The Response of the Intracardiac Impedance Signal to Provoked Regional Ischemia during PTCA

J. WITTE, R. REIBIS
Humboldt Universität Berlin, Charité, Germany

J. STRÖBEL, M. SCHALDACH
Zentralinstitut für Biomedizinische Technik, Friedrich-Alexander-Universität Erlangen-Nürnberg, Erlangen, Germany

Summary

The electrical impedance between the tip of a ventricular electrode and a remote counter electrode changes with the geometries of the cardiac tissues and blood volume. The impedance waveform measured during each heart cycle reflects the speed of cardiac contraction and, thus, the contractile state of the myocardium.

Simulations and calculations have shown that the unipolar impedance waveform is primarily determined by the cardiac dynamics in the close vicinity of the electrode tip. This hypothesis has been verified with seven patients (mean age 53 ± 4 years, 2 female, 5 male) undergoing percutaneous transluminal coronary angioplasty (PTCA). During balloon inflation, the contractile state of the ischemic myocardium decreases and the non-ischemic tissue increases.

Introduction

The impedance measured via an electrode tip placed at the *right ventricular apex* (RVA), depends on the relative amount of low conductive myocardial tissue and high conductive blood within a region of 1 cm^3 around the electrode tip. Its value, therefore, changes during contraction. The morphology of the impedance waveform reflects the contractile process in close proximity to the electrode tip and, thus, of the local inotropic state of the myocardium.

Upon dilatation of the PTCA balloon, the sympathetic tone rises. The impedance waveform measured via the electrode tip will indicate an increased contractile state unless the electrode tip is placed at a region where the myocardium becomes ischemic. In this region, contractility decreases due to insufficient oxygen supply.

Methods

During PTCA, patients were provided with temporary atrial and ventricular electrodes. A guide wire in the inferior vena cava served as the counter electrode. All electrodes were connected to an external pacemaker system, which measured the impedance between the ventricular electrode tip and the counter electrode. Parameters were programmed to achieve P-wave synchronous ventricular pacing.

Patients with a previous myocardial infarction involving the area of measurement RVA, primary chronotropic insufficiency, or diabetes mellitus type I (insulin-dependent) were excluded from the study.

No cardiovascular drugs were administered to the patients on the day of the examination.

Before balloon inflation, impedance curves were generated for resting and exercise conditions to serve as a reference. Exercise consisted of approximately two minutes of weight lifting with small dumbbells. After exercise, the patient was allowed to recover for at least ten minutes.

Next, impedance curves were recorded from one minute prior to balloon inflation through one minute after reperfusion. Data was collected for the first dilatation only, since post-treatment application of drugs (*i. e.*, nitrates) alters the myocardial mechanics.

All impedance waveforms were measured between 122 and 286 ms after each pacing stimulus pulse. The impedance waveform is obtained after phase demodulation of the voltage signal generated by a 40 µA measurement current (4 kHz square wave). This waveform is digitized with a resolution of 0.4 W at a sampling frequency of 128 Hz.

Results

The procedure was performed for a total of 14 patients. For seven patients, data collection and evaluation failed due to a significant ventricular ectopy, the induction of ventricular fibrillation during dilatation, repositioning of the electrode between calibration and balloon inflation, or a highly unstable signal.

For seven balloon inflations, *left anterior descending* (LAD) proximal, LAD D1, *right circumflex* (RCX), and *right coronary artery* (RCA), appropriate data could be obtained. The age of the patients concerned was 53 ± 4 years (2 female, 5 male).

We compared the impedance curves recorded at rest and at the end of exercise as well as the curves just prior to balloon inflation, at the end of balloon inflation, and one minute after reperfusion.

RCA

The right vetricular wall is predominantly perfused by the RCA. With the electrode placed in the right ventricular apex, the impedance curves change due to exercise (Figure 1).

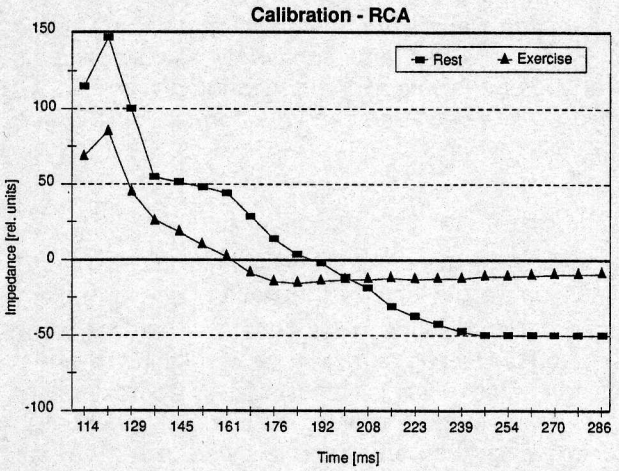

Figure 1. *The impedance curves for exercise of patient W. Z. rotate counterclockwise.*

