9:15

C-11 HYDROXY-EPHEDRINE KINETICS IN PATIENTS WITH HYPERTROPHIC CARDIOMYOPATHY

Markus Schwaiger, Gary D. Hutchins, Sunil K. Das, Donald Wieland. University of Michigan, Ann Arbor, MI

Alterations of the cardiac sympathetic nervous system have been reported in patients with hypertrophic cardiomyopathy (IHSS). To evaluate the exogenous catecholamine uptake and storage in myocardial sympathetic nerve terminals, we assessed the tissue kinetics of the new catecholamine analogue C-11 hydroxy-ephedrine (HED) in 5 IHSS patients and compared the results to those obtained in 5 healthy volunteers. Twenty mCi C-11 HED was injected intravenously and dynamic PET scanning performed for 60 min. A 2-compartmental tracer kinetic model was used to fit regional time activity curves derived from myocardium and blood pool yielding HED tissue distribution volume as a marker of catecholamine storage capacity in sympathetic nerve terminals. Uptake of HED was homogeneous throughout the left ventricle paralleling the blood flow distribution as determined by the flow tracer rubidium-82. The HED distribution volume was with 6.7 ± 3.3 significantly decreased as compared to the average value of 14.9 ± 3.4 observed in the normal control population.($p<.05$) These data suggest globally decreased uptake and storage of exogeneous catecholamines in patients with IHSS, which may reflect loss of neurons or increased endogenously released catecholamines competing with C-11 HED for the uptake in sympathetic nerve terminals.

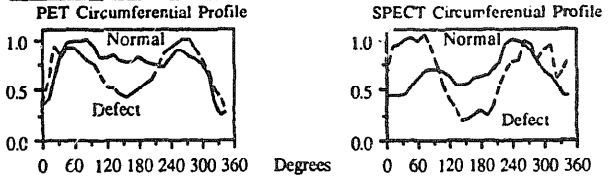
9:30

(^{60}Cu) COPPER PYRUVALDEHYDE THIOSEMICARBAZONE POSITRON EMISSION TOMOGRAPHY IN PATIENTS

Charles K. Stone, Charles C. Martin, Scott B. Perlman, and Robert J. Nickles. University of Wisconsin and VA Hospital, Madison WI.

No-carrier added (^{60}Cu)-copper pyruvaldehyde thiosemicarbazone (PTSM), a microsphere-like perfusion agent, has been investigated with PET. To assess the biodistribution, kinetics and image quality of ^{60}Cu-PTSM, PET scanning was performed on patients with normal or abnormal ^{201}Tl-thallium SPECT images. Dynamic PET images were acquired using 10 mCi of ^{60}Cu-PTSM and measured attenuation correction. Resting ^{60}Cu-PTSM PET images were compared by linear and circumferential profiles to resting ^{201}Tl- thallium images in terms of wall thickness (ratio of septum and lateral wall (LAT) to LV diameter) and uniformity (septum/LAT activity).

	Septum/LV Thickness	LAT/LV Thickness	Septum/LAT Activity
201-Tl	0.49±0.03	0.50±0.03	0.83±0.06
60-Cu-PTSM	0.33±0.05	0.41±0.04	1.11±0.04



Conclusions: Rapid uptake of ^{60}Cu-PTSM into the heart, brain, liver and kidneys was seen with a myocardial tissue to blood ratio of 5/1 that was stable over the 30 min scan duration. In addition, the PET images displayed excellent resolution of myocardial structures and perfusion defects, thus confirming the potential of ^{60}Cu-PTSM as a cardiac perfusion agent .

