Measuring Monophasic Action Potentials with Defibrillator Leads - A One Case Study

F. ZIMMERMANN, S. MUURLING, R. SIMON
Department of Cardiology, Christian-Albrechts-University of Kiel, Germany

R. BELKE, V. LANG, M. SCHALDACH
Department of Biomedical Engineering, University Erlangen-Nürnberg, Erlangen, Germany

Summary

Fractally coated electrodes have been used for recordings of monophasic action potentials (MAP) several times. This one case study aimed to proof that the fractally coated defibrillator lead SPS75 (BIOTRONIK, Germany) allows to record the MAP. During implantation of a cardioverter-defibrillator MAPs were successfully recorded in different positions in the apex of the right ventricle. The amplitude was 26.3 ± 0.9 mV with opposing the lead to the endocardium and 15.2 ± 0.15 mV without mandrine respectively. During spontaneous rhythm at 595 ± 3.2 ms cycle length a MAP duration of 258 ± 1.5 ms at 90 % repolarization was measured. Before T-wave shock induced ventricular fibrillation (VF) the MAP duration was 242 ± 1.5 ms at 567 ± 6.3 ms cycle length. Following the successful termination of the VF with a 15 J shock the spontaneous rhythm decreased (cycle length: 597 ± 4.8 ms) while the MAP duration shortened to 236 ± 2.2 ms showing a significant rate independent shortening of the MAP. During VF the morphology of the MAP altered notably showing fractionated signals without clear depolarization course and MAP-similar signals with depolarization course. Immediately after the shock the well-known MAP shape appeared again. This one case study proofed that the SPS 75 allows to record the MAP during spontaneous rhythm and ventricular fibrillation. The incorporation of a MAP signal in ICD classification algorithms could overcome inadequate shock therapies in patients with supraventricular tachycardias or T-wave oversensing and is therefore an oncoming diagnostic challenge.

Key Words

Monophasic action potential, implantable cardioverter-defibrillator, lead technology

Introduction

Recordings of the monophasic action potential (MAP) open new ways in experimental and clinical electrophysiology [1][2]. The MAP allows to monitor the influence of heart frequency, antiarrhythmic drugs, and ischemia on cellular electrophysiology. Nevertheless, MAP recordings were only possible during electrophysiological investigation, because the measurements of the MAP require special leads and electrode surfaces like silver-silverchloride [3]. Fractally coated pacemaker and defibrillator leads are now available, also allowing to monitor the monophasic action potential [4]. This paper presents first clinical data about recording the MAP using a commercially available fractally coated defibrillator lead.

Patient and Methods

The female patient G. J., aged 60 years, suffered from an acute posterolateral myocardial infarction in 1981. Subsequent cardiological examinations revealed furthermore a combined valvular aortic disease. In 1991 a Carpentier-Edwards-bioprosthesis was implanted in aortic position and a single bypass grafting of the RCA was performed due to a one vessel stenosis. In February 1997, the patient suddenly suffered breathlessness, dizziness, and a feeling of anxiety. Ventricular tachycardia was diagnosed and terminated by i.v. 50 mg of Ajmalin. A following electrocardiographic and enzymatic investigation excluded a myocardial reinfarction. Cardiac catheterization revealed a severely reduced left ventricular function and an obliteration

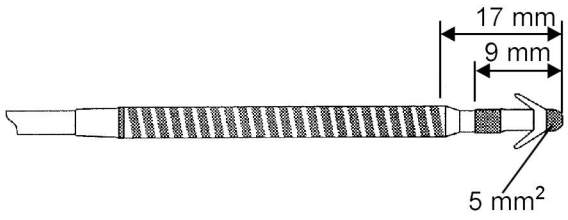

Figure 1. Ventricular defibrillation lead SPS75 (BIOTRONIK, Germany). The electrodes are coated with fractal Iridium.

of the RCA in the proximal segment. LAD and RCX showed only small arteriosclerotic impairments of the wall. The bypass to RCA was morphologically in a good condition. The investigation confirmed no significant aortic insufficiency or stenosis. In the afterwards performed electrophysiological study no sustained malign ventricular tachycardia was inducable.

