The Contribution of \( I_I \) and \( I_{K1} \) to Focal Activity in Atrial Fibrillation


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BACKGROUND

Atrial fibrillation (AF) is the most common arrhythmia and is associated with increased morbidity and mortality. In this progressive disease, electrical and structural remodeling are key components of arrhythmogenesis (2). Electrical remodeling refers to alterations in ion channel expression and activity and can lead to ectopic firing, which can originate from the right (RAA), the left atrial appendage (LAA), or the pulmonary veins (see myocardium, focal activity such as enhanced automaticity, early after depolarizations (EADs) or delayed after depolarizations (DADs) can trigger AF by entering a vulnerable substrate.

The molecular nature of \( I_{K1} \) and its role in the pacemaking process is still a matter of research. There is strong evidence for \( I_{K1} \) to flow through the slow N-type channel. \( I_{K1} \) is discussed to contribute to a stable pacing rhythm in autonomic cardiac cells (8) and may form the sodium-dependent background current \( I_{Na} \) (9). Taking into account that at least for N-type channels \( I_{K1} \) amounts to approximately 10% of total pacemaker current \( I_{K} \), a physiological role in cardiac automaticity becomes apparent. This contribution of \( I_{K1} \) to depolarizing pacemaker makes this component an interesting candidate for the generation and maintenance of pathological induced arrhythmic activities, in particular when considering that \( I_{K1} \) is known to contribute to autonomic automaticity in cardiac non-pacemaker cells under pathological conditions (7,8).

Definitely, more studies are needed to elucidate the role of \( I_{K1} \) in pacemaking, in particular with regard to atrial fibrillation, since \( I_{K1} \) carries a quantitatively significant inward current within a few milliseconds.

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