Influence of heart motion on EIT-based stroke volume estimation

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Abstract: Cardiac electrical impedance tomography (EIT) signals are affected by myocardial motion. The feasibility of stroke volume estimation using such signals is thus questionable. Results based on a dynamic model show that myocardial motion indeed affects but does not compromise stroke volume estimation.

1 Introduction

In EIT, cardio-synchronous impedance changes in the heart region are assumed to reflect variations of blood volume originating mainly from the ventricles [1]. EIT appears therefore as an interesting continuous and non-invasive modality for monitoring total ventricular volume (TVV), and thus estimating total stroke volume (TSV), defined as the maximal change in TVV over a full cardiac cycle. However, there is increasing evidence that other factors – unrelated to blood volume changes – are contributing to these variations of cardiac-related impedance [2]. In that context, simulations we performed on a finite element 2D extruded dynamic bio-impedance model showed that EIT signals in the heart region might be dominated by myocardial motion-induced changes [3].

These findings raised the question whether heart signals – affected by myocardial motion – remain valid for estimating changes in TVV and thus TSV. The hypothesis that the total impedance change in the heart area remains a true indicator for TVV and TSV is thus investigated here.

2 Methods

To test this hypothesis, we exploited the above 2D dynamic bio-impedance model – created from segmented magnetic resonance (MR) data imaged in the heart horizontal long axis plane – and considered three scenarios: In Scenario A, we reproduced cardiac blood volume-related impedance changes by simulating the filling and emptying of the cardiac cavities. In Scenario B, myocardial motion-induced changes were reproduced by simulating the dynamics of the heart muscle. Finally, Scenario C is the real-case scenario and simulates both blood volume-related and motion-induced changes [3].

These simulations were performed on our finite element model over a full cardiac cycle (corresponding to 20 simulated EIT frames) using the open source EIDORS toolbox, with image reconstruction carried out by the GREIT approach [4]. For each scenario, the impedance change ΔZ – with respect to end-diastole in the heart area – was computed for all frames, thus providing an EIT-based indicator for TVV, according to our hypothesis. Hereafter referred to as TVV_EIT, it was expected to perform best with Scenario A (no heart motion) and worse with Scenario B (heart motion only).

The reference TVV_REF was obtained by summing the volumes V_{LV} and V_{RV} of the left and right ventricles. V_{LV} and V_{RV} were computed via the area-length method [5] – with the areas (Σ_{LV} and Σ_{RV}) and lengths (L_{LV} and L_{RV}) coming from the MR data used to create our model:

TVV_{REF} = c_{LV} \cdot Σ_{LV}/L_{LV} + c_{RV} \cdot Σ_{RV}/L_{RV} (ml),

where c_{LV} = 8/(3π) [5] and c_{RV} = 2/3 [6]. L_{RV} was measured in the vertical long axis plane [6]. The total end-diastolic volume TEDV_{REF} and the total stroke volume TSV_{REF} inferred from TVV_{REF} (see Figure 1) were used to compute TVV_EIT by translating ΔZ – normalized by its maximal (systolic) value – into millilitres:

TVV_EIT = TEDV_{REF} – TSV_{REF} \cdot ΔZ_{NORM} (ml).

3 Results

The estimation error (mean±SD) between TVV_{REF} and TVV_EIT was of 1.9±13.3, –14.1±19.5 and –10.1±15.7 ml for Scenario A, B and C, respectively.

Figure 1: Total ventricular volume estimation using simulated EIT cardiac signals originating from blood volume-related impedance changes (A), motion-induced changes (B), or both (C).

4 Conclusions

In agreement with our expectations, simulations showed that myocardial motion increased the error on TVV_EIT and thus EIT-based TSV estimation, without however compromising the approach. When both blood volume changes and myocardial motion are in action (Scenario C, real-case scenario) an EIT-based TVV estimation error of –10.1±15.7 ml was obtained, which is sufficiently low to be clinically useful in normal subjects [5].

References