Origins of Cardiosynchronous Signals in EIT

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Abstract: EIT waveforms have components synchronous with cardiac activity, which are often assumed to originate from the blood perfusion. There are numerous other contributions to this signal. The goal of this paper is to provide a list of the factors which could be at the origin of these cardiac-related components, to help guide future research.

1 Introduction

As a functional imaging modality, EIT is understood to be sensitive to the movement of conductivity-contrasting gasses ($\downarrow\sigma$) and fluid ($\uparrow\sigma$). It is thus common to assume that the component of EIT images synchronous with breathing originates in the movement of air, and that the cardiac-frequency component originates from the perfusion of blood. The validity of these assumptions is important for the interpretation of EIT results. For example, simulations of chest-wall movement identified a breathing-frequency contribution to lung images [1], which contribute up to 20% of the signal due to ventilation, and depend on the geometry of chest wall movement. Thus, changing breathing movements (e.g. due to posture change) affects the ventilation-EIT signal relationship.

For the case of cardiosynchronous EIT signals, the problems of interpretation are more difficult. EIT is quite sensitive to heart activity, and measures of almost any part of the body show clear cardiosynchronous signals. In several publications, these signals have been called "EIT perfusion", suggesting the EIT signal is caused by blood flow. However, it is clear that continuous blood flow, by itself, cannot create EIT conductivity changes (Fig. 1). To avoid this problem various other terms have been proposed: 1) "perfusion-related EIT signals", which emphasizes that there is a relationship but not necessarily a correlation to blood flow, and 2) "pulsatilility" which emphasizes the relationship to the pulsatile signal. For a review of the terminology and its use, see [2]. Here, we recommend the neutral term "cardiosynchronous" EIT signals.

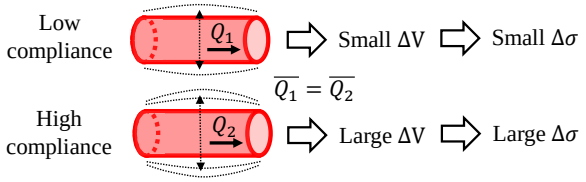

Figure 1: Arteries with equal perfusion (i.e. mean flow $\overline{Q_1} = \overline{Q_2}$) but different compliance lead to different conductivity changes $\Delta\sigma$.

Our goal in this paper is to collect a list of the possible sources for these cardiosynchronous signals. Some work has been done to model and understand the effect of heart deformation and displacement[3], and flow-induced blood conductivity changes[4], but a detailed list of the possible sources is not available.

2 Origin of Cardiosynchronous Signals

We first classify phenomena by their physical mechanism:
- Cardiosynchronous mechanical deformations: heart motion and the resultant movements of other structures.
- Blood volume changes: within the heart, a vessel or organ
- Red blood cell reorientation due to pulsatile blood flow [5]
- Reorientation of anisotropic structures: heart muscle, etc.

We then identify the possible sources and locations where the mechanisms listed might occur (Fig. 2).

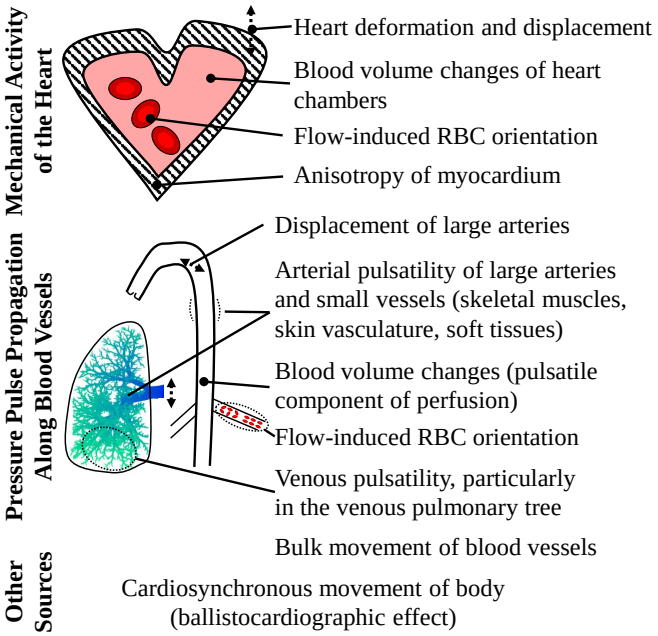

Figure 2: Diagram of possible sources of cardiosynchronous signals (by mechanism). The pressure pulse propagation is modulated by the pressures and compliances in the veins and arteries, and the compression and displacement of lung tissue due to lung arterial pulsatility and heart motion. Also, the pulsatile signal from venous return varies with intra-thoracic pressure. Finally, all factors will be affected by the spatial sensitivity of the EIT imaging system given the electrode positions and stimulation and measurement patterns.

EIT is sensitive to blood flow and many other cardiosynchronous effects, which we list here. Only some effects have been studied [4]. Our hope is that this list serves as a starting-point to better understand the origin and relative contribution of these signals, as a fraction of that from perfusion.

References

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