# Effect of high intensity drive train stimulation on dispersion of atrial refractoriness: role of autonomic nervous system

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## 摘要

## Abstract

OBJECTIVES: This study evaluated the effect of high intensity drive train (S1) stimulation on the atrial effective refractory period (ERP) and its relation to the autonomic nervous system. BACKGROUND: High intensity S1 stimulation was demonstrated to shorten the ventricular ERP and to increase dispersion of refractoriness. These effects may be due to local release of neurotransmitters. The response of the atrium and ventricle to neurotransmitters was different. The effects of high intensity S1 stimulation at the atrial tissue were evaluated. METHODS: Forty patients without structural heart disease were studied. In group 1, 20 patients, the atrial ERP was measured at 0, 7, 14, 21 and 28 mm away from the S1 site under both twice diastolic threshold and high intensity (10 mA) S1 stimulation. The same protocol was repeated after sequential administration of propranolol (0.2 mg/kg body weight) and atropine (0.04 mg/kg). In group 2, the other 20 patients, the atrial ERP was studied at three atrial sites (high lateral right atrium [HLRA], right posterior interatrial septum [RPS] and distal coronary sinus [DCS] with twice diastolic threshold and high intensity S1 stimulation at baseline and after sequential autonomic blockade. The three atrial sites were randomly assigned as the S1 location. RESULTS: In group 1, high intensity S1 stimulation shortened the atrial effective refractory period most prominently at the site of S1: (mean +/- SD) 13.3 +/- 6.4% ($p < 0.001$), 8.1 +/- 3.8% ($p < 0.001$), 4.8 +/- 4.3% ($p < 0.001$), 3.7 +/- 4.7% ($p < 0.001$) and 0.5 +/- 2.6% at 0, 7, 14, 21 and 28 mm from the S1 site,

respectively. The effect of high intensity S1 stimulation was blunted with propranolol and autonomic blockade but persisted after atropine alone. High intensity S1 stimulation also increased dispersion of refractoriness (from 23 +/- 11 ms to 31 +/- 12 ms, p = 0.01), which was eliminated with autonomic blockade. In group 2, high intensity S1 stimulation had similar effects at different locations (ERP shortening of 10.8 +/- 2.7%, 10.8 +/- 2.2% and 12.2 +/- 4.6% at the HLRA, RPS and DCS, respectively). The responses to sequential autonomic blockade were similar to those in group 1. However, high intensity S1 stimulation at HLRA increased dispersion of refractoriness, but at DCS it reduced dispersion of refractoriness. CONCLUSIONS: High intensity S1 stimulation led to local shortening of the atrial ERP and increased dispersion of refractoriness. These effects were blunted with propranolol and autonomic blockade. High intensity S1 stimulation at the HLRA increased dispersion of atrial refractoriness, whereas the same stimulation at the DCS decreased dispersion of atrial refractoriness.