

Editorials

Magnesium: an antiarrhythmic drug, but only against very specific arrhythmias

See page 1177 for the article to which this Editorial refers

Magnesium has been shown to have important electrophysiological effects when given acutely intravenously^[1,2] and some studies have suggested that magnesium depletion has an important role in the occurrence of postoperative atrial arrhythmias^[3]. The most important electrophysiological effect of magnesium is, without any doubt, its ability to prevent the episodes of drug-induced torsade de pointes^[4]. Because torsade de pointes is a polymorphic arrhythmia at the ventricular level, and because of the above-mentioned observations, it is reasonable to ask whether magnesium may have a preventive effect on polymorphic atrial arrhythmias such as atrial fibrillation. That is the question addressed by Frick and co-workers in this issue^[5]. The authors compared, in a well designed and executed study, the effects of sotalol alone or in combination with oral magnesium, to placebo in the prevention of recurrences of atrial fibrillation after cardioversion. Their results unequivocally show that magnesium alone does not prevent atrial fibrillation, and that when added to sotalol does not contribute to a decrease in the incidence of recurrences. The authors conclude that oral magnesium was not effective in the treatment of atrial fibrillation, including the control of the ventricular rate during the arrhythmia. However, the most important, though justified, speculation the authors make is that electrophysiological effects reported after intravenous magnesium may derive from the transient production of hypermagnesaemia and not from correction of hypomagnesaemia. This has some practical consequences in the daily utilization of magnesium in patients with cardiac arrhythmias.

Although not registered as an antiarrhythmic drug, magnesium is extensively used by general practitioners (at least in our country) as an adjuvant to antiarrhythmic drugs given for a variety of indi-

cations, from ventricular extrasystoles to atrial fibrillation. For this last arrhythmia at least, the study by Frick *et al.* clearly shows that oral magnesium is not indicated. Magnesium has important electrophysiological effects when given intravenously and will remain the main drug in the treatment of torsade de pointes, but no other indications exist for it as an antiarrhythmic drug. Interestingly, both oral and intravenous magnesium is available without medical prescription in several European countries, including Belgium. It is unlikely that oral magnesium will ever be taken in such high doses (except in suicidal attempts) as to result in conduction disturbances and even asystole. On the contrary, a rapid intravenous injection of magnesium can result in immediate asystole. With these data it seems justified not to register oral magnesium as an antiarrhythmic drug, but at the same time all countries should forbid the sale of intravenous magnesium without medical prescription.

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