ADENOSINE-INDUCED ST-SEGMENT DEPRESSION DURING THALLIUM-201 SCINTIGRAPHY IN CORONARY ARTERY DISEASE: ANGIOGRAPHIC AND HEMODYNAMIC DETERMINANTS

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To examine the angioographic and hemodynamic determinants of adenosine (AD)-induced ST-segment depression in patients with angioraphic coronary artery disease, 66 patients with angiographically documented coronary artery disease who underwent AD infusion (140 μg/kg/min for 6 min) thallium (Tl-201) myocardial scintigraphy were studied. Patients with previous myocardial infarction were excluded. AD-induced ST-DEP occurred in 22 patients, 14 of whom had chest pain. The onset of ST-DEP was at 3.9 ± 1.1 min after the beginning of AD infusion. Systolic blood pressure was higher in patients with than those without ST-DEP, both at baseline (152 ± 28 and 135 ± 23, respectively, p < 0.05) and during AD infusion (141 ± 27 and 126 ± 22 mmHg, respectively, p < 0.05). Rate-pressure product during AD infusion was significantly higher in patients with than in those without ST-DEP, both at baseline (1200 ± 345 and 1023 ± 3053, respectively, p < 0.05). The angiographic and scintigraphic results were as follows: ST-DEP: No ST-DEP

- Multivessel disease: 14 (64%) vs. 25 (58%), p = 0.1
- Collateral vessels: 18 (82%) vs. 11 (25%), p = 0.0001
- Tl-201 defect (area %): 82 ± 16 vs. 14 ± 16, p < 0.05
- Tl-201 lowest count (area %): 54 ± 14 vs. 52 ± 11, NS
- Tl-201 lung-heart ratio: 35 ± 0.8 vs. 31 ± 2.8, p < 0.05

Thus, AD-induced ST-DEP is related to 1) increase in rate-pressure product, reflecting myocardial oxygen demand, and 2) the presence of angiographic collateral circulation, which may predispose to coronary steal.

ALERT LEFT VENTRICULAR DEPOLARIZATION SEQUENCE IN LEFT BUNDLE BRANCH BLOCK IS NOT A CAUSE FOR FALSE-POSITIVE THALLIUM-201 DEFECTS.


False-positive myocardial perfusion defects (PD) are occasionally observed on exercise Tl-201 images in pts with complete left bundle branch block (LBBB) and normal coronary arteries. The cause for these artifacts is not well understood. Experimental animal data suggested that altered LV depolarization sequence reduces septal myocardial blood flow. We tested this hypothesis by comparing quantitatively myocardial distribution of Tl-201 during rapid RA pacing (normal depolarization) with that during rapid RV pacing (electrical LBBB) in 7 pts. who were 17±7 months after cardiac transplantation. Six of 7 pts had angioraphic normal coronary arteries and normal LV wall motion. One pt. with normal conduction on ECG, had 30-40% coronary stenosis and interosseous hypokinesia. During right heart catheterization for cardiac biopsies, RA pacing was performed at a mean peak heart rate of 140±14 bpm and Tl-201 was injected. During RV pacing again all 6 pts had normal quantitative Tl-201 images with RA pacing. With RV pacing again all 6 pts had normal quantitative Tl-201 images. Thus, Tl-201 myocardial distribution during pacing stress and electrically induced LBBB is normal. Therefore, it seems unlikely that altered sequence of LV depolarization itself can account for false-positive PD in pts with LBBB. The cause for these erroneous stress PD in LBBB remains elusive. Other physiological or functional factors should be considered.