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Intra-operative mapping of atrial fibrillation: Lessons to be learned

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Epicardial mapping is routinely performed during cardiac surgery in our centers. One of the most recent projects include simultaneously endo-epicardial mapping of the right atrial free wall. The presence of focal fibrillation waves during atrial fibrillation (AF) can, beside ectopic activity, also be explained by asynchronous activation of the atrial endo- and epicardial layer and transmurally propagating fibrillation waves. In order to provide direct proof of endo-epicardial asynchrony, we performed simultaneous high-resolution mapping of the right atrial endo-and epicardial wall during AF in humans. Intra-operative mapping of the endo- and epicardial right atrial wall was performed during (induced) AF in 10 patients with AF (paroxysmal: N=3, persistent: N=4, longstanding persistent: N=3) and 4 patients without a history of AF. A clamp made of two rectangular 8x16 electrode arrays (inter-electrode distance 2 mm) was inserted into the incision in the right atrial appendage. Recordings of 10 seconds of AF were analysed to determine the incidence of asynchronous endo-epicardial activation times (≥15 ms) of opposite electrodes. Asynchronous endo-epicardial activation ranged between 0.9-55.9% without preference for either side. Focal waves appeared equally frequent at endocardium and epicardium (11% vs 13%, p=0.18). Using strict criteria for breakthrough (presence of an opposite wave within 4mm and ≤14 ms before the origin of the focal wave), the majority (65%) of all focal fibrillation waves could be attributed to endo-epicardial excitation. So in this study, we provided the first evidence for asynchronous activation of the endo-epicardial wall during AF in humans. Endo-epicardial asynchrony may play a major role in the pathophysiology of AF and may offer an explanation why in some patients therapy fails.

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New theory of cardiac arrhythmias in humans

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Objective: A new attempt to study the mechanism of arrhythmias and sudden cardiac death (SCD) analysis.

Methods: Information search in the literature, participate in conferences, discussions with Russian leading cardiologists.

Results: I was able to show that when considering the arrhythmia mechanisms, researchers have forgotten about the possibility of CMC excitations by mechanical pulses. Tests on the device "Cardiocode" were carried out. Under the influence of stress can be opened large arteriovenous anastomoses (AVA). Periodically, the pressure change in the arteries and veins. Venacava expands, increases tone and pulse waves start to path through the AVA on the walls of the vena cava to right heart. Mechanical impulses can excite the CMC from various points of the atria or ventricles, disrupting the sinus rhythm. The result appear extra systoles, tachycardia attacks, blocking blood flow and to any peripheral venous network sites, swelling. Longevity attacks of tachycardia and arrhythmia can lead to progressive fibrosis of the heart because of myocardial ischemia. This increases the likelihood of the onset of fibrillation and SCD.

Conclusions: Unhealthy lifestyle, the presence of opening/closing AVA can sometimes lead to a lot of diseases. To get rid of attacks cardiac arrhythmias and to get SCD prevention is necessary in some way to suppress the mechanical waves, jogging on circuit: aorta- artery- AVA- vein- vena cava- atria- ventricles. That is why the extra hearts beats occur with the same coupling intervals or the strictly identical periods of beats. In such cases "reentry" phenomenon has a mechanical nature.

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