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Electrical And Hemodynamic Evaluation Of Ventricular And Supraventricular Tachycardias With An Implantable Dual-Chamber Pacemaker

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Abstract

The discrimination between ventricular (VT) and supraventricular tachycardia (SVT) and the evaluation of their hemodynamic impact are essential issues in the arrhythmia management. A new pacing device features a tachycardia diagnostic system relying on simultaneous recording of the transvalvular impedance (TVI) and a special integrated electric signal derived by the whole set of endocardial electrodes (iECG). The iECG waveform is sensitive to the pattern of ventricular activation, similarly to the surface ECG. The TVI increases in systole and decreases in diastole and the amplitude of this cyclic fluctuation is an expression of the effectiveness of the pump function. In order to test the value of these signals in the analysis of a tachycardia, we have assessed the iECG and TVI modifications induced by different SVTs and tolerated and non-tolerated VTs, during electrophysiological (EP) studies.

In case of SVT, the ventricular component of the iECG maintained the same morphology as in sinus rhythm. The peak-peak amplitude of the TVI fluctuation was reduced to 66 ± 11 % of the individual sinus rhythm reference, but the signal was present at every beat and showed a remarkable stability (variation coefficient 0.19 ± 0.01). In case of VT, the ventricular component of the iECG was strikingly different than in sinus rhythm. Regular TVI fluctuation was observed with tolerated VTs (peak-peak amplitude 74 ± 6 %; variation coefficient 0.21 ± 0.04). In contrast, with non-tolerated VTs the TVI amplitude was depressed below 40%, and the signal was virtually absent in the event of very fast VT or VF.

Our results confirm that the iECG is a reliable tool to quickly discriminate VTs from SVTs and that TVI can provide information on the severity of the hemodynamic impairment produced by a tachycardia, with potential clinical benefit in the follow-up of pacemaker patients. Furthermore, the application of these signals to automatic algorithms of arrhythmia recognition might improve the specificity of therapy administration by an implantable defibrillator (ICD).

Introduction

Implantable devices for cardiac stimulation, originally designed to ensure appropriate electrical therapies for rhythm disorders, have progressively become in addition a valuable source of information on the heart activity in daily life. Pacemakers and ICDs, which continuously monitor the cardiac rate, can provide report on the rate trend and frequency distribution, as well as on the occurrence of tachyarrhythmia episodes. The electrograms recorded by the device are stored to document the arrhythmia and allow the physician to check whether the right treatment was administered, properly choosing between a defibrillation shock, antitachycardia pacing (ATP), or just passive surveillance. Thanks to the increasing availability of remote monitoring systems, the relevant information can be transmitted to the center responsible for the patient's follow-up in the very short term.

At present, implantable devices are considered fully reliable in tachycardia detection, as a sensitivity close to 100% has been reported in most studies. However, the specificity of fast ventricular tachycardia (FVT) discrimination with respect to supraventricular tachycardia (SVT) is still far from perfect.[1-4] This is regarded as the main reason for the relatively high incidence of inappropriate shock administration by ICDs reported in the clinical setting. In spite of technical improvement, 12 to 29% of shocks delivered by single-chamber ICDs are ascribed to SVTs misclassified for VTs.[5] Arrhythmia discriminators available in single-chamber devices include rate, cycle length stability, sudden onset and electrogram morphology, which is expected to change in case of a VT. Dual-chamber devices can consider in addition the relationship between atrial and ventricular events to distinguish VT and SVT. However,

Disclosures:
None

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the usefulness of dual-chamber algorithms in the prevention of inappropriate SVT treatment has not definitely been confirmed.[3,6,7]

The electrogram morphology could be crucial to arrhythmia classification. Different systems relies on different recording vectors, ranging from near-field bipolar electrograms to far-field potentials derived between a defibrillation coil and the device case.[8-11] Very recently, a novel approach has been proposed, which is based on the integration of the electric signals detected by the electrodes available in a dual-chamber pacemaker. The resulting waveform reflects both atrial and ventricular activity in a single tracing, similarly to a surface ECG lead, and is therefore referred to as intracardiac ECG (iECG). The ventricular component of the iECG is strongly influenced by the ventricular activation pattern, so that ventricular ectopic beats can be easily distinguished. On these bases, the iECG might be applied as a sensitive tool in the morphological discrimination of VT and SVT.

