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ABSTRACTS

Abstracts of Papers to Be Presented at the 39th Annual Scientific Session of the American College of Cardiology, New Orleans, Louisiana, March 18–22, 1990

This year 4,074 abstracts (original contributions) were submitted for evaluation. Each was graded by eight recognized authorities in a special area of interest. Acceptance for presentation was based on the relative grade ranking in each of the 30 categories.

Ample meeting space combined with the inclusion of poster sessions again this year permitted the 1990 Annual Scientific Session Program Committee to accept 1,032 abstracts, approximately 25% of the number submitted. Many excellent contributions were received for this year's competition, and we appreciate your support and interest.

Carl J. Pepine, MD, FACC
Chairman, 1990 Annual Scientific Session
Program Committee

Monday, March 19, 1990
10:30AM–12:00NOON, Room 23
Young Investigators' Awards Competition
The following five abstracts are from the winning entries in the Young Investigators' Awards Competition, 39th Annual Scientific Session.

IONTOPHORETIC TRANSMYOCARDIAL DRUG DELIVERY: A NOVEL APPROACH TO ANTIARRHYTHMIC DRUG THERAPY.

Boaz Avitall, MD,PhD, John Hare, BS, Gary Zander, MS, Charles Bockoff, MD, Patrick Tchou, MD, FACC, Mohammad Jazayeri, MD, FACC, Masood Akhtar, MD, FACC. Sinai Samaritan Medical Center, Milwaukee, WI.

Antiarrhythmic drugs often fail to achieve therapeutic effects without toxic systemic levels. Direct transport of drugs into the myocardium may circumvent this problem. Procainamide (PA) was delivered via a specially designed titanium lined chamber for 10 min. passively (PASS) or iontophoretically (IONTO) into 5 day MI's in 9 dogs (CHRO) and 3 hour MI's in 10 dogs (ACUT). PA concentrations in the CENTER of the exposed zone and at a REMOTE site were compared, along with systemic PA levels, refractory periods (ERP) and VT inducibility.

	CENTER		REMOTE	
Group	Endo	Epi	Endo	Epi
IONTO CHRO	36±21*+	2386±789*+	8±4*	11±6*
PASS CHRO	4±2	92±90	2±1	3±2
IONTO ACUT	24±12	1723±698+	10±3	58±33
PASS ACUT	19±17	1356±310+	10±9	69±76

*p<0.001 IONTO vs PASS CHRO +p<0.01 vs REMOTE Endo-Endocardium, Epi-Epicardium Values are μGm/Gm.

Intramural PA concentrations were intermediate between Endo and Epi. ERP increased in IONTO CHRO by 38±28 p<0.05. For over 3 hours after PA, the VT rate decreased by 95±13 B/M in 3 of 4 IONTO CHRO in which sustained VT was induced, and in 1 the VT was supressed over the same period. PA was distributed transmurally and was highly concentrated in the infarct zone (HPLC and immunohistochemistry). The data indicate: 1. Delivery of PA into the infarcted myocardium is feasible and effective. 2. PA concentrations in the infarcted tissues were above theraputic levels for three hours after PA exposure with only trace amounts systemically. 3. VT due to an old infarct can be slowed or suppressed by direct PA administration to the infarct zone.

FLOW ACTIVATES A SPECIFIC ENDOTHELIAL POTASSIUM CHANNEL TO RELEASE AN ENDOGENOUS NITROVASODILATOR

J.P. Cooke, M.D., Ph.D., F.A.C.C., E. Rossitch, Jr., M.D., N. Andon, J. Loscalzo, M.D., Ph.D., F.A.C.C., V.J. Dzau, M.D., F.A.C.C. Brigham & Women's Hosp, Boston, MA

Flow-mediated vasodilation in conduit arteries is endothelium-dependent. We now provide evidence that flow activates the K_{Ca} channel on endothelial cells to induce the release of an endogenous nitrovasodilator. Bovine aortic endothelial cells (EC) were cultured on microcarrier beads. Vascular rings without endothelium were contracted to norepinephrine in a flow chamber. EC added to the chamber released a diffusible vasodilator when stimulated by flow (at physiologic levels of shear stress). This vasodilation was abrogated by the antagonist of EDRF synthesis, L-NMMA and by the potassium channel antagonists tetraethylammonium (10^{-3}M; TEA) and charybdotoxin (10^{-8}M; CT) but not by glybenclamide (10^{-5}M). Conversely TEA or CT had no effect on endothelium-independent relaxations to sodium nitroprusside (SNP) or endothelium-dependent relaxations to acetylcholine (ACh). These observations were confirmed by perfusing iliac arteries in vitro at a constant pressure of 60±2mmHg, and monitoring diameter changes in response to flow rate using a videomicroscopy technique. Increases in flow from 1-4cc/min induced vasodilation (from 102-125% of vessel dia.) which were abolished by CT. Conversely endothelium dependent and independent vasodilations to ACh and SNP were preserved. These studies suggest that flow activates the K_{Ca} channel to release EDRF. We hypothesize that the K_{Ca} channel is the sensor/transducer of the flow stimulus, and that EDRF is the effector of flow mediated vasodilation.