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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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## Background:

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

## Purpose:

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

## Method:

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

## Results:

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

## Conclusion:

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

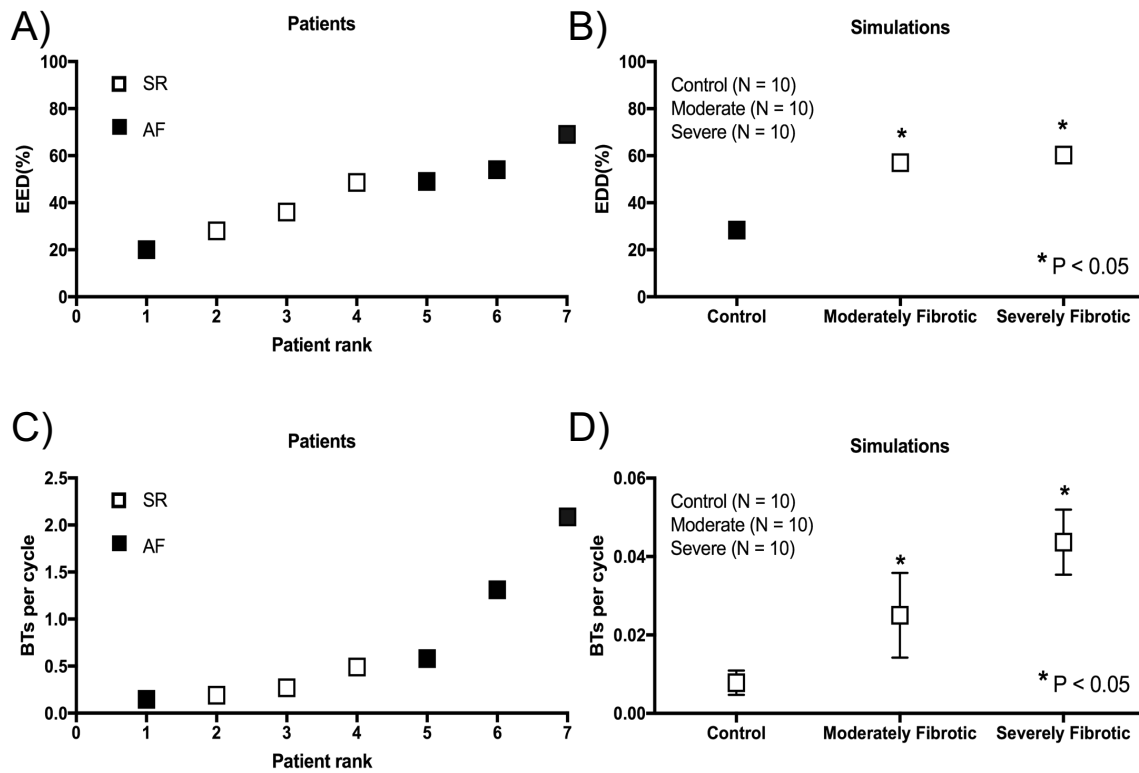


Figure: EED and breakthroughs. A) Endo-epicardial dissociation (EED) in patient recordings. B) EED in simulation. C). Number of breakthroughs (BTs) per cycle in human recordings. G) Number of BTs per cycle in simulations.

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44  
45