Another essential issue in the evaluation of a tachycardia is the severity of the associated hemodynamic impairment, which is a direct expression of the risk run by the patient. An ideal ICD should be capable to continuously monitor the pump function, delaying the shock delivery as long as reliable blood outflow is detected. This way, ATP could safely be continued, trying different stimulation protocols even in the presence of FVT.[11] Conversely, a cardiac rate slower than the FVT lower limit could be poorly tolerated in patients affected by cardiomyopathies or coronary disease, who might require therapy administration even if the rate criteria are not met.

Valuable information on ventricular mechanical activity can be gained by cardiac impedance recording, which can be performed by a stimulation device by means of the same electrodes involved in the pacing and sensing function. In particular, the impedance recorded in transvalvular configuration between right atrium and ventricle (TVI) allows precise beat-by-beat ejection surveillance, as the systolic reduction in ventricular volume entails a clear-cut TVI increase and the following diastolic filling results in gradual TVI decrease back to the baseline level.[12-14] The peak-peak amplitude of cyclic TVI fluctuation reflects the stroke volume.[15] and is correlated with the velocity-time integral of pulmonary outflow.[16] In the absence of ejection, the TVI fluctuation is abolished.[17] Therefore, TVI can be proposed as an indicator of the hemodynamic implications of a tachycardia, relying on standard electrocatheters.

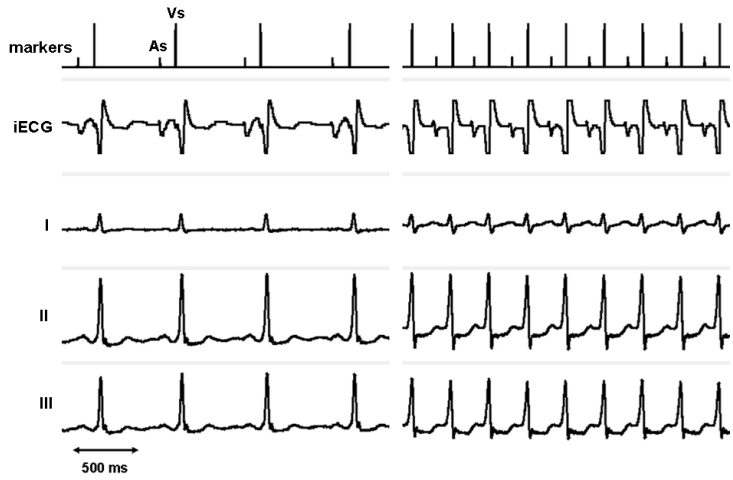

Figure 1: Sinus rhythm (left-hand panel) and orthodromic AVRT (right) in the same patient. From top to bottom tracing: pacemaker event markers (As = atrial sensing; Vs = ventricular sensing), iECG, surface ECG lead I, II and III. Full description in the main text.

Matched TVI and iECG recording can be performed by a dual-chamber pacemaker recently approved for clinical use. We have studied the acute changes in these signals induced by the transition from sinus rhythm to different forms of STVs and tolerated or non-tolerated VTs.

Material And Methods

During routine EP studies aimed at tachyarrhythmia induction and ablation, TVI and iECG were detected by an Eos DR pacemaker (Medico, Padova, Italy) used as an external recorder (ODO pacing mode setting). The device was connected through a specific patient cable with the proximal electrode pair of tetrapolar endocardial temporary leads positioned in high right atrium and right ventricular apex, while the distal dipole of each lead was used for cardiac stimulation with a separate pulse generator and electrogram detection by the polygraph of the EP lab. The polygraph also stored the 12 lead surface ECG and, only in patients tested for VT, the blood pressure recorded in the radial artery. The pacemaker derived conventional intracavitary bipolar electrograms together with TVI (scaled in Ohm) and the iECG, which was scaled in arbitrary units (bits) using the same gain in all recordings to allow relative comparison. Atrial an ventricular event markers and TVI and iECG tracings were transmitted by real-time telemetry to a Master 1000 pacemaker programmer (Medico, Padova, Italy), which acquired in addition 4 leads of the surface ECG (usually I, II, III and aVF). All signals were simultaneously displayed on the programmer screen, storing in memory the last 1 minute recording. Whenever required, the acquisition could be frozen and the content of the buffer memory transferred to the programmer hard disk, in the form of a progressively numbered file.

