Elective Cardioversion of Atrial Fibrillation. Are Quinidine and Electrical Shock Really Equivalent?

To the Editor:

The two modalities of cardioversion for converting atrial fibrillation, chemical and electrical, have never been compared in a randomized study. Nonetheless, the experience acquired in clinical practice and the results of numerous studies have generated enough evidence for a consensus that electrical cardioversion is more effective than chemical cardioversion, especially when atrial fibrillation lasts more than one week.[1] It also seems clear that electrical cardioversion is very safe, in spite of requiring deep sedation, and rarely needs prolonged hospital monitoring. This cannot be said of chemical cardioversion. Antiarrhythmic drugs often produce cardiovascular side effects, particularly after the first doses, and these side effects can occur at any time. Although all antiarrhythmics can produce potentially mortal ventricular arrhythmias, the antiarrhythmics with class III effect do so most often. From 1% to 5% of treated patients develop polymorphic tachycardia. In the case of quinidine, this effect so is well known that it has already become part of the history of medicine.[2] The administration of a loading dose of quinidine to convert atrial fibrillation has such a pronounced proarrhythmic effect that it is advised against in recent guidelines for clinical practice on atrial fibrillation[3] (class IIb indication). Authors understand that we now have better alternatives. In a publication reporting the findings of a registry of 1152 consecutive patients treated with electrical or chemical cardioversion to convert atrial fibrillation, 6 cardiac deaths occurred among 570 patients under the age of 65 years. All the deaths were sudden and associated with the administration of quinidine in high doses (1000-2000 mg/day).[4]

Given this background, it is easy to understand our surprise when we read the article by Valencia Martín et al,[5] whose conclusions undermine the certainties of 50 years of accumulated evidence. The authors conclude, literally: «Both therapeutic modalities (electrical cardioversion and pharmacological cardioversion with quinidine) are valid and the decision to choose one or the other one will depend on the experience of the cardiologist.» This conclusion is supported by a non-randomized, retrospective study, to mention only one of its limitations. They detected a single episode of *torsades de pointes*, of unknown consequences for the patient, which meant that the incidence was 1.16%, which is lower than has been reported in the literature. It is possible that this finding could be explained by the fact that patients did not undergo continuous electrocardiographic control, which would seem to be mandatory, and that less symptomatic cases passed unnoticed. To our knowledge, the effect on the QT interval was not controlled either. The elevated effectiveness reported for quinidine is also notable, since it is much higher than would be expected in a population of patients with sustained arrhythmia for such a long time (mean 58 weeks). In contrast, the effectiveness of electrical cardioversion would have been greater if an alternative paddle configuration on the chest and/or a second 360-J shock had been used.

For these reasons, we think that the conclusion reached by the authors is venturesome considering that it contradicts numerous studies and clinical practice guidelines, and magnifies the role that quinidine now has in cardioversion for atrial fibrillation.

Luis Tercedor Sánchez
and Miguel Álvarez López

Unidad de Arritmias. Servicio de Cardiología.
Hospital Universitario Virgen de las Nieves.
Granada. España.

REFERENCES

1. Almendral J, Marín E, Medina O, Peinado R, Pérez L, Ruiz R, et al. Guías de práctica clínica de la Sociedad Española de Cardiología en arritmias cardíacas. Rev Esp Cardiol 2001;54:307-67.
2. Selzer A, Wray HW. Quinidine sycope: Paroxismal ventricular fibrillation occurring during treatment of chronic atrial arrhythmias. Circulation 1964;30:17-26.
3. Fuster V, Rydén LE, Asinger RW, Cannon DS, Crijns HJ, Frye RL, et al. ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee into Develop Guidelines for the management of Patients with Atrial Fibrillation). Circulation 2001;104:2118-50.
4. Carlsson J, Tebbe U, Rox J, Harmjanz D, Haerten K, Neuhaus K, et al. Cardioversion of atrial fibrillation in the ederly. Am J Cardiol 1996;78:1380-4.
5. Valencia J, Climent VE, Marín F, Vicente J, Martínez JG, García M, et al. Eficacia de la cardioversión programada en la fibrilación auricular. Comparación de dos esquemas de tratamiento: cardioversión eléctrica frente a farmacológica. Rev Esp Cardiol 2002;55:113-20.

Response

To the Editor:

We thank Drs. Luis Tercedor and Miguel Álvarez for the interest they showed in our article «Effectiveness of Scheduled Cardioversion in Atrial Fibrillation. Comparison of Two Schemes of Treatment: Electrical Cardioversion versus Pharmacological Cardioversion,» recently published in REVISTA ESPAÑOLA DE CARDIOLOGÍA.[1] However, we would like to clarify certain points and address some of the considerations in their commentaries.

In the first place, it should be noted that, as mentioned in

the article, the study was a comparative study of two consecutive series of patients who underwent cardioversion, pharmacological cardioversion at one hospital and electrical cardioversion at another. Therefore, it is a registry of cardioversions carried out at two hospitals in our community, with the limitations that this involves.

