Intracardiac Impedance and Tiered Therapy for Ventricular Arrhythmias

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Summary

The addition of a hemodynamic sensor to current ICD arrhythmia detection would help to direct more appropriate therapy for ventricular arrhythmias. This study analyzed change in unipolar intracardiac impedance during sinus rhythm, right ventricular pacing and ventricular arrhythmias induced at electrophysiological study. There was a significant correlation between the change in amplitude of the unipolar intracardiac impedance waveform and the change in mean systolic blood pressure at the onset of ventricular arrhythmias. The results at arrhythmia onset were corroborated by an additional analysis of the correlation between the area under the impedance curve and the pulse pressure during separate episodes of sinus rhythm, right ventricular pacing and ventricular tachycardia.

Keywords

Ventricular tachycardia, impedance, hemodynamic sensor, ICD

Introduction

Implantable cardioverter defibrillators (ICDs) are effective therapy for the treatment of ventricular tachyarrhythmias [1,2]. Their functioning requires not only that the device is capable of administering appropriate therapy but that the device also properly identifies the presence of a clinically significant arrhythmia. The modes of therapy most frequently used are antitachycardia pacing, low energy cardioversion and high energy defibrillation. The use of rate criteria to detect ventricular arrhythmias has consistently been found to offer a high level of sensitivity and continues to be the gold standard by which all other arrhythmia detection schemes are judged [3]. Although in general the occurrence of syncope during ventricular tachycardia induced at programmed electrical study is inversely related to arrhythmia cycle length and the degree of hypotension that ensues, the threshold cycle length at which clinical symptoms develop is not clearcut [4]. Patients with ventricular tachycardias of equivalent cycle length may experience significantly different symptoms [4,5]. Whilst many ventricular tachyarrhythmias require prompt high energy defibrillation, a substantial proportion of ventricular tachycardias are hemodynamically well tolerated. An ideal algorithm, based on cycle length detection, but incorporating an index of the hemodynamic status of the patient might enable one or more attempts of anti tachycardia pacing during a hemodynamically stable monomorphic ventricular tachycardia. If unsuccessful or if the arrhythmia became hemodynamically unstable, this might be followed by low energy cardioversion and ultimately high energy defibrillation.

Tiered therapy for ventricular tachyarrhythmias offers many advantages. Firstly, patient tolerance of ICD therapy would be dramatically improved by the avoidance of multiple high energy defibrillations that are inevitably painful and frightening. Secondly, device longevity may be prolonged by the avoidance of high energy defibrillations and thirdly the proarrhythmic potential of device therapy is minimized and rapidly corrected if hemodynamic compromise is detected.

Hemodynamic studies have shown that, at the onset of ventricular tachyarrhythmias, both right and left ventricular systolic function is compromised, in particular

myocardial contractility and relaxation are adversely affected [6,7]. Changes in unipolar intracardiac impedance reflects changes in ventricular wall tension and thickness and hence the contractility of the myocardium [8]. An assessment of myocardial contractility can be made by measuring intracardiac impedance using an INOS² DR pacemaker (Biotronik, Germany). We have assessed continuous changes in unipolar intracardiac impedance during ventricular arrhythmias to assess their hemodynamic stability.

Methods

Study Population

The study involved 8 patients, 6 men and 2 women, aged 53 to 77 years (mean 66 ± 8), undergoing diagnostic cardiac electrophysiological testing. The study group consisted of patients with a history of sustained ventricular tachycardia, ventricular fibrillation or unexplained syncope. Additional patients underwent electrophysiological assessment, but were excluded because ventricular tachyarrhythmias were not induced. Written informed consent concerning the research portion of the study was obtained prior to each procedure.

Experimental preparation

All studies were performed with the patient in the supine position. Under local anesthesia, an intracardiac quadripolar electrode catheter was introduced through the right femoral vein. The catheter was fluoroscopically positioned as close as possible to the right ventricular apex. The catheter was used for electrical stimulation/recording and right ventricular intracardiac unipolar impedance measurement. A 4F catheter was inserted into the right femoral artery to monitor the arterial pressure in all patients. Programmed electrical stimulation was performed using a digital stimulator (Biotronik Universal Heart Stimulator 20) that delivered a 0.5 ms constant current square wave. A modified Wellens protocol was used to induce ventricular arrhythmias. A surface patch electrode was placed between the patient's scapulae (surface area 187 cm²). The unipolar impedance signal was measured between the distal electrode of the quadripolar catheter and the external patch electrode. An external INOS² DR pacemaker was used to measure impedance, which in turn was continuously monitored by a modified pacing system analyzer, called a Unilyzer. Ventricular arrhythmias induced were categorized into hemodynamically stable or unstable, and defined the latter if they resulted in presyncope, syncope or a systolic blood pressure of less than 90 mmHg.

