Response to Letter Regarding Article, “Dabigatran and Thrombin Modulate Electrophysiological Characteristics of Pulmonary Vein and Left Atrium” by Chang et al

We appreciate the concise comment from Dr. Lin et al1 regarding our recently published article entitled “Dabigatran and thrombin modulate electrophysiological characteristics of pulmonary vein and left atrium.”2 Thrombin has been shown to induce expression of multiple cytokines and proinflammatory and profibrotic responses in addition to its procoagulation effects.3 However, warfarin blocks several coagulation factors, which can activate various protease-activated receptors with different signal transduction pathways and alter ion channel expression.4 Thus, we do not know whether the electrophysiological and mechanical effects modulated by direct thrombin inhibitor could be extrapolated to the miscellaneous effects of warfarin on pulmonary veins and left atrium. In the previous studies, Warfarin and Antiplatelet Therapy in Chronic Heart Failure (WATCH) and Warfarin versus Aspirin in Reduced Cardiac Ejection Fraction (WARCEF) trials found a marginally higher incidence of paroxysmal atrial fibrillation in aspirin-treated group than in the warfarin-treated group in heart failure patients.5,6 These differences might theoretically be caused by the deleterious electrophysiological effects of aspirin, which interfered prostacyclin and nitric oxide production or by beneficial effects of warfarin.7 However, this study only treated healthy animals with dabigatran for 3 days, which is different from the long-term use of warfarin in clinical studies. It would be really helpful to compare the differences among various anticoagulant agents (warfarin, direct thrombin inhibitor, and factor Xa inhibitors) and antiplatelet agents (aspirin, clopidogrel, ticagrelor) on the electrophysiological and mechanical characteristics of the pulmonary veins and left atrium in models of disease and over longer durations.

Disclosures

None.

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