

Date:

Title: PRE-OP DPTI/SPTI: PREDICTOR OF INTRA-OP ISCHEMIA?

Authors: I.R. Thomson, M.D., D.G. Lappas, M.D., C.W. Emerson, M.D., M. Ohtaka, M.D.,
B. Lytle, M.D., F.H. Levine, M.D., M.J. Buckley, M.D., E. Lowenstein, M.D.

Affiliation: Cardiac Anesthesia Group, Anesthesia Laboratory of the Harvard Medical School
at the Massachusetts General Hospital, and Cardiac Surgical Unit,
Massachusetts General Hospital, Boston, Massachusetts 02114.

Introduction. The diastolic pressure time index to systolic pressure time index ratio (DPTI/SPTI) has been proposed as an estimate of the myocardial oxygen supply-demand ratio.¹ Decreases in DPTI/SPTI are associated with subendocardial ischemia in experimental animals. We evaluated DPTI/SPTI and ECG ST segment changes before and during the periinduction period in patients with coronary artery disease to determine whether individuals who developed ECG changes of ischemia could be differentiated from their non-ischemic counterparts on the basis of DPTI/SPTI.

Methods. The study was approved by a human studies committee and informed consent obtained from all subjects. 44 patients about to undergo elective coronary artery revascularization were studied following premedication with morphine, scopolamine, and diazepam, radial and pulmonary artery cannulation, and placement of eighteen precordial ECG leads. In all patients, anesthesia was induced with thiopental (3-4 mg/kg) and pancuronium (0.1 mg/kg) administered to provide muscle relaxation. 23 patients received N₂O/O₂/halothane anesthesia. 21 received morphine anesthesia (1 mg/kg) with N₂O added following endotracheal intubation. Respiration was controlled throughout. Simultaneous AP, PCW, and precordial ECG were recorded 7 times: awake control; 1 min. post induction (PI), 5 and 10 min. halothane (or 0.5 and 1.0 mg/kg morphine) plus 1, 5, and 10 minutes post endotracheal intubation (PET). DPTI/SPTI was obtained directly by planimetry of high speed pressure tracings. Myocardial ischemia was defined as an increase in ST segment depression of at least 1.0 mm in any precordial lead. Statistical analysis was performed using Student's t-test for paired and unpaired data.

Results. Table 1. Significant changes in DPTI/SPTI were present one minute after induction of anesthesia and returned to control only at 10 minutes after endotracheal intubation. Maximal decreases in DPTI/SPTI were present 1 min. PI and 1 min. PET.

Seven patients (16%) developed ECG changes meeting our criteria for myocardial ischemia. Mean DPTI/SPTI was lower prior to anesthesia in these patients than in the patients who did not become ischemic (p < .05). The difference in DPTI/SPTI

between the ischemic and non-ischemic patients remained present five of six subsequent periods. In contrast, the systolic arterial pressure-heart rate product of ischemic patients was not significantly different from the non-ischemic patients prior to anesthesia.

Discussion. The periinduction period was associated with significant and sustained decreases in DPTI/SPTI in these patients with severe ischemic heart disease. These decreases in DPTI/SPTI were accompanied by development of ECG changes characteristic of subendocardial ischemia in 16% (7/44) of subjects studied. Patients who developed periinduction ischemia constituted a discrete subgroup characterized by significantly lower DPTI/SPTI prior to induction than the group which did not develop ischemia. This observation may provide a simple method for preoperative identification of patients at increased risk of developing intraanesthetic myocardial ischemia.

TABLE 1

DPTI/SPTI IN PATIENTS WITH SEVERE CORONARY ARTERY DISEASE

(Mean ± SEM)

	All Subjects (n = 44)	Non- Ischemic (n = 37)	Ischemic (n = 7)
Awake	1.30±.05	1.33±.05	1.06±.06 ⁺
PI 1 min.	1.06±.04*	1.07±.04	1.01±.11
5 min.	1.12±.05*	1.17±.05	.89±.13*
10 min.	1.18±.05*	1.22±.29	.95±.04*
PET 1 min.	1.02±.04*	1.06±.05	.81±.06*
5 min.	1.14±.05*	1.19±.05	.87±.08 ⁺
10 min.	1.21±.05	1.25±.05	.98±.07 ⁺

*p < .05 vs Awake

⁺p < .05 vs Non-Ischemic

References.

- Hoffman, J.I.E.: Determinants and prediction of transmural myocardial perfusion. Circulation 58:381-391, 1978.