Myocardial Electrical Impedance Responds to Ischemia and Reperfusion in Humans

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Abstract

Myocardial electrical impedance (MEI) is correlated to ischemia and reperfusion of the heart muscle. The entire body of work with MEI to this point has been carried out in nonhuman animal subjects and excised tissue samples. In this study we measured MEI clinically for the first time in human patients who were undergoing off-pump coronary artery bypass (OPCAB) surgery. Our patient population had a 70-100% stenosis of the coronary artery targeted for bypass. We measured MEI continuously during surgery and at 3, 6, 24 and 72hrs postop from two temporary pacing electrodes attached to heart. MEI increased with occlusion of the diseased artery prior to bypass. The percent increase from baseline was correlated to the percent stenosis of the artery. MEI decreased below baseline immediately on reperfusion following bypass and continued decreasing over the measurement period. MEI is a reliable clinical indicator of ischemia and reperfusion in humans.

1.   Introduction

Myocardial electrical impedance (MEI) holds promise in detecting and assessing various disease states of the heart tissue. Through animal and tissue experiments, MEI has been shown to correlate with the vitality of the myocardial tissue. Myocardial electrical impedance changes predictably with regional and global ischemia [1-3], edema [4], pathologic tissue ultrastructural changes (4), ATP depletion and lactate accumulation [1]. Recently researchers have demonstrated that MEI can reliably detect humoral rejection episodes following heart transplantation [5-7]. Myocardial impedance has been used to compare myocardial preservation methods [3], measure the revivability of the heart [4] and to gauge tissue protection by beta-blockade from reversible ischemic damage [1]. We have shown in animal experiments that MEI increases immediately with ischemia and decreases with reperfusion [8]. In this study we measured MEI for the first time in human patients. Our patients were undergoing off-pump (moving heart)

coronary artery bypass (OPCAB) surgery, a procedure that gave us the opportunity to study acute ischemia and reperfusion of the heart tissue and follow patients post operatively for several days.

2.   Materials and methods

We obtained patient informed/written consent and institutional/FDA approval to conduct this study. Our MEI monitor has been designed and developed in this laboratory and has been described elsewhere [9,10]. Briefly, it consists of a laptop computer that communicates with and controls custom analog circuitry that, in turn, connects to the heart via two temporary pacing electrodes attached to the ischemic area of the myocardium approximately 1cm apart. These electrodes are routinely attached to the heart during cardiac surgeries at this institution. The control program for the MEI monitor is written in LabVIEW (National Instruments, Austin, TX). The monitor impresses a 5μA 100μs current impulse on the myocardium and measures the voltage response. The current and voltage signals are band-pass filtered between 0.27-5.90kHz, digitized at 22.0kHz and transformed by Fourier processing into the frequency domain. In this domain, MEI is calculated as the ratio of voltage and current at each frequency. Here, we report the average MEI modulus in the frequency range of the monitor. The monitor produces a new measurement every 3s.
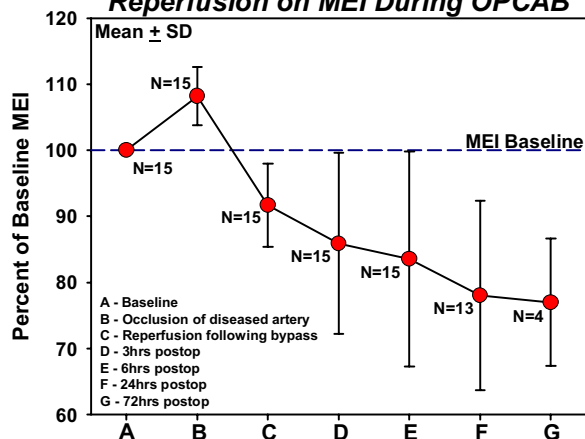
Fifteen patients undergoing OPCAB surgery (14 – left anterior descending coronary artery; 1 – right coronary artery) were enrolled in the study. The targeted surgery in this study involved 1) isolating the left interior mammary artery (LIMA), 2) occluding the diseased coronary artery, 3) anastomosing the LIMA to the diseased artery distal to the stenosis and 4) unoccluding the diseased artery and the LIMA thus causing reperfusion of the ischemic myocardial tissue. The stenosis of the diseased artery of our patients ranged from 70-100% as determined via cardiac catheterization. MEI was measured continuously during surgery and for 5min intervals at 3, 6, 24 and 72hrs postop or until the temporary pacing leads were removed from the patient's heart.

