Supporting Information

Provost et al. 10.1073/pnas.1011688108

SI Text

Movies S1–S5: Electromechanical Wave Imaging (EWI) in Open-Chest Canines During Different Pacing Schemes. Strains displayed between −0.25% and 0.25%. Electromechanical activation resulted in positive strains (red) in most of the ventricles, with the exception of the apical region, where electromechanical activation resulted in negative strains (blue). This can be explained by the fact that the axial direction of the beam in that specific region is closer to the longitudinal direction resulting in longitudinal shortening (negative) instead of radial thickening (positive). Star indicates pacing site. Pacing time, 0 ms.

Movies S6 and S7: EWI in Humans. Strains displayed between −0.25% and 0.25%; 0 ms corresponds to the onset of the P-wave.

Movies S8 and S9: EWI in Humans (Electromechanical Activation Only). These movies were generated by inverting the sign of the strains in the ventricular regions showing negative strains at the onset of the QRS complex. Following this operation, strains larger than −0.025% were not shown. Strains displayed between −0.25% and 0.25%; 0 ms corresponds to the onset of the P-wave.

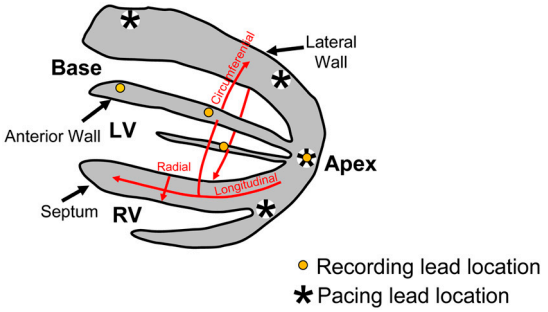


Fig. S1. Locations of the pacing and recording electrodes and representation of the radial, longitudinal, and circumferential coordinates of the heart.

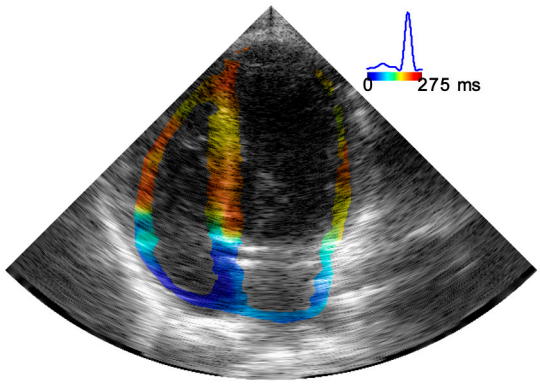
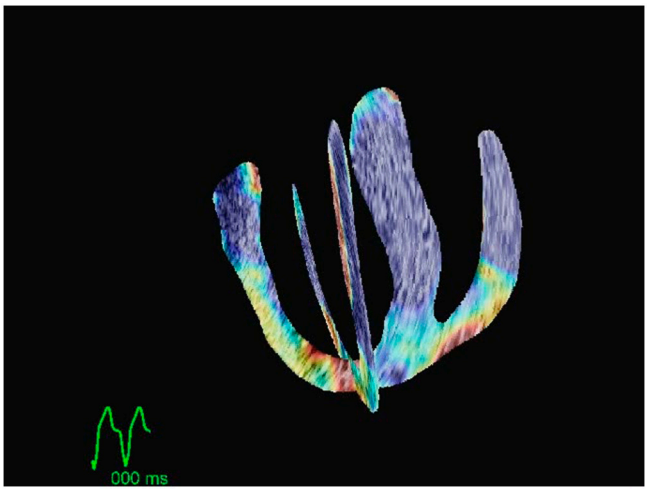
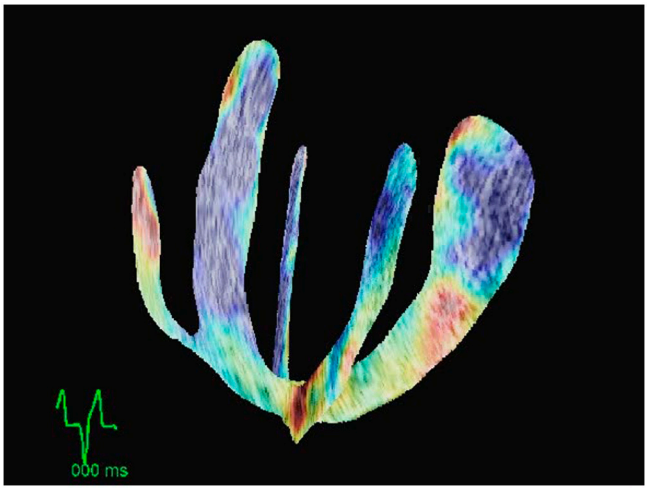


