. Oscillations in physiological functions that occur over a 24-h period are known as circadian rhythms.2 During sleep, there is a decrease in BP in the cardiovascular system. Melatonin is one of the main hormones serving as an endocrine signal in the circadian rhythm.3 Its secretion is mainly controlled by light via the suprachiasmatic nucleus (biological clock), such that darkness stimulates its secretion and light inhibits it.6 Recently, our group demonstrated an association between an abnormal pattern of melatonin secretion and alterations in BP in healthy subjects.4 Second, the authors discuss from a physiological point of view the important role of nighttime BP in remodeling and growth of the left atrium, possibly mediated by activation of the renin-angiotensin system (RAS).1 Several articles have been published on the association between the RAS and melatonin.5,7 Angiotensinogen is the precursor of the RAS and has been identified in pineal glial cells and the receptors type AT1b in pinealocytes.5 Angiotensin II, as part of the RAS, acts on receptors type AT1b in pinealocytes to influence the synthesis and activity of tryptophan hydroxylase, an enzyme that limits melatonin production.7 The demonstration of a functional pineal RAS interfering with melatonin synthesis indicates that this may affect the modulation of circadian rhythms. In fact, the majority of published studies suggest that the relationship between angiotensin and melatonin synthesis in cardiovascular disease is antagonistic.7

Finally, the administration of low pharmacologic doses of melatonin (1 mg) reduces BP as a consequence of various mechanisms, such as a direct hypothalamic effect, a lowering of catecholamine levels, the relaxation of the smooth muscle wall and, above all, as a result of its antioxidant properties. There is evidence suggesting that melatonin may have a hypotensive effect,7 especially in non-dipper hypertensive patients.9 Thus, the interaction between the RAS and melatonin in relation to BP should be taken into account. From a clinical standpoint, more research is needed on the interaction between angiotensin and melatonin to further our understanding of the pathophysiology of cardiovascular disease, with a possible impact on chronotherapeutic strategies.

Alberto Domínguez-Rodríguezb,c,* and Pedro Abreu-Gonzálezc,d

*Servicio de Cardiología, Hospital Universitario de Canarias, Sta. Cruz de Tenerife, Spain
bFacultad de Ciencias de la Salud, Universidad Europea de Canarias, Sta. Cruz de Tenerife, Spain
cInstituto Universitario de Tecnologías Biomédicas, Sta. Cruz de Tenerife, Spain
dDepartamento de Fisiología, Universidad de La Laguna, Sta. Cruz de Tenerife, Spain

*Corresponding author:
E-mail address: adrvdg@hotmail.com (A. Domínguez-Rodríguez).

Available online 30 July 2013