The spontaneous event of ventricular tachycardia and the missing reproducibility of the arrhythmia on the one hand and the conservative therapeutic concept of the coronary disease in combination with the impaired left ventricular function on the other hand implies the indication for implantation of a cardioverter-defibrillator (ICD), which was performed in March 1997.

We implanted the ICD under the condition of local anasthesia in the catheter laboratory (EP lab). The ICD lead (SPS 75, BIOTRONIK, in figure 1) was introduced into the heart by puncture of the vena subclavia sinistra in the Seldinger-technique and placed in a right ventricular apico-septal position with a preshaped mandrine. Using usual chirugical preparation the subpectoral pocket for the device was shaped.

Intracardial signals were recorded directly after the first placement of the ICD lead in apico-septal position in the right ventricle. Subsequently, several repositionings were performed with continuos recording of the (MAP) and threshold testing. Insertion of a mandrine into the lead allowed to vary the pressure of the tip of the ICD lead exerted to the endocardium. The MAP signal was measured between tip and first ring of the ICD lead. The signal, which was available at the output of external threshold analyzer (ESA 400 in combination with ERA 300, BIOTRONIK), was magnified by the electrophysiological recording device (EP-Lab of Quinton, band pass filter 0.05 - 40 Hz) and forwarded via the analogue output to the A/D converter

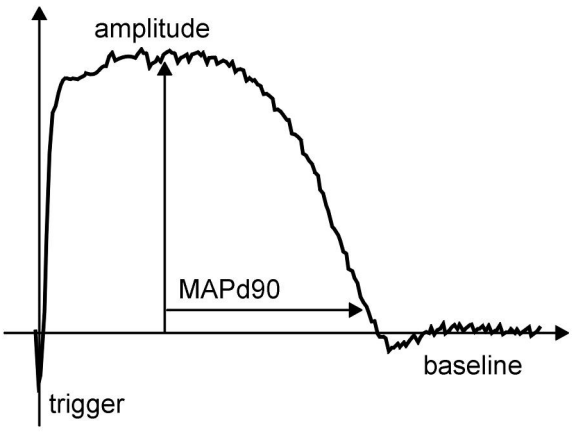

Figure 2. A semiautomatic software (BioView) evaluates the monophasic action potential and calculates the amplitude, MAP duration (MAPd90) and cycle length (distance between 2 consecutive triggerpoints) for each individual MAP.

(12 bit, 500 Hz sampling rate per channel). Semiautomatic MAP analysis yielded signal intervals, MAP duration (MAPd 90) and signal amplitude as shown in figure 2.

Results

The defibrillator lead SPS 75 allowed to record the monophasic action potential at all locations in the right ventricle after repositioning as depicted in figure 3. The amplitude of the MAP was higher, when the mandrine was inserted into the lead tip, indicating a firmer contact between lead tip and myocardium (see table 1). In some locations the MAP showed distortions in the repolarization course, which were avoided by repositioning the SPS 75-lead, bringing it into a more perpendicular position to the endocardium. The simultaneously performed threshold (1.0 V at 0.5 ms) and R-wave measurements (R-wave: 25 mV, slew rate: 3.2 V/s) indicate that a good sensing and pacing capability was achieved in those positions, in which an undistorted MAP measurement was possible.

