



IMAGING VIGNETTE

On-Line Visualization of Ischemic Burden During Repetitive Ischemia/Reperfusion

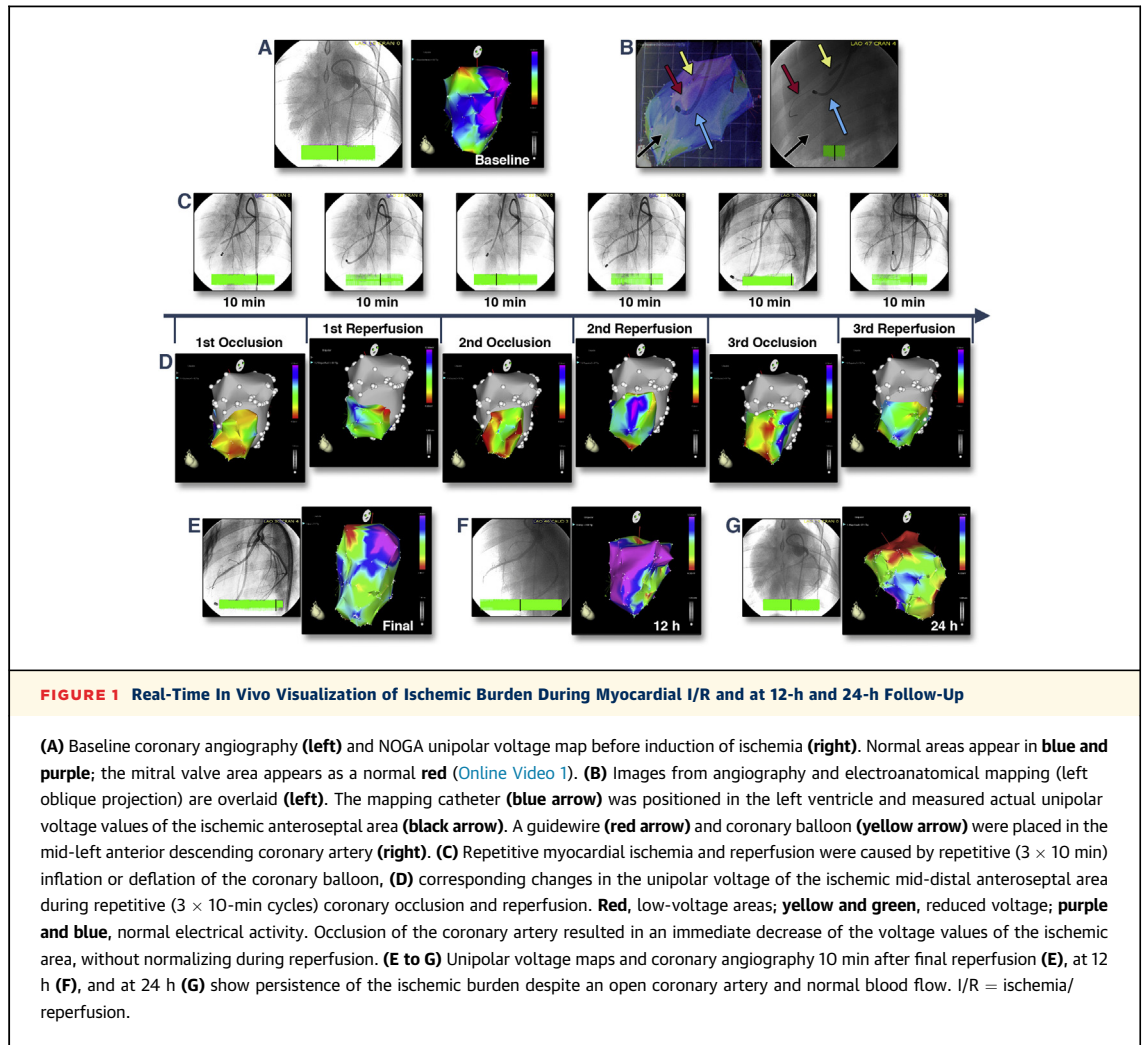


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ISCHEMIA/REPERFUSION (I/R) INJURY-INDUCED ALTERATIONS IN SARCOLEMMAL AND SARCOPLASMIC RETICULAR ION TRANSPORT result in typical changes in the electrical activity of myocardial cells that are manifested in surface and intracardiac electrocardiograms. Myocardial ischemia results in a progressive loss of the amplitude of the monophasic action potential, as evidenced by decreased endocardial unipolar voltage. To carry out in vivo on-line visualization, quantification, and explorative analysis of I/R injury and ischemic conditioning of the heart (1), we measured endomyocardial unipolar voltage with an endocardial mapping catheter (NOGA STAR, Johnson & Johnson, New Brunswick, New Jersey) (2) during 3 cycles of 10-min coronary occlusion/reperfusion in a closed chest I/R model in swine. Our goal was to simulate the ischemic burden of the human myocardium during repetitive episodes of angina pectoris. Here we display on-line regional unipolar voltage maps of the ischemia-affected myocardium (area mapping) and demonstrate immediate changes in unipolar voltage values taken from a single endocardial location within the ischemic area (single location mapping) during I/R.

