Characterization of low right atrial isthmus as the slow conduction zone and pharmacological target in typical atrial flutter

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摘要

Abstract

BACKGROUND: Previous electrophysiological studies in patients with typical atrial flutter suggested that the slow conduction zone might be located in the low right atrial isthmus, which is a path formed by orifice of inferior vena cava, eustachian valve/ridge, coronary sinus ostium, and tricuspid annulus. The conduction characteristics during atrial pacing and responses to antiarrhythmic drugs of this anatomic isthmus were unknown. METHODS AND RESULTS: Forty-four patients, 20 patients with paroxysmal supraventricular tachycardia (group 1) and 24 patients with clinically documented paroxysmal typical atrial flutter (group 2), were studied. A 20-pole halo catheter was situated around the tricuspid annulus. Incremental pacing from the low right atrium and coronary sinus ostium was performed to measure the conduction time and velocity along the isthmus and lateral wall in the baseline state and after intravenous infusion of procainamide or sotalol. In both groups, conduction velocity in the isthmus during incremental pacing was significantly lower than that in the lateral wall before and after infusion of antiarrhythmic drugs. Furthermore, gradual conduction delay with unidirectional block in the isthmus was relevant to initiation of typical atrial flutter. Compared with group 1, group 2 had a lower conduction velocity in the isthmus and shorter right atrial refractory period. Procainamide significantly decreased the conduction velocity, but sotalol did not change it. In contrast, sotalol significantly prolonged the atrial refractory period with a higher extent than procainamide. After infusion of procainamide,

the increase of conduction time in the isthmus accounted for 52+/-19% of the increase in flutter cycle length, and 5 of 12 patients (42%) had spontaneous termination of typical flutter. After infusion of sotalol, typical flutter was induced in only 6 of 12 patients (50%) without significant prolongation of flutter cycle length. CONCLUSIONS: The low right atrial isthmus with rate-dependent slow conduction properties is critical to initiation of typical human atrial flutter. It may be the potentially pharmacological target of antiarrhythmic drugs in the future.