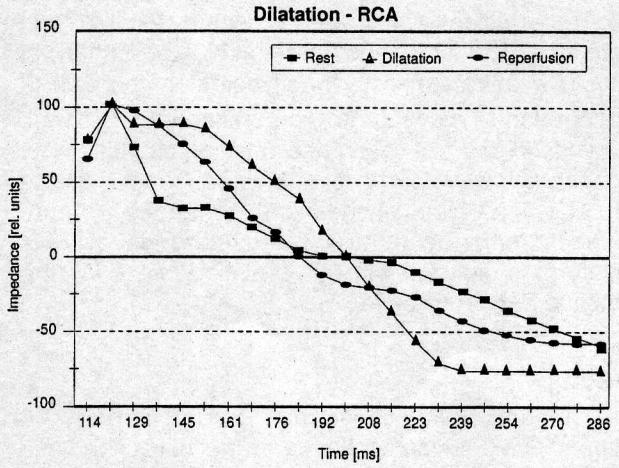

Figure 2. *Upon balloon inflation, the curves of patient W. Z. show a clockwise rotation. Upon reperfusion, the curves rotate toward their original direction.*

In patient W. Z., this change is characterized by a counterclockwise rotation of the impedance curves around a "pivot" point of 200 ms after the pacing stimulus.

During balloon inflation, the impedance curve morphology changes with a clockwise rotation. This change is opposite to that found during exercise, and indicates a decrease in myocardial contractility as a reflection of the ischemia caused by the balloon inflation (Figure 2).

Note that during reperfusion (1 minute after balloon deflation), the impedance curves rotate toward the resting condition.

In another patient, exercise also provoked a counterclockwise rotation of the impedance curves, but no significant change was seen during balloon inflation in the RCA. In this patient, the electrode position was documented to be closer to the septum than the RV apex. Since the septum is perfused by the LAD artery (which was not occluded in this case), the impedance curve did not show the characteristic change due to ischemia. Thus, depending upon the position of the electrode with reference to the ischemic or non-ischemic tissue, the dynamic nature of the impedance signal will be appropriately affected.

LAD and RCX

The LAD and RCX arteries both mainly supply the left heart. As a result for all patients with balloon inflation of the LAD or RCX, a paradoxical increase of the contractile state in the right heart was found.

An example is shown in Figures 3 and 4. Upon balloon inflation of the proximal LAD, a counterclockwise rotation of the impedance curve is effected which correlates to an increase in contractility. On reperfusion, the impedance curve changes toward the rest state.

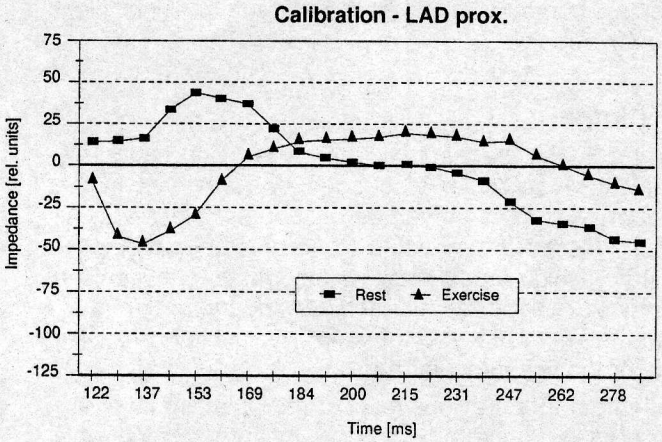

Figure 3. *The impedance curves for exercise of patient P. K. show a counterclockwise rotation that increases with the duration of the exercise.*

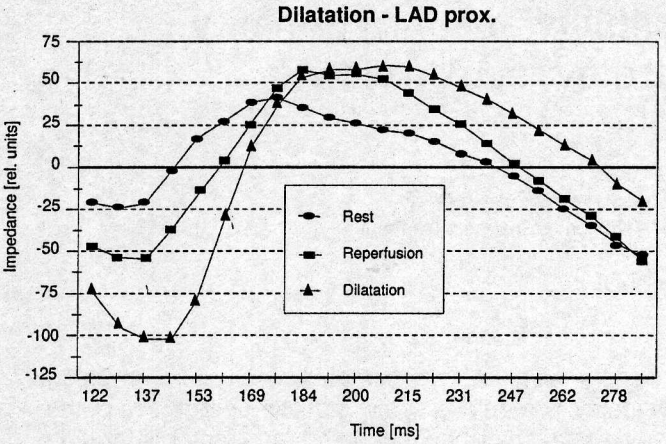

Figure 4. Upon balloon inflation, the curves of patient P. K. rotate clockwise. Upon reperfusion, the curves rotate toward their original direction.