Table 1 shows that during spontaneous rhythm at 595 ± 3.2 ms cycle length a MAP duration of 258 ± 1.5 ms at 90 % repolarization was measured. Before T-wave shock induced ventricular fibrillation (VF) the MAP duration was 242 ± 1.5 ms at 567 ± 6.3 ms cycle length. Following the successful termination of the VF with a 15 J shock the spontaneous rhythm decreased

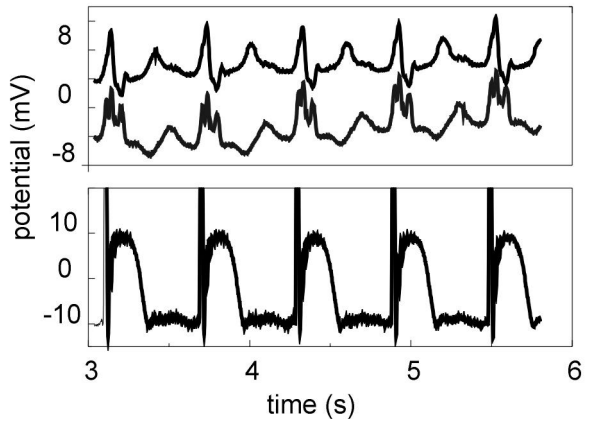

Figure 3. Simultaneous recordings of the monophasic action potential (MAP, lower row) and the surface ECG (upper row) after fixation of the defibrillation lead SPS75 (BIOTRONIK, Germany).

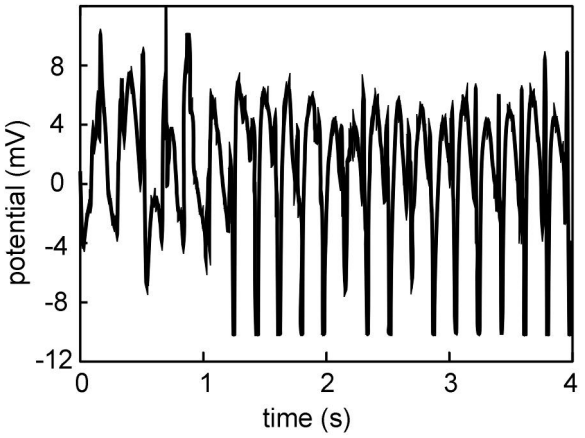

Figure 4. Recordings of the monophasic action potential during T-wave shock induced ventricular fibrillation.

(cycle length: 597 ± 4.8 ms) while the MAP duration shortened to 236 ± 2.2 ms showing a significant rate independent shortening of the MAP. Programmed stimulation (standardized ventricular stimulation protocol) did not alter the MAP duration or amplitude.
During VF the morphology of the MAP altered notably showing fractionated signals without clear depolarization course in the first second in figure 4. Without any interventions the signals altered and a MAP-similar signal with a typical depolarization course appeared in the last 3 seconds of figure 4. Immediately after the shock the well-known MAP shape appeared again.

Discussion

Our initial investigations show, that the ICD lead SPS 75 allows the measurement of excellent right ventricular MAP signals. Comparison of measurements with and without mandrine proofed that the signal amplitude is markedly influenced by the pressure of the tip to the endocardium. After removal of the mandrine an excellent signal morphology is preserved without a diminution in signal amplitude. Furthermore, intraoperative initiation of ventricular fibrillation and the subsequent application of an internal defibrillation shock does not result in remarkable changes in MAP signal quality or amplitude.

	Cycle length ± SD (ms)	MAPd90 ± SD (ms)	Amplitude ± SD (mV)
with mandrine	635 ± 8.0	276 ± 1.99	26.3 ± 0.9
without mandrine	636 + 3.7	256 + 1.8	15.7 + 0.75
after fixation	595 + 3.2	256 + 1.5	35.0 + 0.38
before S1-S2 stim.	584 ± 5.8	256 ± 2.2	35.0 ± 1.41
after S1-S2 stim.	587 ± 5.9	251 ± 5.3	34.0 ± 2.68
after 1st shock	567 ± 6.3	242 ± 1.5	32.3± 0.20
after 2nd shock	597 ± 4.8	236 ± 2.2	44.0 ± 2.75

Table 1. Mean MAP duration, cycle length and MAP amplitude with standard deviation of a 60 s recording intervals before and after repositioning and before and after a successful 15 J shock.

First investigations concerning long-term stable measurements of ventricular evoked potentials (VER) and monophasic action potentials were already performed successfully using DDD pacemakers (Physios CTM 01, Logos, BIOTRONIK) [6]. They showed only minor changes between the signals recorded directly after implantation and 12 months later.

