Early Defibrillation Training Courses for Lay Volunteers Without Cardiopulmonary Resuscitation Education: The Placenza Progetto Vita Experience

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Background. The major determinant of survival after witnessed out-of-hospital sudden cardiac arrest due to ventricular fibrillation is bystander cardiopulmonary resuscitation (CPR) and early defibrillation. The role of CPR is not still clarified.

Methods. The aim of Progetto Vita, the first European project of early defibrillation in the community, was to train lay volunteers to early defibrillation with an AED(Heartstart FR). We focused our effort on training lay volunteers to perform early defibrillation. No specific instruction for CPR was provided. The training courses included 4 hours of theoretical and practical lessons. Four instructors trained 12 volunteers during each session. In particular participants were instructed to recognize the absence of breath and check for caution of the AED: if no they were instructed to turn on the device and to follow the voice instructions of the AED. A final examination was performed. At 6 months interval all lay volunteers after first certification, performed review tests. Results. Thirty-nine AEDs were distributed covering a 173,114 inhabitants area (1 AED/4438 inhabitants). A total of 1285 lay volunteers were trained. From June 6th 1999 to April 30, 2001 a total of 354 sudden cardiac arrest occurred. The lay volunteers trained 143 sudden cardiac arrest patients. They needed an average time of application of the AED of 40±13 seconds. At the retraining course of 1 hour performed with a practical exam, only 16 lay volunteers (1%) failed the review test. Survival rate from ventricular fibrillation was 44.1% compared to 21.2% (p < 0.05) in the group treated by EMS only. No negative consequences have been recorded.

Conclusion. The lay volunteers intervention in the community doubled survival rate from ventricular fibrillation. A simple method of instruction to early defibrillation without CPR instruction seems feasible, reliable, safe and low expensive and create a group of competent AED operators.

POSTER SESSION

1025 Genomics and Myocardial Ischemia

Sunday, March 17, 2002, Noon-2:00 p.m.

Georgia World Congress Center, Hall G

Presentation Hour: 1:00 p.m.-2:00 p.m.

1025-31 The ScaI Anti-natriuretic Peptide Gene Polymorphism in Relation to Nonfatal Myocardial Infarction Patients With Angiographically Confirmed Coronary Heart Disease

Marcin Gruca, Danusz Cieczew, Wiltold Dubaniewicz, Bartosz Wasag, Radoslaw Targonski, Karolina Ochman, Rafał Dworakowski, Wojciech Sobociński, Bartosz Cunylo, Andrzej Rynkiewicz, First Department of Cardiology Medical University of Gdańsk, Gdańsk, Gdańsk, Poland, Department of Biology and Genetics Medical University of Gdańsk, Gdańsk, Poland.

Background: Coronary artery disease (CAD) and its major thrombotic complication, that is, myocardial infarction is multifactorial and interrelated phenotypes that are partially determined by genetic factors. Transition T2238C→C leading to the loss of the ScarI restriction site in the atrial natriuretic peptide (ANP) precursor gene and potentially to the translation of ANP with two additional arginines, has been suggested to be associated with salt-sensitive hypertension. The aim of our study was to investigate whether there is an association between the ScarI ANP gene polymorphism and history of nonfatal myocardial infarction patients with significant coronary artery stenosis confirmed by angiography. The presence of ScarI polymorphism was confirmed by means of an angiography (at least one coronary artery with ≥50% lumen narrowing).

