Mapping Myocardial Elasticity Changes after RF-Ablation Using Supersonic Shear Imaging

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Abstract

Shear Wave Imaging was used to monitor radiofrequency ablation (RFA) of myocardial tissues in vivo and in vitro. This technique was used to quantify and to map the myocardial stiffness before and after cardiac ablation. Experiments were performed in vivo on a sheep and in vitro samples of bovine muscle. The feasibility of mapping the myocardial elasticity was demonstrated in vitro after RFA. A strong increase of the myocardial stiffness was found after RFA. In vivo, the normal variation of the myocardial stiffness was measured during the cardiac cycle. The Young's modulus was found 8 times higher in the systolic phase than in the diastolic phase. During ablation a significant increase of the Young's modulus was observed in the diastolic phase whereas a sudden decrease was observed in systole.

1. Introduction

Radiofrequency ablation (RFA) has become a popular treatment for many cardiac arrhythmias [1][2]. Monitoring the ablation process is important to control the lesion size and ensure the complete destruction of the cardiac tissue causing the arrhythmia. Several imaging techniques have been investigated to monitor RFA of cardiac tissues. MRI has been shown to visualize accurately the lesion formation with good spatial resolution [3]. However, MR images are performed with a poor temporal resolution and the clinical use of MRI remains highly limited by its cost and the need of MR compatible treatment materials. On the contrary, intracardiac echocardiography is recognized as an excellent tool for real time guidance of the treatment, but is not able to detect accurately the lesion dimension after RF ablation [4]. Indeed, tissue echogeneicity does not always vary significantly after RFA, and in many situations the lesion is not visible at all using B-mode ultrasound imaging. Recently, elastography imaging has been proposed as an alternative technique to monitor the variation of tissue stiffness during RF ablation. Due to the strong temperature elevation in the ablated region, the coagulation of proteins induces a stiffening of the tissue that can be detected using several ultrasonic techniques based on the myocardium deformation such as strain rate imaging [5] or based on a remotely induced local mechanical excitation such as acoustic radiation force impulse imaging [6]. However, these techniques are sensitive to tissue motion which is very rapid in the heart and do not provide a quantitative estimation of the myocardial stiffness.

Shear Wave Imaging is a novel ultrasound-based technique for imaging non-invasively and quantitatively the elastic modulus of soft tissues [7]. This method is based on the generation of a shear wave (typically in the low kHz range) that propagates in soft tissues at a velocity of a few meters per second depending on the elastic modulus. The shear wave is generated via the acoustic radiation force of an ultrasonic beam focused in the myocardium by a conventional linear imaging array. The originality of this approach consists in acquiring ultrasound images of tissues at very high frame rates (up to 12,000 frames per second) just after the shear wave generation. Thanks to an ultrafast scanner, a complete movie of the transient event corresponding to the shear wave propagation through the organ can be provided and used to map the tissue stiffness. This technique has the advantage of being quantitative and very little sensitive to tissue motion thanks to its ultra high frame rate acquisition ability.

The objective of this study is to show the feasibility of using Shear Wave Imaging for monitoring tissue stiffness changes during RFA. The ability of mapping the thermal lesion is first shown on in vitro myocardium. Secondly, in vivo experiments are performed on a sheep heart and show that a significant variation of the myocardial stiffness is found after RFA.

2. Methods

Imaging sequence

Shear wave imaging is based on the remote generation of shear waves in tissues by the acoustic radiation force at the focus of the ultrasound field. A short duration burst (200µs) of focused ultrasound is transmitted by a conventional diagnostic ultrasonic probe (8MHz central frequency) to induce tissue displacements in a small focal zone of the arterial wall thanks to the acoustic radiation force. In response to that transient mechanical excitation, a shear wave is generated in the low kHz frequency range and propagates in the myocardium at relatively low velocities (between 1 and 10 m/s), depending on tissue elasticity according to Eq.1. The shear wave propagation is imaged at ultra-high frame rate (up to 12,000 images/s) using the same diagnostic probe. From the spatiotemporal data of the shear wave propagation, the shear velocity was computed and the elastic modulus E was derived from:

$$E = 3\rho c^2$$
 Eq.1

where c is the shear velocity and ρ the volumic mass.

For in vivo experiments, successive measurements were repeated rapidly over one single cardiac cycle. Each elasticity measurement was achieved in less than 15 ms and was repeated 18 times every 30 ms during 0.54 second allowing to measure variation of elasticity within one cardiac cycle.

In vitro experimental procedure

The experiment was performed in ex vivo bovine myocardium. The transducer was set on the top of the sample whereas the RFA catheter was positioned on the opposite side. A radio-frequency ablation device (EPTechnology, EPT-1000XP) was used to perform a 45 Watt, 60 s ablation. In order to achieve elasticity imaging of the entire sample, shear waves were successively induced at three lateral positions 10, 20 and 30 mm. For each position, the shear wave propagation was imaged, and thanks to an inverse problem algorithm the elasticity imaging was not possible during ablation due to a strong electrical noise induced by the RF device. However, elasticity measurements were performed immediately after ablation.