The permanent and long-term stable possibility to measure an MAP signal via an implanted ICD lead opens new diagnostic and correlated therapeutic possibilities for the future. It should be, for example, possible to increase specificity of classification algorithms in ICD systems by the use of MAP signals or to reliably exclude T-wave oversensing.

In ICD therapy optimization focuses apart from more technical improvements (reduction in size of device, longevity, integration of antibradycardial components like VVIR/DDDR pacing) on increasing therapeutic specificity. Especially numerous inadequate shock applications due to paroxysmal supraventricular tachycardias or to T-wave oversensing have partly led to severe uncertainties in patients suffering these events and in their doctors. The incidence of inadequate therapies is currently estimated at 10 to 40 % [5][7-11].

The pathophysiological solution to overcome this diagnostic failure must either incorporate the atrial signal into the classification algorithms or must evaluate the ventricular signal in a totally new manner. Furthermore an early detection of changes in MAP signal morphology could be used as a trigger mechanism for ventricular pacing to avoid appearances of ventricular tachyarrhythmias. Whereas the incorporation of the atrial signal is already available in ICD systems with a DDD/DDDR pacemaker needing an additional atrial lead, analyses of the ventricular signal presenting itself as a MAP like potential due to the measurement with a fractally coated ICD lead is an oncoming challenge. Furthermore, the number of patients who need ICD therapy and additonal dual chamber pacing, which justifies a separate atrial lead, is estimated at not more than 5 to 10 % of all ICD indications.

References

[1] Franz MR. Bridging the gap between basic and clinical electrophysiology: What can be learned from monophasic action potential recordings. J of Cardiovasc Electrophysiol; 5(8) (1994): 699-710.

[2] Yuan S. Monophasic action potentials: Concepts to practical applications. J Cardiovasc Electrophysiol 5 (1984): 287-308.

[3] Franz MR. Method and theory of MAP recording. Progress in Cardivasc Diseases; XXXIII 6 (1991): 347-68.

[4] Bolz A. Die Bedeutung der Phasengrenze zwischen alloplastischen Festkörpern und biologischen Gewebe für die Elektrostimulation. Berlin, Fachverlag Schiele und Schön, 1995.

[5] Bardy G, Troutman C, Poole J, et al. Clinical experience with a tiered-therapy, multiprogrammable antiarrhythmia device. Circulation 85 (1992): 1689-1698.

[6] Dauer W, Fröhlich R, Müssig D, et al. High Resolution Monitoring of Intracardiac Signals with a DDD-Pacemaker. Prog Biomed Res Vol. 1, No. 4 (1996) 50-55.

[7] Fogoros RN, Elson J, Bonnet C. Actuarial incidence and pattern of occurence of shocks following implantation of the automatic implantable cardioverter defibrillator. Pacing clin Electrophysiol. 12 (1989) 1465-1473.

[8] Grimm W, Flores B, Marchlinski F. Electrocardiographically documented unnecessary, spontaneous shocks in 241 patients with implantable cardioverter defibrillators. Pacing clin Electrophysiol. 15 (1992) 1667-1673.

[9] Hook BG, Callans DJ, Kleinman R, et al. Implantable cardioverter-defibrillator therapy in the absence of significant symptoms. Rhythm diagnosis and management aided by stored electrogram analysis. Circulation 87 (1993) 1897-1906.

[10] Trappe H, Pfitzner P. Häufigkeit von inadäquaten Therapien. In Wietholt D, Ulbricht LJ, Gülker H. Implantierbare Kardioverter-Defibrillatoren. Georg Thieme, Stuttgart, New York, 1997. 137-146.

[11] Troup P, Nisam S. Complications associated with automatic implantable cardioverter defibrillator. In Alt E, Klein H, Griffin JC. The Implantable Cardioverter Defibrillator. Springer, Berlin, 1992 (253-271).