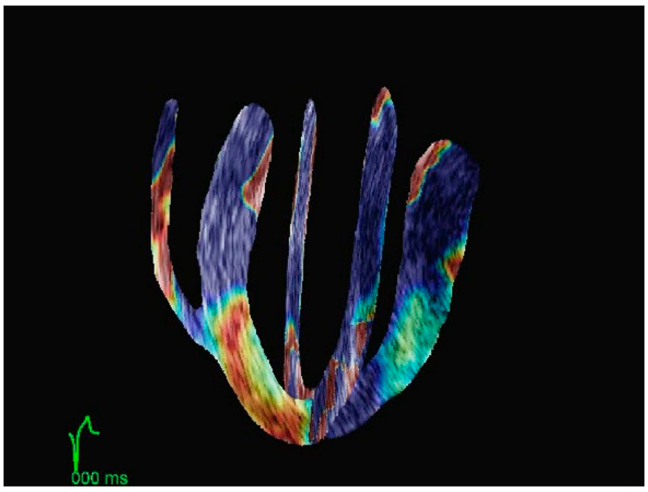
Fig. S2. Isochrones corresponding to Movies S7 and S9.



Movie S1. Pacing from the basal region of the lateral wall. At approximately 20 ms, the electromechanical wave (EW) originated at the pacing site and reached the apex at approximately 70 ms. At 60 ms, the EW is also observed at the base of the anterior wall. This could be due to the three-dimensional propagation of the EW. The EW then propagates in the septum and right-ventricular wall. Arrows show a single EW front.
Movie S1 (MOV)

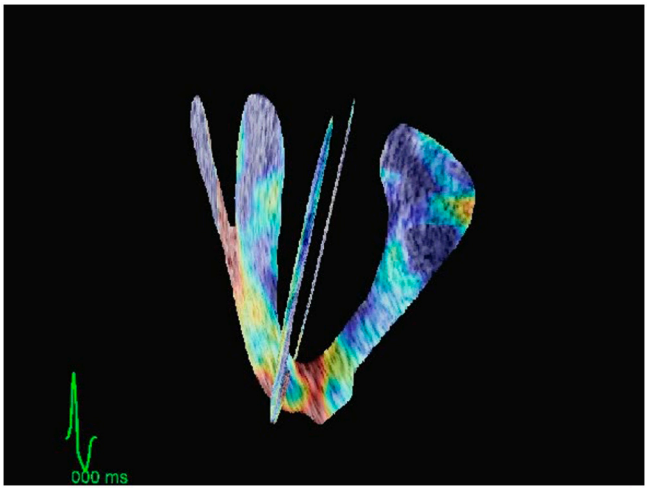


Movie S2. Pacing from the left-ventricular apex. At approximately 35 ms, the EW originated at the pacing site and propagated toward the base. Contrary to Movie S3, the EW propagated in a more symmetric way; i.e., the EW reached the base of the five walls approximately at the same time (i.e., at 120 ms).
Movie S2 (MOV)



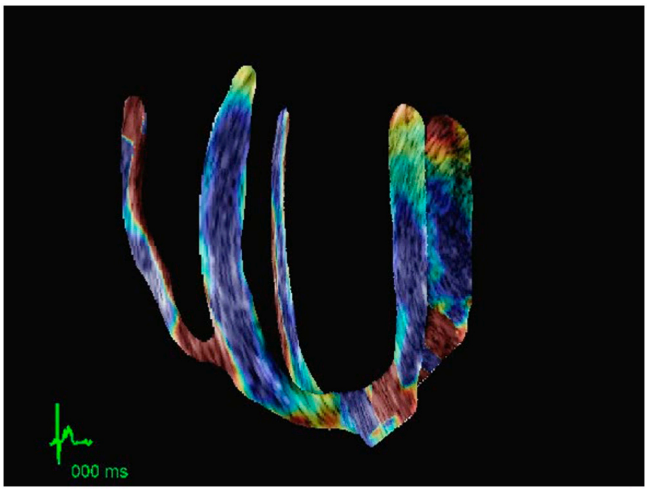
Movie S3. Pacing from the apical region of the antero-lateral wall. At approximately 20 ms, the EW originated from the pacing site and propagated toward the base. The EW first propagated in the lateral, posterior, and anterior walls and then in the septum and right-ventricular wall. Effectively, the EW reached the basal region of the lateral, posterior, and anterior walls at 80 ms, the basal level of the septum at 100 ms, and the basal region of the right-ventricular wall at 120 ms.