At the end of the procedure, the files were downloaded to a PC to be analyzed off-line with commercially available software (Acqknowledge 4.1, Biopac Systems Inc.). Data analysis was focused on the amplitude of TVI fluctuation and the comparison of iECG waveform in sinus rhythm and tachycardia. The TVI signal amplitude was expressed by either the excursion from telediastole to end-systole (considering the rising phase only and excluding the decay from the analysis), or by the peak-to-peak amplitude throughout a whole cardiac cycle. In case of a rate exceeding 200 bpm, the peak-peak amplitude of TVI fluctuation was assessed within consecutive time intervals of 400 ms, independent of the electric cycle. A minimum of 50 measurements were performed for each tachyarrhythmia episode and normalized to the mean TVI amplitude in sinus rhythm. Episode mean, standard deviation and variation coefficient of the relative TVI amplitude were calculated and further averaged in each arrhythmia subgroup (AVRT, AVNRT, tolerated VT and non-tolerated VT). A VT was considered as non-tolerated when associated with loss of consciousness or symptomatic hypotension (arterial blood pressure < 80 mmHg).

The pattern of the iECG atrial or ventricular waveforms (iP; iQRS) was classified as monophasic if a positive or negative peak accounted for more than 90% of the waveform peak-peak amplitude. In the opposite case, negative-positive and positive-negative biphasic patterns were distinguished. The second parameter to be evaluated in waveform comparison was the duration of the iQRS, which was deemed as unchanged if the difference between tachycardia and sinus rhythm did not exceed 30 ms. Possible modifications in iQRS peak-peak amplitude were considered as non-specific if occurring alone

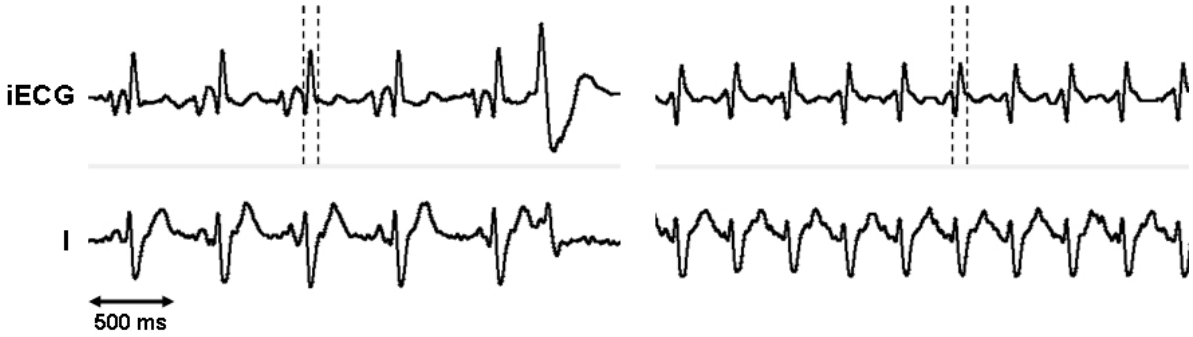

Figure 2: Left-hand panel: sinus rhythm with a premature ventricular contraction (last beat). Right-hand panel: AVNRT of slow-fast type, in the same patient. iECG (upper tracing) and surface ECG lead I (lower); the hatched vertical lines indicate the limits of the iQRS interval.

and therefore did not represent a tachyarrhythmia discriminating feature. Special care was taken to separate the iQRS waveform from antegrade or retrograde atrial signals which could be detected during or very close to the ventricular complex.

Recordings were performed in 32 patients. Isoproterenol infusion was applied to maximize the test sensitivity, thus a mild drug-induced tachycardia was present even in sinus rhythm. The study was negative in 11 cases, where a tachyarrhythmia was not triggered with standard stimulation protocols. SVTs were induced in 16 patients, including 7 cases of atrioventricular nodal reentrant tachycardia (AVNRT; all of slow-fast type), 5 of atrioventricular reentrant tachycardia (AVRT; all with orthodromic conduction), 3 of atrial fibrillation (AF) and 1 of atrial flutter. Ventricular tachycardia was present in 4 patients with 10 different QRS morphologies. One patient developed both AF and episodes of non-sustained VT. The 21 patients of either sex who developed a sustained arrhythmia (longer than 30 s or requiring prompt cardioversion/defibrillation for severe hemodynamic impairment) were included in the study. Written informed consent was obtained before the procedure from all the patients in whom TVI and iECG recording was performed. The study protocol was approved by the Ethical Committee of the San Filippo Neri Hospital.