Our data were reported in percent normalized per patient to baseline impedance measurements made following exposure of the heart but prior to beginning the targeted procedure.
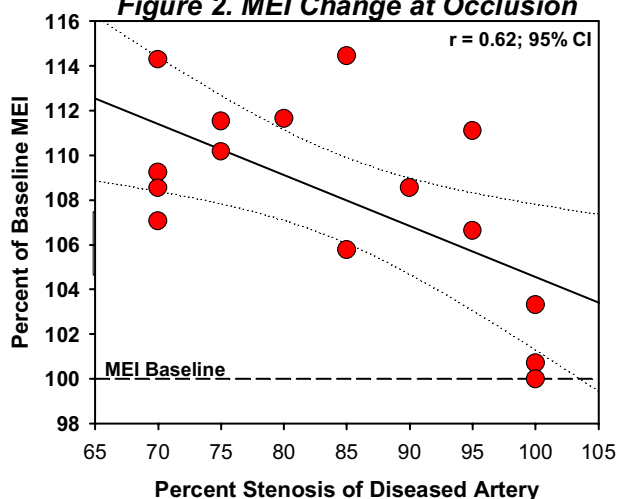
3. Results

Baseline MEI for our patients ranged from 173Ω to 769Ω. The large range in absolute impedance was due, for the most part, to differences in electrode separation and orientation with the myocardial fibers. On average, MEI increased to 108.2%±4.3% of baseline on occlusion of the diseased artery and decreased to 91.7%±6.3% on reperfusion (Figure 1, time point B and C, respectively). A significant relationship (r=0.62, p<0.05) existed between the percent increase in MEI on occlusion and the percent stenosis of the diseased artery taken from the cardiac catheterization report (Figure 2).

Figure 1. Effect of Occlusion and Surgical Reperfusion on MEI During OPCAB

A - Baseline
B - Occlusion of diseased artery
C - Reperfusion following bypass
D - 3hrs postop
E - 6hrs postop
F - 24hrs postop
G - 72hrs postop

Figure 2. MEI Change at Occlusion

MEI continued to decrease during the measurement period (Figure 1, time point D, E and F). The final measurement was 80.0%±9.6% of baseline (n=4) at 72hrs (Figure 1, time point G). There were no significant ST-segment changes observed during surgery in these cases.

4. Discussion

This is the first report of MEI measurements made clinically in human subjects to our knowledge. Our results in these clinical surgical patients corroborate an extensive amount of data reported in animal and excised tissue experiments over the past century.

It is not surprising that there were no measurable ECG ST-segment changes indicating ischemia or reperfusion during the surgeries. The electrical circuit pathways were disrupted because the chest of the patient was open and the heart was lifted and twisted to some extent in order to facility the coronary surgery. Under these conditions little significance could be paid to ST-segment analysis.

The mechanism involved in MEI changes with ischemia and reperfusion is a matter of conjecture. Our data, from a small number of subjects, suggest that MEI may be associated with the volume of conductive tissue, namely blood, in the region of the myocardium under study. Occlusion of the diseased artery stops the small amount of blood that is still flowing in the artery from perfusing the myocardium. In this study, there was little or no MEI increase in patients with a 100% coronary artery stenosis. The myocardium, in these patients, was being perfused by collateral circulation that had developed over the course of the coronary disease. On the other hand, MEI increased by as much as 14% in patients with a 70% stenosis. Their myocardium was still being perfused, to a small extent, by the limited coronary blood flow. MEI, however, may also be changing, to some extent due to electrolyte redistribution in the myocardium during ischemia and reperfusion. In any case, because MEI changes immediately with occlusion and reperfusion, it appears to be an effective measure of the vitality of the heart muscle and may indicate the effectiveness of coronary bypass surgery.

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