Data Collection

Data derived from the INOS pacemaker was recorded digitally onto a PC card. The surface ECG, intracardiac ECG and intra-arterial blood pressure data were recorded simultaneously by a Omnibook XE2 laptop computer (Hewlett Packard, USA). Specialized custom built software packages, 'Bioview' and 'Cardiolyzer' (both Biotronik) were used to synchronize and analyze the impedance, blood pressure and ECG data off-line.

Statistical Analysis

The peak systolic blood pressure value and peak to trough amplitude of the impedance waveform were averaged from ten successive sinus beats prior to the onset of ventricular tachycardia. Corresponding values over the first ten beats of ventricular tachycardia were averaged and the values were normalized with respect to those calculated during sinus rhythm. The impedance and blood pressure signals at arrhythmia onset were carefully synchronized to allow beat to beat analysis. In addition, in five of the patients, episodes of sinus rhythm, high frequency pacing (burst) and ventricular tachycardia were analyzed to evaluate the relationship between the area under the impedance curve (Za) and the differences between systolic and diastolic blood pressure (peak to peak blood pressure amplitude, BPpp). Up to nine episodes were selected from each recording. The episodes of ventricular tachycardia in this additional analysis were not specifically taken at arrhythmia onset. To compute the area under the curve a peak detection algorithm was used to detect the minima and maxima of the signals. The minima served as upper and lower limits for the calculation of Za (Figure 1). Again, the resulting impedance and blood pressure values were normalized with respect to the individual's maximum to allow inter individual comparison of the resulting data. Linear regression analysis was used for correlation between the change in impedance and blood pressure values.

Results

Figures 2 and 3 show typical recordings of the unipolar impedance waveform and the corresponding arteri-

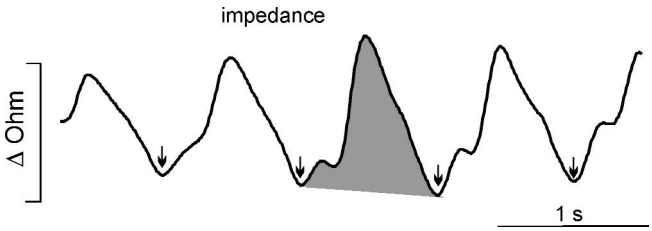

Figure 1. Calculation of the area under the Z curves using minima and maxima as boundaries.

al pressure tracing during sinus rhythm, right ventricular pacing and at the onset of ventricular arrhythmia. During sinus rhythm, the impedance waveform has a uniform, stable morphology. Each sinus beat is associated with a single cycle of the impedance waveform. At the onset of ventricular arrhythmia, both the impedance waveform and the arterial pressure tracing lose their stable morphology and drop substantially in amplitude. During monomorphic ventricular tachycardia, the impedance waveform maintains its cyclical appearance coincident with the cycle length of the arrhythmia allowing beat to beat analysis. Figure 4 summarizes the results obtained from the 8 patients. We found a linear correlation with a correlation coefficient of 0.74 between the change in mean systolic blood pressure and the resulting change in impedance amplitude at arrhythmia onset. In general, hemodynamically unstable ventricular tachycardia resulted in a drop in mean systolic blood pressure to 37 % of the value found in sinus rhythm. The corresponding drop in mean impedance amplitude was 41 % of the sinus rhythm value. Table 1 shows the characteristics of the ventricular arrhythmias induced and the clinical status of the patient following the onset of the arrhythmia.

