

Recordings of the Monophasic Action Potential Using New Implantable Leads

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Summary

The monophasic action potential (MAP) was recorded with new fractally coated implantable electrodes from the epicardial surface over extended periods of up to 10 days. The MAP morphology and MAP derived parameters such as amplitude and duration are similar to MAP's recorded with traditional Ag-AgCl electrodes. MAP duration was documented during AAI stimulation, carotid sinus massage and antiarrhythmic therapy, and showed the expected inverse linear relationship with heart rate. In conclusion, fractal electrodes present a new tool allowing a more thorough investigation of proarrhythmic mechanisms and the evaluation of antiarrhythmic therapy.

Introduction

The inotropic and chronotropic status of the heart are critically dependent on the temporal relationship of the electrically active ion flux in and out of the myocardial cells. The monophasic action potential (MAP) is known to represent the cellular action potential of a small number of cells around the electrode tip. Clinically, the MAP has been used as both a diagnostic tool as well as for evaluating antiarrhythmic medication.^[1]

The limitation of the existing MAP recording technology using Ag-AgCl electrodes has been that only short term measurements were possible in vivo. As the electrode-tissue interface deteriorates MAP signals recorded endocardially during electrophysiological investigations or epicardially in patients undergoing cardiac surgery, become unrecordable after three hours at the maximum.^[2]

This study presents measurements of the MAP recorded with new fractally coated electrodes from both ventricular and atrial epicardial surfaces over a period up to 10 days following open heart surgery. Many prior publications have emphasised the diagnostic significance of the MAP, most notably for arrhythmia monitoring.^[2] In the first part of our study, we report on changes of the MAP during autonomic stimulation, changes in heart rate and the presence of arrhythmias and antiarrhythmic therapy. The focus of interest is placed on atrial recordings, as supraventricular arrhythmia are the most common complications observed in patients following heart surgery,^[3] especially valve replacements.

Material and Methods

In 8 patients (age 60.7 ± 15.0 years) undergoing open heart surgery (aortic valve replacement, Ross-OP), a new specially designed epicardial electrode was implanted similarly to the usual temporary pacing wires and placed mostly on the lateral aspect of the right atrium. The bipolar titanium electrode is fractally coated with iridium which markedly increases capacitance, thereby ensuring low polarization with a good frequency response^[4], characteristics otherwise only seen with Ag-AgCl electrode configuration.

The MAP signals were recorded with a high-impedance isolation amplifier, digitized with an analog-digital converter (12-bit resolution, sampling frequency 500-1000 Hz) and stored on a computer. A computer-based analysis of the MAP duration, amplitude, depolarization velocity and cycle length was then performed off-line.

During the in-patient stay (5 to 10 days postoperatively) the MAP was recorded daily along with the ventricular electrogram (from the temporary pacing wire), the mean arterial blood pressure (MABP) and the central venous pressure (CVP). Measurements were made during intrinsic sinus rhythm, overstimulation using temporary atrial pacing leads and carotid sinus massage. The dynamic changes in the morphology and time course of the MAP were also monitored during alterations in sympathetic tone and during the occurrence of arrhythmias and application of antiarrhythmic drugs.

Results

Figure 1 demonstrates that the morphology of the recorded MAP's is identical to that reported by other investigators using the Ag-AgCl electrode.^[2,5] The amplitude, duration and depolarisation velocity of the MAP's were initially of a similar quantity as those reported in the literature. Over the 5-10 day recording period the amplitude decreased but stabilized at around 60% of the starting values. Importantly, however, signal morphology and MAP derived parameters were stable over the entire recording period, as shown in Table 1, implying no deterioration in the electrode-tissue interface.

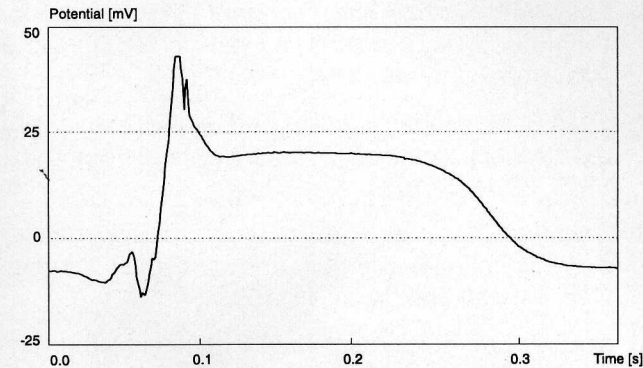


Figure 1: Ventricular MAP from the epicardial surface recorded with fractally coated electrode

electrode	Ag/AgCl	fractal (initial < 1h)	fractal (chronic > 5d)
amplitude atrial	<10 mV [3]	3-15 mV	2-10 mV
ventricular	10-50 mV [3]	15-45 mV	15-25 mV
duration (MAP _{d90})	83-362 ms [1]	175-380 ms	75-360 ms
depol. velocity	6.4 V/s [1]	5.9 ± 2.1 V/s	5.7 ± 1.9 V/s

Table 1: Comparison between Ag-AgCl and fractal electrode

MAP duration measured during intrinsic rhythm as well as during overpacing in an AAI-Mode with different rates, showed the well described inverse proportional linear relationship to the heart rate.^[6]

With the initial infusion of dopamine a relative lengthening of the atrial MAP was observed. After continuing i.v. infusion of dopamine the heart rate increased markedly and was associated with a shortening of the MAP.

