

Ivabradine as an Atrioventricular Node Modulator. Promise or Reality? Response



Ivabradina como modulador del nódulo auriculoventricular. ¿Promesa o realidad? Respuesta

To the Editor,

First, we would like to thank Dr Álvarez-Acosta et al. for their comments, which we will try to address here.

In accordance with the relevant guidelines,¹ we routinely implant cardiac resynchronization devices to treat heart failure in optimally treated and nondecompensated patients. Our patient² was stable at the time of implantation and his heart rate, although controlled, was insufficient to guarantee an adequate pacing percentage. Nevertheless, simply implanting a resynchronization device in a patient with heart failure rarely confers a clinical improvement in subsequent weeks if the biventricular pacing percentage is only about 70%. It would be as incredible as a drug left untouched by a patient in a bedside drawer exerting a clinically relevant effect. Because we can rule out an “inherent improvement” from a resynchronization device unable to achieve adequate pacing and there were no changes in any other treatment between the 2 consecutive revisions, we must delve into the eventual role of ivabradine in our patient’s heart rate control.

The criteria of causation include temporality, biological plausibility (there is a high-density If current in the atrioventricular node), analogy (ivabradine reduces heart rate during atrial fibrillation in animals), and experiment (ivabradine decreased heart rate in atrial fibrillation vs placebo in a human trial). If the pacing percentage were to decrease after ivabradine withdrawal, our hypothesis would be strengthened but such an approach would be ethically questionable. The possible effects of ivabradine on heart rate are in no way ruled out by the publications on ivabradine, which make no mention of this mechanism of action. However, it is not necessary to turn to rare genetic mutations to explain the inhibitory effect of

ivabradine on node conduction because the United States prescribing information for this drug states that first-degree atrioventricular block is a frequent adverse reaction.

We would also like to take this opportunity to report that the same effect on percentage of pacing was seen in another patient administered ivabradine in the same clinical setting.

Promises can become reality if we are proactive in the search for therapeutic options by not only researching new molecules, but also by exploring new indications for existing ones.

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Implantation of Ventricular Assist Devices in Hypertrophic Cardiomyopathy. Is It a Safe Option?



Implante de dispositivo de asistencia ventricular en miocardiopatía hipertrófica. ¿Es una opción segura?

To the Editor,

We read with great interest the article published in *Revista Española de Cardiología* by Varela-Falcón et al.¹ about their experience of a left ventricular assist device (LVAD) in a patient with hypertrophic cardiomyopathy. We would like to raise a number of points for consideration in relation to this article.

First, we would like to congratulate the authors for the good outcome in this case, given the challenge it presented. In the last decade, LVADs have become a standard treatment option for improving survival and quality of life in patients with dilated cardiomyopathy and advanced heart failure, whether as a bridge to transplant or as a destination therapy²; however, there is little experience of this therapy in patients with cardiomyopathy and restrictive physiology, and it is not without complications.³

The article described a patient with hypertrophic obstructive cardiomyopathy in an advanced stage of heart failure, but did not provide details on the patient’s left ventricular function before implantation. Nor did it explain why surgical myectomy was not performed, given the high dynamic left ventricular outflow tract gradient that was reported. An improvement in this gradient could have reduced the wedge pressures and improved the transpulmonary gradient.

One of the main complications during follow-up of patients with cardiomyopathy associated with apical hypertrabeculation are suction events and the increased incidence of thrombotic and embolic events. The authors state that in this case they decided not to perform surgical resection of the trabeculae due to the risk of incomplete resection. However, in our experience, incomplete resection of apical trabeculae increases the likelihood of suction events and thrombosis, particularly in hypertrabeculated ventricles, and careful examination of the ventricular cavity is recommended, putting the patient on bypass if necessary.⁴

We would also like to comment on the difficulty of inotropic treatment when initiating LVAD support and in the immediate postoperative period in these cases. In most centers, the usual