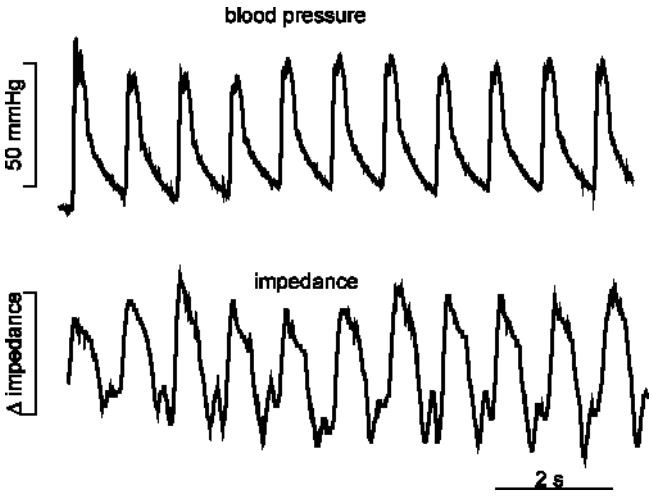

Figure 2. Intra-arterial blood pressure tracing (top panel) and unipolar intracardiac impedance waveform (bottom panel) during sinus rhythm. Impedance is measured in arbitrary units.

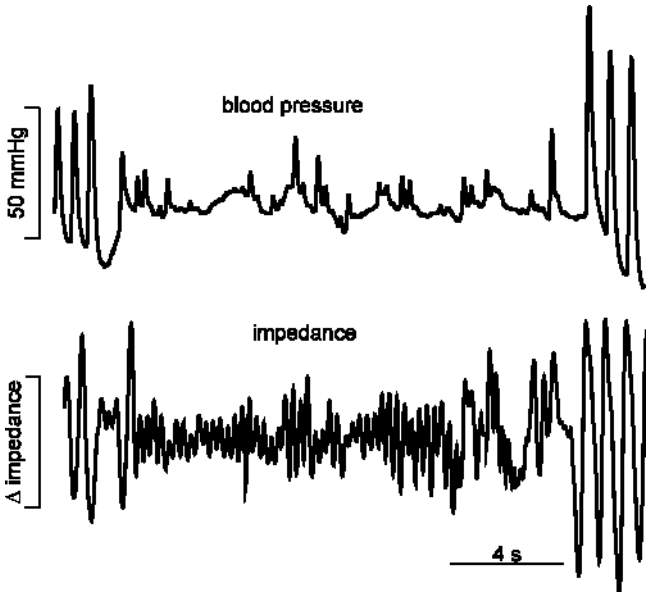

Figure 3. Intra-arterial blood pressure tracing (top panel) and unipolar intracardiac impedance waveform (bottom panel) at onset and the entire course of a ventricular tachycardia.

The individual normalized data sets comparing Za and BPpp are shown in Figure 5. In sinus rhythm, BPpp

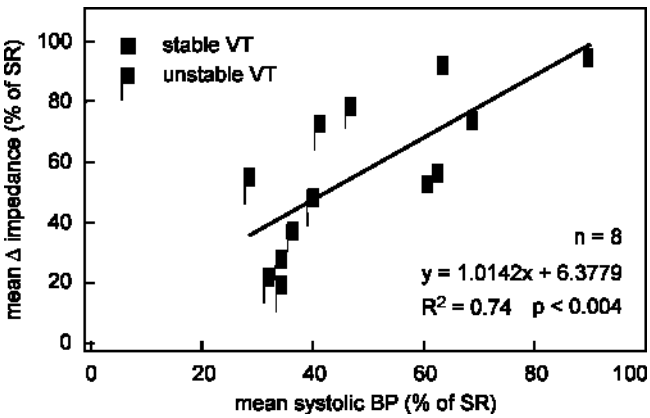

Figure 4. Correlation between the mean impedance amplitude and the mean systolic BP at arrhythmia onset.

Case	Age (yr & sex)	LVEF %	VT cycle length (ms)	Stable / unstable	VT episodes
1	72 F	61	315 + 440	U + S	2
2	60 M	20	200	U	1
3	66 M	48	280	U	1
4	75 M	17	330 + 440 + 440	U + S + S	3
5	64 F	60	330	U	1
6	60 M	46	PMVT + PMVT	U + U	2
7	77 M	45	260	U	1
8	53 M	29	380 + 380	S + S	2
Mean ± sd		66 ± 8	41 ± 17		

Table 1. Clinical characteristics of study patients and arrhythmias induced. F = Female; LVEF = Left Ventricular Ejection Fraction; PMVT = Polymorphic Ventricular Tachycardia; S = Stable; U = Unstable

