

Detrended Fluctuation Analysis of Heart and Respiratory Rhythm in Atrial Fibrillation

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Introduction: Atrial fibrillation (AF) is a heart rhythm disorder, in which the origin of the heart cycle is displaced from sinoatrial node onto various places in the atrium. Heart rhythm becomes irregular and has an influence on respiratory rhythm as well as on cardiopulmonary coupling.

The aim: We applied detrended fluctuation analysis (DFA) method to find correlation properties of heart and respiratory rhythm in order to clarify behavioral pattern of cardiopulmonary coupling in AF.

Materials and methods: Patients with permanent atrial fibrillation were included (mean age 73; range 51-89 years). The control group was consisted of healthy subjects (mean age 41; range 35-45 years). Subjects were gender matched, 10 men and 3 women. ECG and respiratory signal were measured simultaneously. RR intervals were extracted from ECG. Then, RR and respiratory time series were formed. DFA method was applied on these time series in order to calculate scaling exponents: α_1 (short-range correlations) and α_2 (long-range correlations). Their relationship with heart rate (HR)/breathing frequency (BF) was examined by regression analysis.

Results: There was a statistically significant difference in breathing frequency between the groups, but there was not significant difference in heart rate. In comparison to the control group, there was a significant reduction of α_1 (RR) and α_2 (RR) in AF group, while α_2 (Resp) was significantly augmented in AF group. In regression analysis, there were significant intrasystemic relationships (α_1 (RR) vs. HR in AF group and α_1 (Resp) vs. BF in both groups).

Conclusion: There is an implication of a compensatory mechanism in regulation of the respiratory rhythm in AF, but we did not notice the existence of another neural compensatory mechanism in regulation of the heart rhythm. We can conclude there is a cardiopulmonary coupling in direction heart-respiratory system in AF, but not in the opposite direction because of impaired vagal stimulation of sinoatrial node.