In vivo experimental procedure

We imaged the beating sheep heart. The study was approved by the Institutional Animal Care and Use Committee at Ecole de Chirurgie du Fer à moulin. A lateral thoracotomy was performed and the imaging probe was held in contact with the heart while attempting to maintain a fixed imaging plane for each acquisition. By holding the transducer in this manner, the transducer restricted the full expansion.

The transducer was on the postero-lateral wall of the left ventricle parallel to its short axis. Two-dimensional B-mode and shear wave propagation images were acquired for 0.54 s at a sampling rate of 33 Hz. ECG was recorded at the same time and synchronized with the acquisitions.



Figure 1: In vitro mapping of the ablated zone. Bmode ultrasound images and elasticity maps performed immediately after ablation (left side images) and 5 minutes after (right side images). Elasticity maps are given as shear velocity scale (m/s). The lesion is shown by the red box. The blue box represents normal tissues.

The previous RFA device was used to perform a 22.5 Watt, 240 s ablation on the epicardial surface of the free wall of the left ventricle. Every 30 seconds the RF probe was removed and stiffness measurements were performed. The ablation site was manually irrigated with 0.9% saline solution to maintain low temperature elevation on the RF catheter probe. Following the ablation, a lesion was visible on the surface of the heart.

3. **Results**

In vitro experiments

Immediately after ablation, the lesion was visible both on the elasticity map and on the Bmode ultrasound images as a hyperechogenic zone (See Figure 1). Elasticity maps show clearly a strong increase of the stiffness in the ablated region of several millimeters large. Elasticity maps performed every minute after RFA show that the stiffness continued increasing after ablation and reached a constant value only after 3 minutes. The stiffness in the ablated region was found to be approximately twice the stiffness of normal tissues (see Fig.2).



Figure 2: Comparison of the Young's modulus of in vitro normal tissue and ablated tissue. The values were computed in the blue and red boxes defined on Fig.1 for four different elasticity maps performed after RFA every minute.

In vivo experiments

The normal stiffness variation in myocardium before ablation over a single cardiac cycle is shown on Fig. 3. A large variation is found over the cardiac cycle: the myocardium is 10 times stiffer in systole than in diastole. The contraction of the fibers induces an important stiffening of the myocardium in the systolic phase followed by the muscle relaxation and softening in diastole. In the following results, a systolic myocardial stiffness is arbitrary defined as the Young's modulus measured at the end of the ST segment, and a diastolic myocardial stiffness was defined at the P wave.

RFA was performed, the ablation was stopped every 30 seconds, the catheter was removed and a stiffness measurement was performed. The lesion is clearly visible on the surface of the heart on Fig. 4 and its approximate location on the Bmode ultrasound image is shown on Fig. 5. The lesion was not visible at all on the Bmode images so that ultrasonic array was positioned approximately on the ablated region.



Figure 3: In vivo variation of myocardial stiffness as a function of time over one cardiac cycle in normal tissue (before ablation). The ECG is shown on the bottom.



Figure 4: In vivo ablation procedure. The lesion is visible at the surface of the left ventricle.

The variation of systolic and diastolic myocardial stiffness during ablation is reported on Fig. 5. The diastolic stiffness is found to increase slowly with the ablation time and after 240 s the myocardium was three times stiffer than before RFA. In systole, however, a sudden decrease of 36% of the initial stiffness was immediately observed when the ablation started. This decrease was followed by a slow recovery but the final value was 12% below the initial myocardial stiffness.



Figure 5: In vivo short axis Bmode images of the left ventricle at the ablated location. The red box indicates the approximate position of the lesion.



Figure 6: In vivo variation of the systolic and diastolic stiffness during RFA.

4. Discussion and conclusions

The feasibility of mapping RF ablated myocardial tissues using shear wave imaging was demonstrated in vitro. The myocardial stiffness was found to increase immediately after ablation and continued to increase slowly during a few minutes after ablation to reach approximately twice the initial stiffness. The lesion was imaged with a good contrast and millimetre resolution.

In vivo experiment shows that the myocardial stiffness of the normal heart varies strongly during the cardiac cycle. Due to the contraction of the myocardial fibers during the systolic phase, the stiffness increases rapidly by a factor of 8 compared to the diastolic phase. We defined arbitrarily a systolic and a diastolic myocardial stiffness at timings of the cardiac cycle where stiffness was almost constant. During RFA, we observed an important increase of the diastolic stiffness. In relaxed myocardial tissue, the effect of RFA was similar to the in vitro behaviour. On the contrary, the systolic myocardial stiffness which is mainly linked to the myocardium contraction was affected immediately by RFA and decreases significantly. This could be due to the fact that the myocardium contractility decreases rapidly just after tissue ablation. These preliminary data shows that myocardial stiffness can be used to assess the outcome of RFA. More experiments and animals are needed to understand the effect of thermal ablation on systolic and diastolic stiffness. Further work will focus also on in vivo mapping of the myocardial stiffness.

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