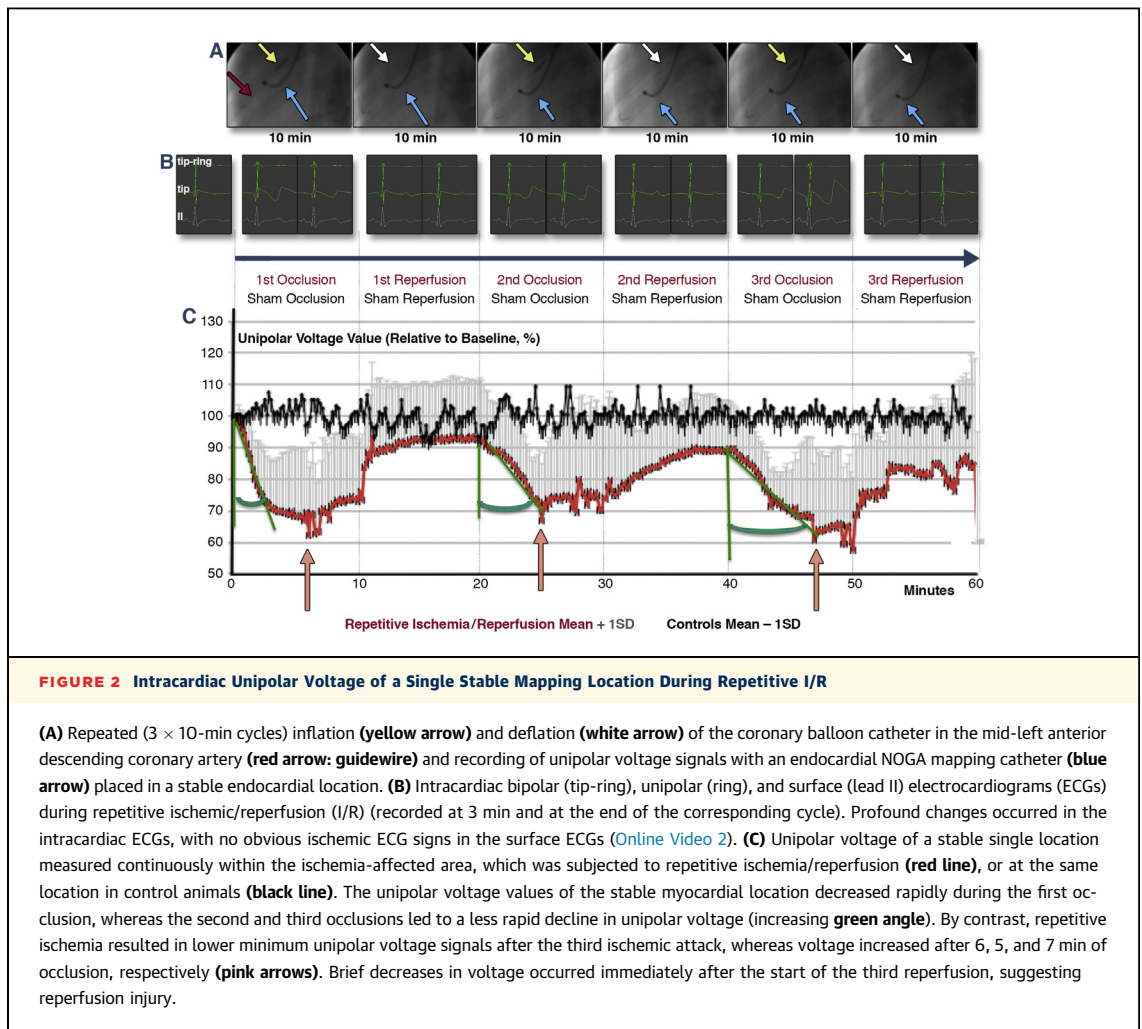
Domestic pigs (n = 5) underwent baseline electroanatomical mapping (Online Video 1) and cardiac catheterization (Figures 1A and 1B) followed by 3 cycles of 10-min I/R via percutaneous intracoronary balloon inflation/deflation of the mid-left anterior descending coronary artery (Figure 1C). All animal investigations conformed to the “Position of the American Heart Association on Research Animal Use,” adopted by the American Heart Association on November 11, 1984. After baseline mapping of the left ventricle, ischemic burden was displayed by moving the NOGA STAR catheter within the ischemia-affected mid-distal anteroseptal area between the 5th and 10th min of the ischemia or reperfusion (Figure 1C). The voltage values of the ischemic area immediately decreased during repetitive occlusion without normalizing during reperfusion; the ischemic burden persisted after the final reperfusion (Figure 1D), at 12 h (Figure 1E), and at 24 h (Figure 1F), despite the restoration of normal coronary blood flow.

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We recorded the unipolar voltage of a single stable distal anterior left ventricular location within the ischemic area during the I/R cycles without changing the location of the NOGA STAR catheter tip (n = 7), and compared these data to measurements from sham-procedure animals (n = 3) (Figure 2A). Surface and intracardiac electrocardiograms were continuously monitored (Figure 2B and Online Video 2). Due to the developing hypokinesia within the ischemic area, the amplitude of the endocardial catheter movement decreased, and the direction changed (Online Video 2). The unipolar voltage values of the stable myocardial location decreased rapidly during the first occlusion, while the second and third occlusions led to a less rapid decline in unipolar voltage (Figure 2C). By contrast, repetitive ischemia resulted in lower minimum unipolar voltage signals after the third ischemic attack. Interestingly, during permanent occlusion of the artery, unipolar voltage values increased slowly after approximately 5 min of ischemia.

In addition to providing basic scientific information on electrical signals of myocardium during ischemia and reperfusion, this method offers in vivo on-line visualization and immediate assessment of the extent of ischemic injury, as well as the efficacy of protective approaches against it, including pharmacological or ischemic conditioning, therapeutic hypothermia, and cardioplegia. Furthermore, this method allows for investigation of electrophysiologic state of the myocardium during pathological conditions affecting the heart (e.g., sepsis) in an animal model ready for clinical translation.



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APPENDIX For supplemental videos and their legends, please see the online version of this article.

KEY WORDS electroanatomical mapping, ischemia/reperfusion injury, ischemic burden