Methods: The study was performed in 847 consecutive, Caucasian patients: 719 males and 128 females (mean age 47±11 years) with significant coronary artery stenosis confirmed angiographically. Site-specific guttural. Genotyping was performed by polymerase chain reaction of genomic DNA, followed by restriction enzyme digestion. Linkage disequilibrium signals for C→T variation were noted in the C and T alleles compared with the non-ischemic and control groups (only up to 20 myocyte layers deep). Immunolabeling for Cx40 protein in the ventricles of all hearts was performed by polymerase chain reaction, followed by forward slicing for immunohistological examination. Gpl, CD45R/B220, and Thy-1,2 expression was performed to confirm bone marrow reconstitution. Following this, myocardial infarction was induced by ligation of the left coronary artery. At the 4th week after myocardial infarction, recipient mice were sacrificed and slices of myocardial tissue were forwarded for immunohistological examination. The purpose of this study was to test the hypothesis that endogenous bone marrow could be mobilized into the ischemic and non-ischemic area after myocardial infarction. Methods: Bone marrow cells of enhanced-GFP transgenic mice were transplanted into lethally irradiated adult normal mice (C57BL/6J) at 5×10⁶ cells intravenously. Five weeks after bone marrow transplantation, flow cytometric analysis of GFP, Mac-1 (CD11b), CD3, CD8, and Thy-1,2 expression was performed to confirm bone marrow reconstitution. The results of this study demonstrate that the potential importance of Cx40 in human heart disease. An increased transcript steady-state level results in a significant up-regulation of Cx40 in subendocardial regions of ventricles in ischemic hearts, a feature that could potentially result in the anergy of conduction velocities and thereby increase the possibility of lethal ventricular arrhythmias.

1025-33 Circulating Bone Marrow Cells Differentially Into Cardiomyocytes in Infarcted Heart

Yuki Kurumizaki, Nippon Medical School, Tokyo, Japan.

Background: We tested the hypothesis that endogenous bone marrow could be mobilized into the ischemic and non-ischemic area after myocardial infarction. Methods: Bone marrow cells of enhanced-GFP transgenic mice were transplanted into lethally irradiated adult normal mice (C57BL/6J) at 5×10⁶ cells intravenously. Five weeks after bone marrow transplantation, flow cytometric analysis of GFP, Mac-1 (CD11b), CD3, CD8, and Thy-1,2 expression was performed to confirm bone marrow reconstitution. The results of this study demonstrate that the potential importance of Cx40 in human heart disease. An increased transcript steady-state level results in a significant up-regulation of Cx40 in subendocardial regions of ventricles in ischemic hearts, a feature that could potentially result in the anergy of conduction velocities and thereby increase the possibility of lethal ventricular arrhythmias.

1025-34 A Brief Episode of Myocardial Ischemia in the Rat Turns on Novel Sets of Genes

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Background: Brief episodes of ischemia can have profound effects on the myocardium by inducing cellular entities such as stunning and preconditioning. The purpose of this study was to identify, using DNA microarrays, genes up-regulated or down-regulated with brief ischemia. Methods: Three groups of rats were studied. In control rats (C), hearts were removed following anesthesia. Open chest sham-operated rats (SH) underwent 4 to 20 min intervention, and the ischemia/reperfusion group (IR) received 20 min of proximal coronary artery occlusion and 4 h of reperfusion. Total RNA was isolated from ischemic (I) and non-ischemic (NI) tissue of the IR group, and the SO and C hearts as well. Biotin-labeled cRNA was synthesized and hybridized with Affymetrix Gene Chip microarrays (which allows the study of 12,038 genes). cRNA synthesis and hybridization were repeated twice in each group with new tissue samples and chips in every group. Results: 20 genes were altered (greater or equal to 2.0 fold) in the SO compared to the C group, suggesting that surgery alone can alter gene expression patterns in the heart. I/R caused a significant up-regulation of HSPO70, HSP27, VEGF and INC5 genes (19, 3.9, 3.2 and 1.9 folds respectively) in the IS area compared to the NI tissue. Several other genes not previously considered as being modulated during brief ischemia were up-regulated. These included nerve growth factor inducible genes for factor A (3.2 fold) and antiproliferative secreted protein PC13 (2.1 fold), leukemia zinc protein (2.9 fold), cyanide type II receptor (2.0 fold), and precursor of macrophage inflammatory protein 2 (1.9 fold). Several genes including HSPO70 and HSP27 were confirmed by northern blot hybridization. Conclusion: A brief bout of ischemia as may occur with angina pectoris, turn on a significant number of genes, many of which have not been previously reported but may represent new therapeutic targets.