Model-based estimation of internal heart power in aortic valve disease patients.

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Introduction: Aortic valve disease (AVD) causes pressure overload of the left ventricle (LV) which may trigger adverse remodeling and, eventually, precipitate progression towards heart failure (HF). AVD can be treated by transvenous aortic valve implant (TAVI) which aims at reducing the transvalvular pressure gradient. However, depending on the specific AVD etiology, TAVI does not always reverse HF symptoms. **Objectives:** We aim to develop personalized computer models of LV electromechanics (EM) to predict acute TAVI-induced changes in LV function and derive clinical biomarkers such as internal heart power (IHP) which are believed to offer prognostic value of longer term outcomes. **Methods:** We developed $N = 15$ *in silico* EM models of LV and aorta of patients suffering from AVD. Models comprised electrophysiological, mechanical and circulatory components which were all personalized using clinical datasets including anatomical 3D whole heart MRI scans, LV volume traces, pressure drop across the valve as well as LV peak pressure. EM simulations of a heartbeat fitted to pretreatment conditions were carried out and validated against complementary clinical data such as strain and torsion measurements that were not used for model fitting. Based on the assumption of a reduced transvalvular pressure gradient < 20 mmHg, a change in characteristic impedance of the aorta was estimated to simulate post-treatment

conditions. IHP was computed under pre- and post-treatment conditions and compared against the image-based clinical estimation of these quantities. **Results and Conclusion:** The developed workflow enabled us to efficiently model LV EM in a larger cohort of AVD patients. All *in silico* models replicated clinical markers within prescribed margins of accuracy in terms of LV hemodynamics and deformation as assessed by pressure-volume loop analysis and circumferential slice strains, respectively. Model predictions of IHP were comparable to the clinically derived analog, however, non-negligible discrepancies were also observed in many cases likely attributable to limitations of clinical IHP calculations based on Laplace's law. Our study suggests that computational models provide a more accurate, reliable and robust method of determining IHP than currently used standard clinical procedures.