This change in MAP duration is not solely heart rate dependent. Figure 2 shows the slope of the linear relationship between MAP duration and cycle length. The rise of the slope during the first 120 s indeed points to a relative lengthening of the MAP's with dopamine infusion.

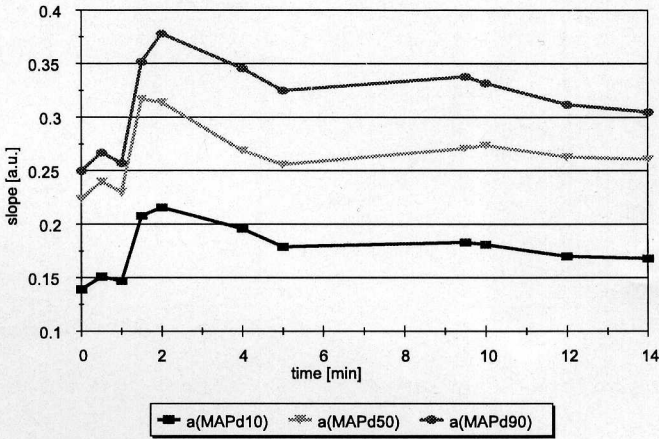


Figure 2: Slope of the inverse linear relationship between the MAP duration and the heart rate during dopamine i.v.-infusion

Carotid sinus massage caused a marked drop in the heart rate, but no significant change in MAP duration. This effect was generally more pronounced when the massage was performed on the right.

In the three cases where patients became arrhythmic, the morphology of the MAP's altered considerably. Figure 3a shows atrial MAP's during atrial flutter and ventricular MAP's during a bigeminal rhythm. During atrial flutter, the MAP duration shortened to less than 75 ms and the depolarization velocity decreased to 2.3 ± 0.4 V/s. The plateau phase (phase 2) is completely absent and in the depolarizations phase (phase 0) a biphasic deflection is observed.^[7] Following medication with sotalol the arrhythmia in this patient was terminated and the MAP's returned to the original morphology but with a relative lengthening (40 ± 5 ms). During the bigeminal rhythm, the ventricular MAP's show a reduced amplitude and a slower repolarization velocity (Figure 3b).

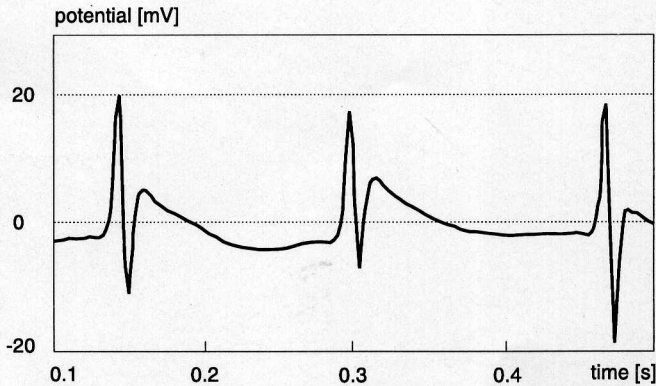


Figure 3a: MAP morphology during arrhythmia in atrium

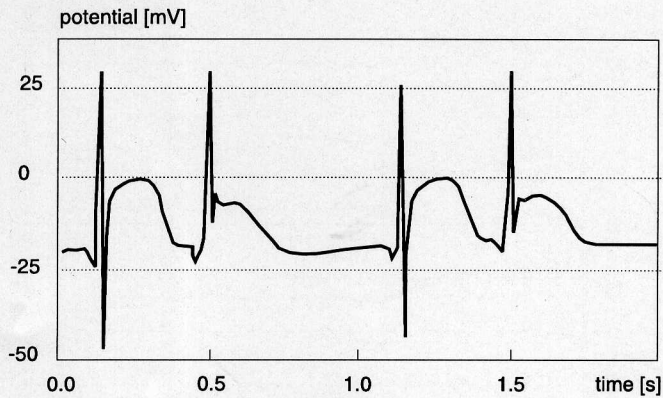


Figure 3b: MAP morphology during bigeminal arrhythmia in ventricle

Discussion

This study presents results using a new electrode technology and confirms the validity of the approach. In this context it can be confirmed that MAP's recorded with the fractally coated electrode follow the well documented changes associated with alterations in heart rate, parasympathetic and sympathetic stimulation as well as antiarrhythmic therapy. Thus, MAP duration was found to vary linearly with heart rate due to a more rapid ion turnover. Dopamine infusion lengthens the MAP corresponding to a greater net Ca^{2+} ion release into the cytoplasm. Atrial flutter leads to ischemic MAP's with a short repolarization corresponding to a rapid Ca^{2+} elimination after incomplete Ca^{2+} release due to less negative resting potentials. Similar changes are seen when ventricular bigeminy occurs in the successive MAP's. Carotid sinus massage, a primarily parasympathetic stimulation, produces little effect on the MAP duration and also a relative shortening of the MAP's. The lengthening of the MAP observed with sotalol is primarily a consequence of slowed ion exchanges and is also well described in literature.^[3]

In conclusion, the new fractal electrode technology provides a promising additional tool for the investigation of arrhythmic mechanisms in the heart. Additional aspects that may be addressed are monitoring of impending arrhythmia as assessed by changing MAP morphology (for use in antiarrhythmic devices such as implantable cardioverter defibrillators), evaluation of antiarrhythmic drugs and their efficacy, as well as post operative monitoring of the cardiac function as an adjunct to normal hemodynamic assessment.

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