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# Validation of a Novel Computer Model of Endo-epicardial Electrical Dissociation and Transmural Conduction during Atrial Fibrillation

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► **To cite this version:**

Ali Gharaviri, Mark Potse, Rolf Krause, Angelo Auricchio, Ulrich Schotten. Validation of a Novel Computer Model of Endo-epicardial Electrical Dissociation and Transmural Conduction during Atrial Fibrillation. EHRA Europace - Annual Congress of the European Heart Rhythm Association, Mar 2018, Barcelona, Spain. hal-01910697

**HAL Id: hal-01910697**

**<https://inria.hal.science/hal-01910697>**

Submitted on 1 Nov 2018

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1 **Validation of a Novel Computer Model of Endo-epicardial Electrical Dissociation and**  
2 **Transmural Conduction during Atrial Fibrillation**

3 Ali Gharaviri, Elham Bidar, Mark Potse, Sander Verheule, Stef Zeemering, Rolf Krause,  
4 Angelo Auricchio and Ulrich schotten

5 **Background:**

6 Recent studies demonstrated that transition from persistent to permanent atrial fibrillation  
7 (AF) in goats is characterized by an increase in endomysial fibrosis, particularly in the outer  
8 millimetre of the atrial wall. This phenomenon leads to loss of side to side connections in the  
9 subepicardial layer as well as between the subendocardial layer and the endocardial bundle  
10 network. Hence, increased fibrosis may contribute to dissociation of electrical activity  
11 between these two layers (endo-epicardial dissociation of electrical activity, EED) giving rise  
12 to transmural propagation adding to the overall complexity of fibrillatory conduction pattern.

13 **Purpose:**

14 The goal of this study was to assess the effect of epicardial fibrosis on endo-epicardial  
15 dissociation and breakthroughs during AF and to validate the data against endo-epicardial  
16 mapping in patients with AF.

17 **Method:**

18 To test this hypothesis, we developed a novel highly detailed computer model of the human  
19 atria, which includes an epicardial layer with two to three layers of fibre orientations and an  
20 endocardial bundle network based on a large variety of human imaging and anatomical  
21 studies. To validate the model and the observed correlation between endo-epicardial  
22 dissociation of electrical activity and breakthrough rates, we performed simultaneous endo-  
23 epicardial high-density mapping in 7 patients' right atria during cardiac surgery, 4 with  
24 longstanding persistent AF (AF) and 3 without a history of AF (SR).

25 **Results:**

26 Both in the clinical recordings and in the simulations, the degree of EED ranged  
27 approximately between 20% and 80%. In the patients, there was a trend towards more endo-  
28 epicardial dissociation in the patients with persistent AF. In the simulations, fibrosis clearly  
29 increased the EED from  $28.4 \pm 1.52\%$  to  $57 \pm 2.62\%$  and  $60.3 \pm 2.87\%$  in control, moderately  
30 fibrotic, and severely fibrotic models, respectively. Interestingly, the increase in EED activity  
31 was associated with an increase in the incidence of breakthroughs both in the patient data as  
32 well as in the simulations. Both in the mapping recordings and in the simulations, number of  
33 breakthroughs per cycle was correlated well with and the degree of endo-epicardial  
34 dissociated activity ( $r=0.61$ ,  $p < 0.05$  and  $r = 0.72$ ,  $p < 0.05$  for the patient recordings and the  
35 simulations respectively).

36 **Conclusion:**

37 Our results derived from the new computer model resembled well the basic  
38 electrophysiological characteristics obtained from simultaneous endo-epicardial recordings in  
39 humans. Importantly, the introduction of isolated atrial fibrosis in the epicardial layer resulted  
40 in an increase in EED and breakthrough rates.

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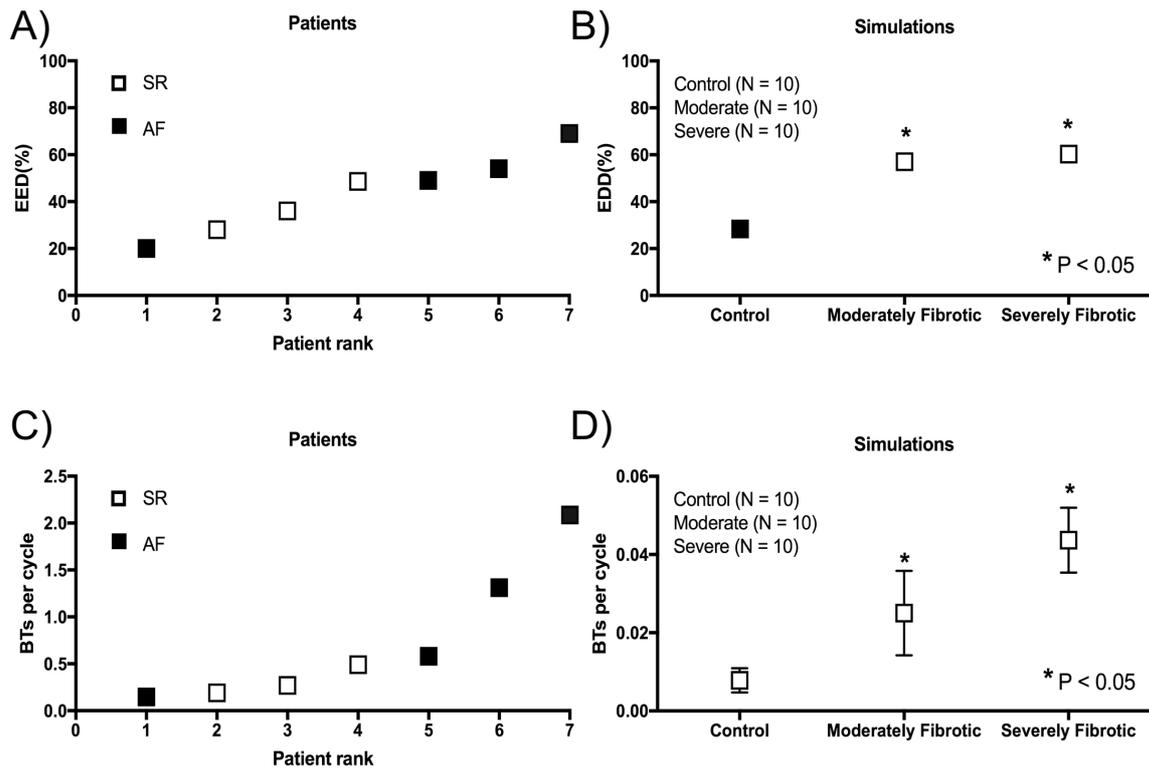


Figure: EED and breakthroughs. A) Endo-epicardial dissociation (EED) in patient recordings. B) EED in simulation. C). Number of breakthroughs (BTs) per cycle in human recordings. G) Number of BTs per cycle in simulations.

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