Title	Expression of Cyclooxygenase-2 Protein in Acute Myocardial Infarction
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S-C-11

Time-Frequency Analysis of Intracardiac Electrograms

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Abstract: Atrial fibrillation (AF) is the most common supraventricular arrhythmia and remains a challenge in curative catheter ablation. The irregularity of atrial activity during atrial fibrillation (AF) makes it very difficult to determine ablation sites in curative catheter ablation operations. The advantages of wavelet transform (WT) over conventional time- or frequency-domain analyses motivate us to develop a measure of the spatial organization of atrial activation processes during AF by means of WT based compression technique. Five channels of right atrial signals from 5 patients are analyzed. Experimental results showed that zero rates (ZR) of WT coefficients after compression can well characterize the complexity of atrial activity during AF and thus can offer helpful information for the choice of ablation sites. Table 1 below summarizes the statistics of ZRs belonging to different intracardiac electrograms.

Table 1: Statistics of ZR of Three Types of Signals

	SI	FL	AF1	AF2	AF3	AF4	AF5
M	96.5	95.0	94.0	94.1	93.8	93.6	93.8
SD	0.08	0.14	0.64	0.67	0.50	0.57	0.74

S-C-12

Expression of Cyclooxygenase-2 Protein in Acute Myocardial Infarction

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Background: Cyclooxygenase is a key enzyme in prostadglandins synthesis. Its inducible forms, cyclooxygenase-2 (cox-2), can be triggered by cytokines, growth factors and hypoxia. Under acute myocardial infraction (AMI), many cytokines are secreted in the heart. However, the role of cox-2 is still unknown. We hypothesized that cox-2 protein is activated in the heart after AMI which may have pathological significance.
Methods: A animal model of AMI was established by ligating the left anterior descending coronary artery of the 10-week old male Sprague-Dawley rats. Sixteen post-AMI rats were killed at day-3, day-7, day-14, and day-28 after AMI. Four sham-operated rats were used as controls. The expression of MIF was revealed by immunohistochemistry. The macrophage infiltration was also studied by immunohistochemical method using anti-ED1 antibody.
Results: There was no cox-2 staining found in sham rats. At day-3 after AMI, macrophages were found infiltrating into the infracted area but without cox-2 staining. At day-7, macrophage infiltration was increased and cox-2 protein was stained inside the cardiomyocytes near the infarct zone of the left ventricle. The cox-2 protein was most abundant at day-14 in the cardiomyocytes. At day-28, cox-2 protein expression was decreased though it was found in both cardiomyocytes and macrophages in the infarct zone, as well as at the walls of the blood vessels in both infarcted and non-infarct areas of left ventricle, compatible with its potential role in angiogenesis after AMI.
Conclusion: This study have demonstrated, for the first time, the expression of cox-2 protein in the cardiomyocytes after AMI which is closed timed to macrophage infiltration. Its role in cardiac remodelling after AMI therefore warrant further investigation. In addition, its late expression in the coronary arteries may implicate its potential role in angiogenesis.