Movie S3 (MOV)



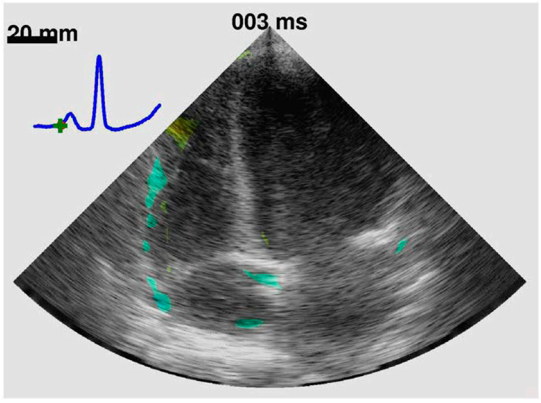
Movie S4. Pacing from the right-ventricular apex. At approximately 40 ms, the EW originated at the pacing site and propagated in the septum, the anterior wall, the posterior wall, the left-ventricular apex, and finally in the lateral wall.

Movie S4 (MOV)



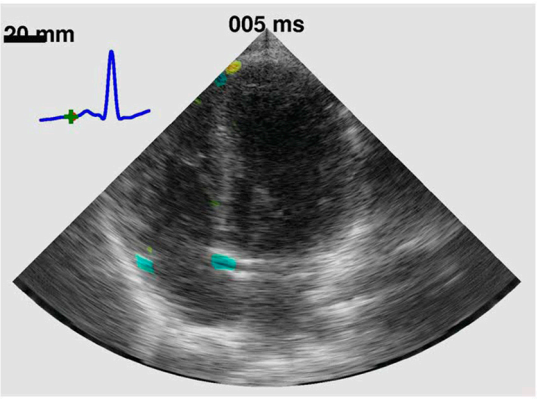
Movie S5. Sinus rhythm; 0 ms corresponds to the onset of the QRS complex. At approximately 30 ms, the EW originated in the septum, and in the anterior, posterior, and lateral walls, at the midlevel, and occurred 5–10 ms later in the right-ventricular wall. At approximately 70 ms, the EW had propagated in the five walls.

Movie S5 (MOV)

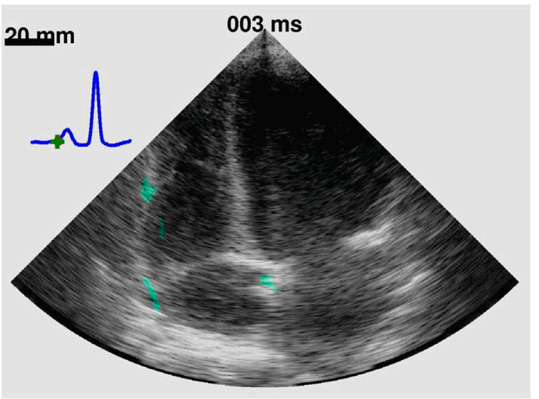


Movie S6. Four-chamber EWI in a normal 23-y-old female subject. At the onset of the P-wave, most regions experience minute myocardial strains; i.e., between −0.025% and 0.025%. This indicates that the heart undergoes no or only rigid motion; i.e., no contraction or relaxation of the muscle occurs. At approximately 30 ms, strains increase in the right atrium and propagate toward the interatrial septum. At approximately 90 ms, both atria undergo negative strain (compression). In contrast, the ventricular walls undergo mostly positive strains, except at the basal level of the right-ventricular wall. This can be explained by the fact that the axial direction of the beam in that specific region is closer to the radial direction resulting in radial thickening (positive) instead of longitudinal lengthening (negative). Shortening of the atria resulted in tethering of the ventricles; i.e., lengthening (positive). A clear discontinuity of the strains at the junction between the atria and the ventricles was observed, indicating that the atria contract while the ventricles remain passive. Over the next 100 ms, only small variations in strain patterns can be observed throughout all four chambers. At 190 ms, activation in the ventricles appear at the midlevel in the septum, and the EW can clearly be observed propagating in the septum toward apex and base. Similarly, in the right-ventricular wall, a previously lengthening region starts to shorten, and vice versa. At the same time, the atria begin to lengthen, from the left to the right atrium. This could be due to the relaxation of the atrial muscle following depolarization, and also from passive tethering caused by the ventricles' contraction. After 230 ms, the EW has propagated through the entire heart.