9:45

METABOLISM OF ISOMERS OF THE FREE FATTY ACID "PHENYLPENTADECANOIC ACID" IN THE HUMAN MYOCARDIUM - CLINICAL APPLICATION IN CORONARY HEART DISEASE

Ernst Vester, Klaus Peter Kaiser, Ludwig E. Feinendegen, Bodo E. Strauer. Med. Klinik B und Nuclear Research Center, University of Düsseldorf, Germany

The ortho-isomer of the Phenylpentadecanoic acid (oPPA) in contrast to the para-isomer (pPPA) is characterized by a markedly prolonged retention ("trapping") in the myocardium (T1/2: 200' vs 45'). To find out the site of trapping we performed in 8 pts an intracoronary injection of the double labelled isomers (131J at the phenylring and 14C in alpha-position) and measured the 14C- and 131J-catabolites in coronary sinus (CS) and aorta. OPPA produced in contrast to pPPA a much lower release of catabolites into the CS. To evaluate the clinical use of oPPA's trapping phenomenon SPECT was performed in 42 pts with CHD and 10 normals. Data were analysed quantitatively and correlated with coronary angiography. After overnight fasting and bicycle exercise 110-220 mBq of 123J-labelled oPPA were injected and SPECT was started. In 15 of the 42 pts a second dose of 123J-oPPA was injected 2 1/2 h later, and SPECT was repeated under resting conditions. OPPA revealed a global sensitivity of 76% for the detection of CHD (degree of stenosis > 50%): 78% for LAD, 84% for RCA and 80% for LCX. In the 15 cases with an additional rest study a redistribution analogous pattern was observed in more than 50% of regions being abnormal under exercise. In general there was an increase in reduction of oPPA-uptake according to the degree of stenosis as well as to the extent of wall motion abnormalities.
Conclusion: our results indicate, that oPPA being mainly retained in the lipid pool, while pPPA is readily metabolized by beta-oxidation, is highly suitable for the assessment of myocardial viability and perfusion abnormalities.

Thursday, March 7, 1991
8:30AM- 10:00AM, Room 205, East Concourse
ICD Therapy II

8:30

A MULTIPROGRAMMABLE ANTIARRHYTHMIA DEVICE FOR THE TREATMENT OF PATIENTS WITH VENTRICULAR TACHYCARDIA AND VENTRICULAR FIBRILLATION.

Gust H Bardy, MD, FACC, Charles Troutman, RN, George Johnson, BSEE, David M Gartman, MD, FACC, Jeanne E Poole, MD, FACC, Peter J Kudenchuk, MD, FACC, G Lee Dolack, MD, FACC, University of Washington, Seattle WA.

Until recently, device therapy of ventricular tachycardia (VT) and ventricular fibrillation (VF) has been limited. We describe our initial experience with a staged therapy multiprogrammable antiarrhythmia device, the Medtronic 7216 and 7217 model pacer-cardioverter-defibrillator (PCD), in 21 cardiac arrest survivors. A two patch epicardial lead system was used in 3 patients and a three patch system was used in 18 patients. The index arrhythmias leading to device implantation were VF in 9, VT and VF in 3, and VT only in 9. Defibrillation thresholds at implant averaged 4±1 J.

Postoperatively, all 21 patients benefitted from the additional functions available in the device. Four patients avoided the need for a pacemaker for bradycardia because of the device's VVI pacing function. Serial adaptive burst and ramp antitachycardia pacing prevented the need for cardioversion in 6 of 12 pts with VT. Low energy cardioversion (0.2-1.0 J) prevented the use of painful high energy shocks when antitachycardia pacing modalities were ineffective in the remaining 6 patients with VT. In addition, the option to program cycle length stability and VT/VF duration prevented inappropriate intervention into atrial fibrillation and nonsustained VT in 8 pts. Two zones for detection and therapy, one for VT and one for VF, improved specificity of device response. However, difficulties in discriminating fast VT from slow VF led to overly aggressive treatment of VT in 6 patients. A detailed log in the PCD memory of the last arrhythmia treated facilitated troubleshooting. Also, noninvasive reprograming allowed for refinements in detection and therapy options in all 21 patients. Finally, the ability to perform noninvasive EPS, obviated the need for recurrent EP study in 7 patients. Thus, the functions available in a multiprogrammable antiarrhythmia device provide a substantial advance in the treatment of patients with disabling or lifethreatening ventricular arrhythmias.