Mechanism of adenosine-induced termination of focal atrial tachycardia

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摘要

Abstract

Focal Atrial Tachycardia. Introduction: Adenosine can terminate most focal atrial tachycardias (ATs). However, information about the termination mechanism is limited. This study investigated the effects and mechanism of adenosine on terminating focal AT using a three-dimensional noncontact mapping system. Methods and Results: The study consisted of 7 patients (4 men and 3 women; age 44 ´ 29 years) with focal AT. Cycle length variation and atrial activation pattern at baseline and just before AT termination by adenosine (3-12mg) were analyzed. Noncontact mapping demonstrated focal AT propagated from the origin (O) with preferential conduction and spread away from the breakout sites to the whole atrium. Compared to baseline AT, termination episodes revealed higher mean beat-to-beat variation of AT cycle length (11.7 ´ 11.7 msec vs 4.7 ´ 4.5 msec, P < 0.001) and standard deviation of normalized AT cycle length (0.033 ´ 0.014 vs 0.011 ´ 0.005, P < 0.001). In termination episodes, adenosine significantly decreased the peak negative voltage of AT-O (-27.2 ´ 15.3%, P < 0.01), preferential conduction (proximal: -32.1 ´ 18.7, mid: -28.4 ´ 22.8, distal portion: -29.6 ´ 21.4%, P < 0.01), and breakout (-31.4 ´ 12.5%, P < 0.01). However, adenosine did not affect voltage in nontermination episodes. Adenosine shifted the locations of AT-O in 5 of 10 AT episodes with termination. Mean number of shifting AT-O was 2.4 ´ 1.5 (range 1-4), with maximum shifting distance of 15.0 ´ 3.1 (range 10-19) mm. Focal activation at AT-O simply disappeared in all termination episodes and therefore was not due to conduction block within preferential conduction or breakout site. Catheter ablation lesions covered 50% of total shifting origins, without late recurrence. Conclusion: Adenosine-induced AT termination was associated with significantly decreased electrogram voltage, shifting AT-O locations, and disappearance of focal activation...