On the one hand, although the high success rate in the quinidine group, superior to that described in earlier studies,[2] may seem surprising, other series have reported similar success rates, in some case over 85%.[3,4] These results are from uncontrolled studies, but similar results have been found for amiodarone[5] and flecainide[6] in comparative studies. It should be emphasized that in our study the hospital where quinidine cardioversion was performed has extensive experience in the management of this drug. This type of cardioversion has been performed for 10 years with scant complications, as described in the article. This long experience may have been responsible for the good results. On the other hand, the complication rate found in the pharmacological group (1.16%), although low, is similar to the rate reported in some published series. It should be remembered that the meta-analysis of Southworth et al[7] encountered a mortality rate similar to that of other antiarrhythmic drugs considered «safer.» In our study, the patients treated with quinidine remained hospitalized throughout treatment. At this time (the first 72 hours) is when the highest incidence of arrhythmias is reported.[8] Only one case of symptomatic *torsade de pointes* was recorded, which was resolved without consequences for the patient. It is possible that the real frequency of arrhythmic complications is underestimated when patients are not being controlled electrocardiographically; however, it is logical to think that all the symptomatic complications were detected. It is possible that some patient had an asymptomatic and, therefore, undetected arrhythmic episode, but its clinical meaning is dubious at best. It has been reported that the proarrhythmic effects of quinidine, although idiosyncratic, are associated with depressed systolic function. The series that we presented consisted of patients with conserved systolic function. In any case, the fundamental problem of proarrhythmia is the long-term treatment, rather than acute treatment to achieve cardioversion, since the patient is hospitalized.

The effectiveness of electrical cardioversion was similar to what would be expected from the literature.[9] In most series, 15%-25% of cardioversions are ineffective, so interventions and methods have been tried in an attempt to obtain better results. As Tercedor and Álvarez point out, the use of an alternative paddle position or greater discharge energy could have increased its effectiveness. Nevertheless, at the time when the patients´ data were being collected, the protocol followed in our hospital was the one described (anteroapical electrode position). Our group has considered this problem and in recent years we have made several studies to try to increase this percentage. In a prospective series of 89 patients with persistent atrial fibrillation studied in our hospital (unpublished data), two different electrode positions were compared randomly (anteroapical versus anteroposterior), but no significant differences were found in the success rate (81% versus 85%, respectively). No differences were found in the total or maximum energy required, number of shocks, and impedance values between the different electrode positions. Likewise, no significant correlations were found among the values of impedance and weight, body mass index, or body surface. In both groups the programmed energy was 200, 300, 360, and 360 J (higher than that used in our study), but the success rate, although slightly higher, was not significantly greater. We have not found that the administration of intravenous flecainide immediately before electrical cardioversion improves its effectiveness in a series of 53 patients with persistent atrial fibrillation[10] (73% success in the flecainide group versus 85% in the control group).

To conclude, our objective was not to recommend the systematic use of quinidine – which we know has a series of limitations that are discussed in our article (like a longer mean stay) – but to remind readers that other alternatives to electrical cardioversion exist, which are often as effective as electrical cardioversion and whose use is conditioned by experience with the drug used.

Vicente Climent Paya, José Valencia Martín, Francisco Marín Ortuño, Fernando García de Burgos[a] and Francisco Sogorb Garri

Servicio de Cardiología. Hospital General Universitario de Alicante. [a]Servicio de Cardiología. Hospital General de Elche. España.

REFERENCES

1. Valencia J, Climent V, Marín F, Monmeneu JV, Martínez JG, García M, et al. Eficacia de la cardioversión programada en la fibrilación auricular. Comparación de dos esquemas de tratamiento: cardioversión eléctrica frente a cardioversión farmacológica. Rev Esp Cardiol 2002;55:113-20.
2. Van Gelder IC, Tuinenburg AE, Schoonderwoerd BS, Tieleman RG, Crijns H. Pharmacologic versus direct-current electrical cardioversion of atrial flutter and fibrillation. Am J Cardiol 1999; 84:147-51.
3. Alpert MA. Medical cardioversion of atrial fibrillation. Chest 2000;117:1529-31.
4. De Nooijer C, Sparling CM. Quinidine treatment of chronic lone atrial fibrillation. Clin Cardiol 1990;13:711-4.
5. Zehender M, Hohnloser S, Muller B, Meinertz T, Just H. Effects of amiodarone versus quinidine and verapamil in patients with chronic atrial fibrillation: results of a comparative study and 2-years follow-up. J Am Coll Cardiol 1992;19:1054-9.
6. Lau CP, Leung WH, Wong CK. A randomized double-blind crossover study comparing the efficacy and tolerability of flecainide and quinidine in the control of patients with symptomatic paroxysmal atrial fibrillation. Am Heart J 1992;124:645-50.
7. Southworth MR, Zarembski D, Viana M, Bauman J. Comparison of sotalol versus quinidine for maintenance of normal sinus rhythm in patients with chronic atrial fibrillation. Am J Cardiol 1999;83:1629-32.
8. Minardo JD, Heger JJ, Miles WM, Zipes DP, Prystowsky EN. Clinical characteristics of patients with ventricular fibrillation during antiarrhythmic drug therapy. N Engl J Med 1988;319:257-62.
9. Lévy S, Brithardt G, Campbell RW, Camm AJ, Daubert JC, Allessie M, et al. Atrial fibrillation: current knowledge and recommendations for management. Eur Heart J 1998;19:1294-320.
10. Climent V, Marín F, Mainar L, Gómez-Aldaraví R, Martínez JG, Ibáñez A, et al. Effects of pretreatment with intravenous flecainide on efficacy of external cardioversion of persistent atrial fibrillation [abstract]. Eur Heart J 2001;22(Suppl):19.