Discussion

The slope of an impedance curve can be quantified by a regional effective slope quantity, RQ, that is the change of impedance (z) over an interval of 24 ms;

$$RQ = Z(t+24ms) - Z(t)$$

The value of t is chosen for each patient, specifically to achieve optimal dynamics, *i. e.*, RQ is evaluated for the interval where its change from rest to exercise is maximal.

Figure 5 shows the ratio of the RQ changes upon balloon inflation and exercise for all patients evaluated. Its value is positive if, upon balloon inflation, the impedance curve rotates in the direction of exercise, and negative if the curve rotates toward rest. The results confirm that the impedance waveforms measured upon balloon inflation of RCX or LAD indicate an increased contractility, and upon balloon inflation of RCA indicate decreased contractility.

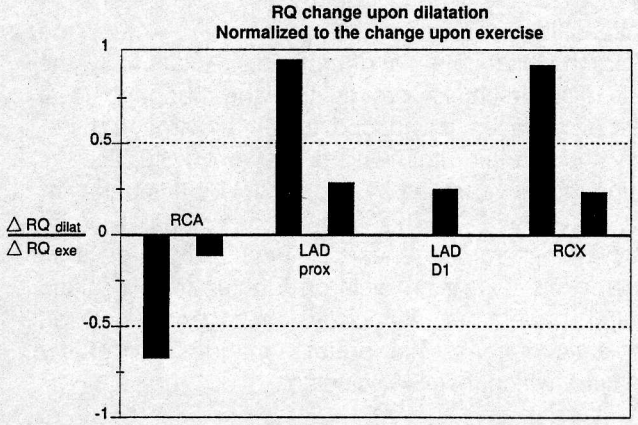

Figure 5. The ratio of the RQ changes upon dilatation and upon exercise for all patients evaluated.

The data collected, thus far, support the hypothesis that the intracardiac impedance curve reflects the contractile state of the myocardium in the close environment of the electrode tip. Statistics for the right heart are, however, poor. Additional data for right heart balloon inflations (RCA) are to be collected.

Myocardial contractility is controlled by the sympathetic nervous system and is a measure of the cardiac output required by the autonomic nervous system. In chronotropically incompetent patients, this is the only control system available to the patient to effect a change in the cardiac output. By using the intracardiac impedance, a pacemaker system measuring the RQ as a control parameter can provide rate-adaptive pacing and, therefore, reestablish closed-loop control of the cardiac output. The functionality of such a principle has already been established.[1,2]

Figure 6 summarizes the effect of local ischemia on the adaptive rate provided by such a closed-loop pacemaker system.

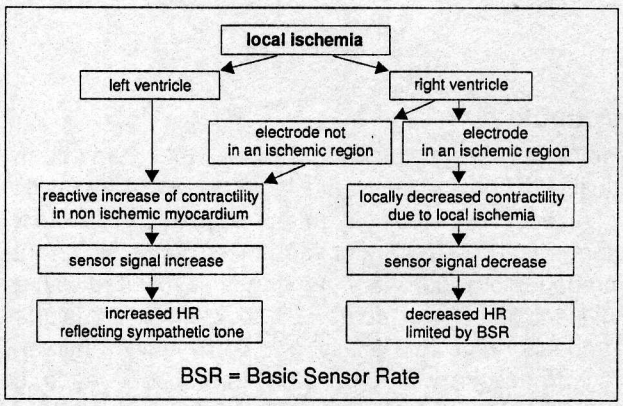

Figure 6. Effect of local myocardial ischemia on the ANS pacemaker rate.

References

[1] Pichlmaier AM, Braile D, Ebner E, Greco OT, Hutten H, von Knorre GH, Niederlag W, Rentsch W, Volkmann H, Weber D, Wunderlich E, Schaldach M. Autonomous nervous system controlled closed-loop cardiac pacing, PACE 15:1787-1791, 1992.

[2] Schaldach M, Hutten H. Intracardiac impedance to determine sympathetic activity in rate-responsive pacing, PACE 15:1779-1786, 1992.