
To the Editor:

In their editorial, “Matter of Fat: Are Lipids Antiarrhythmic?,” Drs Wan and Boyden' comment on our article, “Dyscholesterolemia protects against ischemia-induced ventricular arrhythmias.” In our article, we have presented evidence that increased sarcolemmal cholesterol content provides an antiarrhythmic effect mediated through an increased Ca\(^{2+}\) current and a resulting prolongation of the ventricular action potential.

In their editorial comment, the authors unduly attribute some statements to us and we feel the need to correct the authors on some of these points.

First, Wan and Boyden state that we suggest that the observed antiarrhythmic effect during acute myocardial infarction is akin to that of a diet rich in fish oil. This is not correct. Contrary to hypercholesterolemia (which causes action potential prolongation and decreased ventricular fibrillation susceptibility), a diet rich in fish oil leads to shortening of the ventricular action potential and to increased susceptibility to ventricular fibrillation during acute myocardial infarction.

Second, the authors mention that no effects on the sodium current (\(I_{\text{Na}}\)) were described. This is not correct. Figure 2C contains data of the upstroke velocity of the action potential. As we state in the Discussion: “Action potential upstroke velocity is an adequate measure of \(I_{\text{Na}}\).” This modest reduction of current apparently did not lead to a significant decrease in conduction velocity and did not offset the antiarrhythmic effect associated with the action potential prolongation.

Third, the authors maintain that we “propose that patients with dyslipidemia may be protected from… arrhythmias” and that “The bigger question is whether the findings…should be translated to the clinic.” The authors seem to have read the article as yet another mouse model looking for a clinical application but have failed to appreciate that the described antiarrhythmic mechanism explains how the described antiarrhythmic mechanism explains how the observed antiarrhythmic effect during acute myocardial infarction.

Lipid-lowering therapy is important for the prevention of atherosclerosis and the associated cardiovascular mortality caused by acute myocardial infarction, however, hypercholesterolemia may be protective against reentrant arrhythmias and sudden death. However, shortening of the cardiac action potential by lipid lowering may in turn be protective against arrhythmias in the chronic phase of myocardial infarction, during which arrhythmias commonly originate from triggered activity based on the action potential prolongation. Thus, sarcolemmal lipid content is important for ion channel function. Its anti- or proarrhythmic effects depend on the underlying arrhythmia mechanism.

Disclosures

None.

Antonius Baartscheer, PhD
Marieke V. Weldkamp, PhD
Department of Clinical and Experimental Cardiology
Academic Medical Center
University of Amsterdam
Amsterdam, The Netherlands

Ruben Coronel, MD, PhD
Department of Clinical and Experimental Cardiology
Academic Medical Center
University of Amsterdam
Amsterdam, The Netherlands

L’Institut du Rythmologie et Modélisation Cardiaque (LIRYC)
University of Bordeaux
Bordeaux, France

References

Letter by Baartscheer et al Regarding Editorial, "Matter of Fat: Are Lipids Antiarrhythmic?"
Antonius Baartscheer, Marieke W. Veldkamp and Ruben Coronel

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