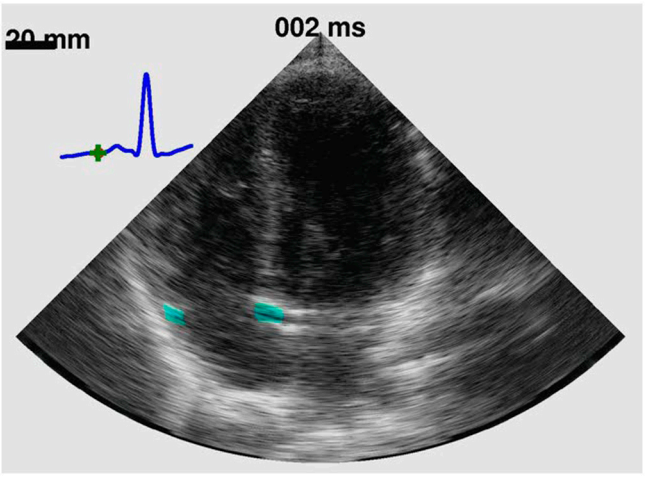
Movie S6 (MOV)



Movie S7. Four-Chamber EWI in a normal 23-y-old male subject. At the onset of the P-wave, most regions experience minute myocardial strains; i.e., between −0.025% and 0.025%. This indicates that the heart undergoes no or only rigid motion; i.e., no contraction or relaxation of the muscle occurs. At approximately 25 ms, strains increase in the right atrium and propagate toward the interatrial septum and the left atrium. At approximately 100 ms, both atria undergo negative strains (compression). In contrast, the ventricular walls undergo positive strains, except at the basal level of the lateral wall, where negative strains are mapped. This can be explained by the fact that the axial direction of the beam in that specific region is closer to the radial direction resulting in radial thickening (positive) instead of longitudinal lengthening (negative). Over the next 80 ms, only small variations in strains can be observed throughout all four chambers. At 180 ms, activation in the septum appears at the midlevel in the septum, and the EW can clearly be observed propagating in the septum toward apex and base. The EW also originated from the apical and basal regions of the lateral wall, propagating toward the midlevel of that same wall. At this same moment, the atria begin to lengthen, from the left atrium to the right atrium. This could be due to the relaxation of the atrial muscle following depolarization, and also from passive tethering caused by the ventricles' contraction. At 225 ms, the EW had propagated in the entire heart. Hence, the propagation in this subject is extremely similar to that of the subject of Movie S6, showing preliminary reproducibility of the normal electromechanical pattern unveiled by EWI.
Movie S7 (MOV)



Movie S8. Electromechanical activation corresponding to Movie S6. At the onset of the P-wave, the heart undergoes no or only rigid motion; i.e., no contraction or relaxation of the muscle occurs. At approximately 30 ms, strains increase in the right atrium and propagate toward the interatrial septum. At approximately 90 ms, both atria undergo negative strain (compression). A clear discontinuity of the strains at the junction between the atria and the ventricles was observed, indicating that the atria contract while the ventricles remain passive. Over the next 100 ms, only small variations in strain patterns can be observed throughout all four chambers. At 190 ms, activation in the ventricles appears near the base in the lateral wall and at the midlevel in the septum. The EW can clearly be observed propagating in the septum toward apex and base. Similarly, in the right-ventricular wall, activation is observed near the right-ventricular apex and propagates toward the base. After 230 ms, the EW has propagated through the entire heart.
Movie S8 (MOV)



Movie S9. Electromechanical activation corresponding to Movie S7. Four-chamber EWI in a normal 23-y-old male subject. At the onset of the P-wave, the heart undergoes no or only rigid motion; i.e., no contraction or relaxation of the muscle occurs. At approximately 25 ms, strains increase in the right atrium and propagate toward the interatrial septum and the left atrium. At approximately 100 ms, both atria undergo negative strains (compression). Over the next 80 ms, only small variations in strains can be observed throughout all four chamber. At 180 ms, activation in the septum appears at the midlevel in the septum, and the EW can clearly be observed propagating in the septum toward apex and base. The EW also originated from the apical and basal regions of the lateral wall, propagating toward the midlevel of that same wall. At 225 ms, the EW had propagated in the entire heart.

Movie S9 (MOV)