Model-based assessment of ventricular contractility

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Introduction: Evaluation of ventricular contractility is known to be one of the key step in assessing and diagnosing cardiac dysfunction. Experimentally, the conductance catheter method allows for determination of indexes of ventricular contractility but requires the recording of several pressure-volume loops during an induced preload reduction (caval vein occlusion). This highly invasive maneuver disturbs the circulation further and that is why it is not readily performed at the bedside for diagnostic purpose. In attempts to find less invasive methods to assess ventricular contractility, we show that computational models of the cardiovascular system in conjunction with clinical data can create patient-specific models that match clinical responses and data. This model-based diagnostic approach is applied to a porcine model of induced septic shock with continuous veno-venous hemofiltration to evaluate the right ventricle contractility. Methods: Septic shock was induced in (N=6) healthy pigs with endotoxin infusion over 30 min. Veno-venous hemofiltration was applied from 60 min. Right ventricular pressure-volume loops were recorded by conductance catheter and end-systolic ventricular elastance was assessed by varying right ventricular preload. All experimental procedures and protocols used in this investigation were reviewed and approved by the Ethics Committee of the Medical Faculty of the University of Liege. The computational model consists of 8 elastic chambers including the heart and circulations. We perform a previously validated integral-based identification method that only requires systolic and diastolic values of both ventricles volumes, as well as pressure in the aorta and in the pulmonary artery. Results: The model accurately captures all the pressures and volumes when compared to measured clinical data. Errors between the identified model and clinical data are within 10%. The model-based right ventricle end-systolic elastance ($E_{esrvf}$) is identified during the identification process and it is shown to correlate well (Fig. 1) with previously reported experimental data ($E_{es}^{*}$) of Lambermont et al. Conclusions: This model-based approach offers a potentially reliable procedure to assess the end-systolic elastance and consequently the ventricular contractility. This method thus opens up a new way to estimate the ventricular systolic function in humans due to its preload independence. It also offers the potential to develop a model-based sensor to monitor the contractility in clinical real-time and thus use it in determining therapy, using only clinical data available at the bedside.

Fig. 1. Experimental vs simulated elastance for all 5 analyzed pigs during the endotoxic shock experiment.

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Reference: