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## OXYDATIVE STATUS AND ATRIAL FIBRILATIONS

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According to the recent epidemiological reports atrial fibrillation (AF) affects about 6 million people in Europe and the number is expected to double in the next 50 years (1). Because of the growing incidence, AF is considered as “the new epidemic” (2).

Despite being recognized as far back as ancient Greece and China, nowadays AF is a real challenge for cardiologists. The numerous antiarrhythmic drugs, namely blockers of  $\beta$ -adrenergic receptors and cardiac ion channels, as well as catheter ablation have inadequate efficacy on AF recurrences and progressive clinical course. Main limitation for these results is the poor understanding of arrhythmia pathogenesis (3). Thus, in the last years, the efforts of scientists are increasingly focused on the underlying mechanisms concerning AF initiation and recurrences.

Having in mind that the human organism is aerobic, and reactive oxygen species are constantly and inevitably produced, great attention is paid to the process of oxidation as a potent mechanism for AF development. Considerable evidence supports the role of oxidative stress for electrophysiological and structural remodeling of the atria (4). Close relationship has been established between rhythm disorder and oxidative stress: studies have established increased protein oxidation in atrial cardiomyocytes, elevated plasma oxidative markers and altered gene expression in patients with AF (5). Undoubtedly, the oxidative status rouses interest as a contributory factor for the arrhythmia onset and persistence. Protecting the heart from oxidative damage is assumed as a promising therapeutic target in AF treatment (6).

Summarizing data concerning oxidative processes in AF we should point out the following facts: most of the studies are *in vitro* and the results need confirmation in clinical practice; usually persistent and permanent AF has been analyzed and the study population is heterogeneous with additional diseases that cause oxidative stress by themselves; information about the oxidative status in the early hours of AF is scarce, incomplete and contradictory.

In this connection, the study performed by Dr. Negreva et al. is of great importance for experimental and clinical practice. Firstly, oxidative status is not examined one-sidedly – the authors determine simultaneously markers of the oxidant (Pl-MDA; Er-MDA) and antioxidant (Pl-GSH; Er-GSH; GSH-Px) processes. This approach allows for a complete and proper evaluation of the oxidative status.

Secondly, oxidative stress is established still in the very early hours of the rhythm disorder. This fact gives us ground to assume that increased oxidation and decreased antioxidant processes are relevant to AF initiating mechanisms.

Thirdly, measuring the oxidative markers' dynamics proves that oxidative stress persists after arrhythmia discontinuation and oxidative balance recovers slowly in time.

### REFERENCES

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