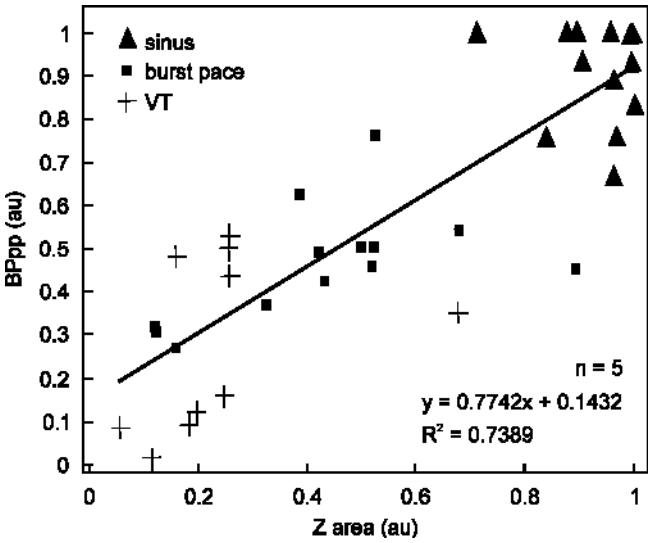

Figure 5. Correlation between the difference of diastolic to systolic blood pressure (BPpp) and the area under the impedance curve (Za).

was highest and showed a pronounced drop during periods of right ventricular pacing and tachycardia. The impedance data resembled the course of the BPpp. Za was highest during sinus rhythm and showed a decrease during pacing and during tachycardia. Figure 5 summarizes the results of all five patients. As can be depicted from the figure, we found a good linear correlation between BPpp and Za.

Discussion

Changes in the intracardiac impedance signal reflect changes in the hemodynamic state. A beat to beat analysis at the onset of ventricular arrhythmia shows a good correlation between the change in amplitude of the impedance signal and the change in peak systolic blood pressure. The calculated correlation coefficient of 0.74 demonstrates the valid association between the change in blood pressure and change in impedance amplitude at arrhythmia onset. The area under the curve of the impedance signal also falls during periods of ventricular pacing and ventricular tachycardia demonstrating an impressive correlation with the associated drop in the pulse pressure.

Because the conductive area of the distal pole of the quadripolar catheter is substantially smaller than that of the surface patch electrode, the current density is concentrated around the catheter tip. Thus, with unipolar impedance measurement, the resultant changes in impedance values occur because of localized changes in myocardial contractility. In addition, during the course of arrhythmia induction, minor displacement of the quadripolar catheter may arise. These factors may explain some of the overlap of impedance values between ventricular arrhythmias classified as hemodynamically stable or unstable in Figure 4. Whilst overall myocardial contractility may be compromised during arrhythmia, localized, well preserved areas of function may exist giving rise to high impedance signals of greater magnitude than would be expected. The converse situation may also be true of course. Despite the potential detriment of using the unipolar impedance electrode configuration, the results are encouraging and worthy of further investigation. The threshold of using a blood pressure of 90 mmHg to classify arrhythmias as stable or unstable was used in an attempt to make this study more clinically relevant. Symptoms of presyncope or syncope immediately classified the arrhythmia as unstable regardless of actual blood pressure value. It was felt that a drop in systolic blood pressure to 90 mmHg in the supine position would invariably translate to presyncopal symptoms in the upright state. Asymptomatic patients with blood pressures greater than 90 mmHg at arrhythmia onset may have

been symptomatic if standing. Further studies to investigate the changes in the impedance signal during tilt are planned.

The results from our study are commensurate to those found by Khoury et al [9]. Their group investigated relative volumetric changes in the right ventricle during ventricular and supraventricular arrhythmias by measuring the stroke impedance. Impedance was measured between electrodes two and four on a quadripolar catheter within the right ventricular cavity. A good correlation between changes in stroke impedance and mean arterial pressure during arrhythmias was found ($r = 0.84$). Measurement of unipolar intracardiac impedance using the Biotronik INOS pacemaker was technically easy. Results obtained using our method of impedance measurement might be more easily translated into a clinically relevant device with minimal extra computing requirements.

Conclusion

An association between the unipolar intracardiac impedance signal and the hemodynamic status of the patient has been demonstrated. Incorporating this sensor into an ICD would be technically simple. Such a device might enhance the implementation of tiered therapy for ventricular tachyarrhythmias.

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