LETTERS TO THE EDITOR

Ventricular Pacing After Atrioventricular Node Ablation

To the Editor:

We have read with interest the article by Dr. Morina et al describing their initial experience with implantation of active fixation electrodes in the His bundle for permanent ventricular stimulation following ablation of the AV node. Their results are interesting as they establish stimulation threshold parameters and stable echocardiography measures after only a few months followup. Nevertheless, it must be noted that the failure rate with electrode implantation was considerable (33%) and the procedure was significantly long (approximately 3 hours). Their data are important because they open a potential treatment path for patients who require long-term ventricular stimulation with the aim of avoiding some of the serious complications that can result from stimulation of the RV apex.

The authors point out that stimulation of the His bundle could be the most appropriate technique in patients with an intact His-Purkinje system (normal HV, normal surface ECG). Nevertheless, we would like to point out that the theoretically ideal candidates for the implantation of this system must be patients with an intact His-Purkinje and structural cardiopathy (prior ventricular dilatation or mitral insufficiency), which are the subgroups identified as being at greatest risk for developing hemodynamic deterioration or worsening of mitral insufficiency after ablation of the AV node.

It is well known that stimulation from the apex of the right ventricle (RV) interferes with the closure of the mitral valve due to changes in the activation sequence of the mitral valve apparatus and the tension generated in the papillary muscles, or both. The function of the mitral valve depends on the integrity of the valve, the tendon cords, the mitral ring, the papillary muscles, and all these structures as a whole during ventricular systole in order to occlude the mitral orifice. The dimensions of the ventricles and the valve ring vary during the cardiac cycle, so that the position of the papillary muscles and their interaction with the mitral valve area can produce changes in regurgitation during the cardiac cycle. The anomalous activation of the papillary muscles during right ventricular stimulation will change the tension of the valve veins in a way that could cause a coaptation fault and an increase in the regurgitation orifice. In these patients, stimulation from the His bundle could be an excellent alternative in the hands of an expert, although this has not been proven in intermediate and long-term studies. In patients without structural heart disease and AV block at the supraventricular level, permanent stimulation of the His bundle is technically possible, as shown by Morina et al; nevertheless, this patient population is not the one that a priori appears would benefit most in terms of mortality and morbidity.

Biventricular stimulation could be an interesting option in patients with possible hemodynamic deterioration, although this would also be subject to various technical limitations.

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Response

To the Editor:

We would like to thank Anguera et al for their kind letter and the interest shown in our article. Basically, we agree with all their comments, and find that they do not differ substantially from the contents of our article; nevertheless, we would like to make several points:

1. The aim of our study was simply to demonstrate the possibility of selectively stimulating the His bundle permanently, and for this we selected a very specific patient population, at low risk for developing future disturbances of infrahisian conduction. We have already commented in the limitations section that this not yet at well-developed technique, and we noted that the long duration of the procedure and the high failure rate at present limits its general application until such time as electrodes, introducers, and above all specific fixation methods are developed. In any case, all new procedures in medicine are subject to a costly learning curve and should not be abandoned for this reason; the modern ablation techniques that were so costly in time and effort some years ago have since been perfected.

2. In a series of patients subjected to ablation of the AV node, later expanded upon by Anguera et al, Vanderheyden et al described (by retrospective analysis) cli-
nical deterioration with the appearance of symptoms of congestive cardiac insufficiency in some patients, and found an association with the existence of mitral insufficiency, ventricular dilatation, and previous ventricular malfunction. They attributed this deterioration, among other factors, to the aberrant activation of the left ventricle (LV) after stimulation of the right ventricle (RV) point; they proposed that the proximal stimulation of the His bundle could avoid the changes described.

3. The negative hemodynamic effects associated with the asynchronous activation of the LV, by blocking the left branch or stimulation of the RV apex, has been documented as occurring independent of the existence of structural disease. The clinical repercussions of these changes obviously depend on the prior state of ventricular function. In the patients described by Anguera et al., with data showing prior ventricular dysfunction in the compensated phase, the additional deterioration caused by asynchronous stimulation of the LV after ablation could trigger the development of a decompensated phase with progressive dilatation, an increase in mitral regurgitation, and congestive cardiac insufficiency.

4. We agree with Anguera that the selective stimulation of the His bundle would be more advantageous in the case of patients with structural pathology, in whom there is minimum deterioration of ventricular function associated with stimulation of the RD point, as could occur in the patients described in his article, and this would be more important as it could lead to clinical deterioration. Nevertheless, in this patient population there is a greater incidence and prevalence of changes in infrahisian conduction; for this reason, we believe that the procedure of choice is synchronous biventricular stimulation in this group of patients with an additional electrode in the coronary vein.

5. Today the importance of asymptomatic ventricular dysfunction is known and early treatment is recommended, for this reason, we believe that if a stimulation technique is available that permits activation of the ventricles via a normal conduction system, avoiding the dysfunction associated with asynchronous activation, we must use it even with patients with prior good function in order to avoid the possible long-term deleterious effects of asymptomatic ventricular dysfunction.

6. All these hypotheses need to be confirmed by prospective studies designed for this purpose to establish the true definitive indications for this technique.

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