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Letter to the Editor

CONTRADICTION OF WHETHER OR NOT ABLATING ASYMPTOMATIC PATIENTS WITH WOLFF-PARKINSON-WHITE SYNDROME

CONTRADICCIÓN DE ABLACIONAR O NO A PACIENTES ASINTOMÁTICOS CON SÍNDROME DE WOLFF-PARKINSON-WHITE

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To the Editor:

There is much debate on whether or not to perform ablation in asymptomatic patients with Wolff-Parkinson-White syndrome (WPW). With this letter we intend to continue the discussion and finally to reach an agreement. It is known that highly dangerous cardiac arrhythmias may occur in patients with accessory pathways. These can be either of syncopal character, group themselves together, represent the clinical presentation of accessory pathways and lead young people with allegedly healthy hearts to sudden death (SD)[1]. We are on the side of those who see the need for ablation of asymptomatic patients with WPW, and we will try to explain the reasons and also add that this therapeutic procedure would be performed to all patients with asymptomatic WPW after being subjected to electrophysiological study (EPS).

We agree with the Pappone's[2] group criteria and other agonists, as we should remember that he states the existence of a subgroup of patients who should be considered at high risk for arrhythmias; also, Barja[3] adds that the incidence of asymptomatic WPW is equal to the expected, following a screening of more than 200,000 electrocardiograms (ECG). In our personal experience, in a study[4] that aimed to assess atrial vulnerability in hyper and prehypertensive children, we found, after 450 electrocardiograms, a 10 year-old patient with WPW (and so far asymptomatic). It is known that children have a higher conduction speed in the normal conduction system, thus reducing the possibility of anterograde arrhythmias, (this child was excluded from our study). The result showed an incidence of 2.2 per 1,000 patients with WPW.

Barja[3] says the results and risks of a group can be extrapolated to groups with similar characteristics, and he refers this as he mentions Wellens' position, which states that the results and risks of a group, cannot be extended to another. Now let us focus on Pappone's[2] group, in which high-risk patients are defined and abla-

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tion of accessory pathways is suggested. Their monitoring has demonstrated the appearance of clinical arrhythmias and their absence in those with low risk. The positive predictive value was higher in high-risk compared with low-risk, taking into account the absolute refractory period of the accessory pathway. However, Dorantes and Méndez[1] mention that the inability to induce arrhythmias in patients with accessory pathway does not guarantee that it will not be inducible at another time, or that its stimulation necessarily implies its appearance in the clinic. Our Group[5] presented a 52-year-old patient whose first manifestation was atrial fibrillation by accessory pathway, which could have led him to ventricular fibrillation and sudden death[5].

Barja[3] mentions that the work of Chiale and Elizari's group state that a supernormal excitability and conduction segment of a sick accessory pathway could explain episodes of malignant arrhythmias in patients with long refractory periods of accessory pathways.

What will happen with asymptomatic patients with prolonged monitoring for years?, and what will happen if another EPS is performed to them at a time when the electrophysiological properties of accessory pathways demonstrate other times of refractory period?, or what if the conduction speed through the normal conduction system decreases?

It should be remembered that for an arrhythmia to develop with the participation of the accessory pathway itself in a patient with accessory pathway, the most important fact is the existence of an asymmetrically decreased conduction, which will not allow the propagation of the stimulus, which would happen with regional variations in excitability. The latter would be modulated by the extracellular concentration of calcium[6]. We would also ask, is it better to keep the isoprenaline use protocol or to use some drug during EPS to decrease conduction speed by the atrioventricular node? Perhaps the latter would facilitate that the difference between the times and conduction speeds of accessory pathway/ normal conduction system turn different and the accessory pathway might manifest itself.

Therefore, there is undoubtedly another important element that we must not forget: autonomic influence. In this case, we would like to mention the vagal predominance. The existence of ion channels as additional currents contributing to the action potential should be remembered, as in the case of K + current activated by acetylcholine (I_{KACh}), which is particularly important in the sinoatrial and atrioventricular nodes, and in the atrial muscle, where they may produce hyperpolarization.

An asymptomatic individual with an accessory pathway, that at any given time, for any reproducible or non reproducible clinical situation, shows a parasympathetic predominance, he/she will present: increased I_{KACh} activity, a decrease in atrial refractory period and slowing of conduction speed through atrioventricular node[7,8]. Regarding the presence of slow conduction through an alternative route, it is considered that such areas with this kind of physiologically determined conduction (atrioventricular node in the WPW), determine that the impulse moves slow enough to find the block area that is ready to be re-excited[9]. For this reason, we could argue that this autonomic situation, reproduced here, would undoubtedly facilitate blocking (slow conduction) in the atrioventricular node, and thus the antegrade conduction by accessory pathway could find the area of the re-excitable atrioventricular node, in an retrograde form. Furthermore, some authors[10] consider the explanation above, as a modulator factor of AF appearance. Therefore, pathophysiologically speaking, it would meet all the conditions for an AF to occur and that it might be anterogradely conducted by the accessory pathway. Dorantes and Méndez[1] state that in patients with accessory pathway there is evidence of a basic atrial disease with abnormally prolonged and fractionated endocardial atrial electrograms, and a significantly higher incidence of demonstrated episodes of AF.

For all of the above, it can be concluded that a patient with asymptomatic WPW, subjected to an EPS, and with no ionic and autosomic conditions "conducive to the accessory pathway"; will not be diagnosed as high risk at that time. But if the above said could ever happen (predominantly vagal autonomic situation, which decreases conduction speeds by the atrioventricular node and facilitates conduction through the accessory pathway), the same patient, at another point in time, could show a positive EPS or could clinically present an episode of arrhythmia. So, what is really the positive and negative predictive value of asymptomatic WPW patients, previously diagnosed as low risk? How long should we monitor them to say they will not present a clinical arrhythmia?

We will not answer these questions, because there are obviously many studies to design in order to answer them. However, if we consider what is explained here, from the physiological point of view, we can justify our adherence to the agonists' view, and our intention to perform ablation in all patients with asymptomatic WPW, to whom EPS is performed although classified as low risk. The latter arises when considering the low incidence of complications of radiofrequency ablation obtained in multicenter studies men-

tioned by Barja[3] (MERFS, NASPE, Atakr).

Experience has shown, in circumstances such as Brugada syndrome that EEF prediction does not always behave the same way. Changes in the internal environment and regulatory mechanisms cannot be predicted by tests that recreate environments which only approximate to reality. In our opinion, the combination of EEF as stratifier, and ablation in all cases, might be suitable, if we consider that although malignant arrhythmias do not have a high prevalence, the sum of the probability of suffering from these and from the so-call benign (orthodromic tachycardia) should increase the overall risk of arrhythmogenesis. In this way, we would be dealing with tachycardias that compromise the quality of life of patients.

This whole idea of ablation has been considered on the basis that EPS with or without ablation, by definition, is a very aggressive test for the patient, creates discomfort, and can lead to complications. If the catheters are already inside the patient's heart, and induction of arrhythmias may certainly not reflect what will happen in reality, why not consider ablation if the most invasive procedure has already been performed? If EPS risk stratification criteria finally prevails, why not perform ablation? What if accessory pathway recurrence occurs and the patient was classified as low risk, would it be wise to maintain regular monitoring through appointments with the doctor?

These questions simultaneously summarize and argue our position on the issue, but ultimately we cannot be sure who is right. Let this letter serve to continue the debate on this subject and to design new studies.

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