Results

Patients' Clinical Characteristics

Mean patients' age was 52 ± 15 years. The 4 patients with inducible sustained VT showed structural heart disease (ischemic cardiomyopathy was present in 3 and non-ischemic cardiomyopathy in 1). Two patients with AF and one patient with atrial flutter had hypertensive cardiomyopathy. All the other patients with SVT featured no structural disease. The mean LVEF was 53 ± 21 % in the entire cohort.

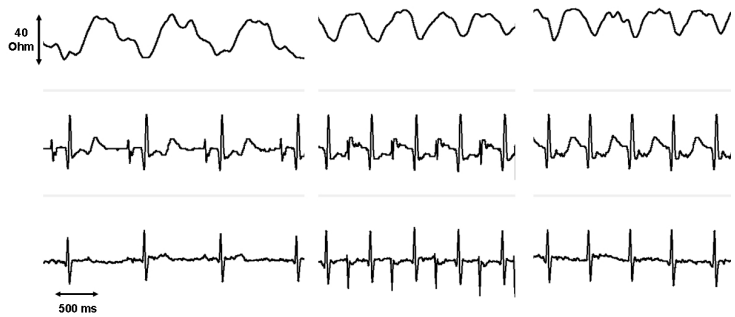

Figure 3: Sinus rhythm (left-hand panel), atrial pacing with 500 ms interval (middle) and AVRT (right hand; cycle length 480 ms). From top to bottom tracings: TVI (peak-to-peak amplitude 30.3 ± 2.9, 21.1 ± 3.0 and 20.4 ± 2.8 Ohm, respectively, from left to right panels), iECG, surface ECG lead III. AV conduction through the His-Purkinje system entailed a constant iECG pattern. In such condition, the TVI reduction was entirely explained by the rate increase.

AV and AV Nodal Reentrant Tachycardia

SVTs due to reentry along an accessory pathway (orthodromic AVRT) featured an essentially unchanged iQRS complex with respect to sinus rhythm. However, in some cases, the basal iQRS started with a deflection which disappeared during tachycardia, strongly suggesting a correspondence with ventricular pre-excitation. The retrograde P-wave was generally visible on the iECG and exhibited a different morphology with respect to the atrial signal detected in sinus rhythm. A representative example is shown in Fig. 1. Either in sinus rhythm or tachycardia, the iQRS pattern was narrow and biphasic (negative-positive), indicating that the arrhythmia was likely a SVT. Consistently, a modified iP waveform was recorded in correspondence with the atrial sensing markers in all tachycardia cycles. The EP study definitely demonstrated an orthodromic AVRT with a left lateral accessory pathway.

In case of SVT produced by AV nodal reentry of slow-fast type, the retrograde iP wave usually occurred before the end of the iQRS complex. This resulted in partial overlap of the two signals with apparent iQRS modification. However, the atrial and ventricular components could still be identified and discriminated. Provided that the part of the waveform which might contain a retrograde iP was excluded from the analysis, a full correspondence between the iQRS pattern and time-course in sinus rhythm and tachycardia was always confirmed. Only the signal portion trailing the pure iQRS was different in slow-fast AVNRT, as a result of retrograde conduction. In contrast, the iQRS waveform was radically changed in the event of an ectopic ventricular beat (Fig. 2).

During AVRT, featuring an average cardiac rate of 162 ± 43 bpm, the peak-peak amplitude of the TVI waveform was reduced to 83 ± 22 % of the reference value measured in sinus rhythm in each patient. During AVNRT, with average rate of 184 ± 15 bpm, the relative peak-peak TVI amplitude decreased to 73 ± 13 % of the individual reference. These changes were mainly due to an increase in the minimum diastolic TVI, while the systolic maximum peak was less affected. Similar effects on TVI were also induced by atrial overpacing at the same rate reached in tachycardia (Fig. 3). Regular and stable TVI fluctuation was detected in all cardiac cycles with both arrhythmias. As a result, the variation coefficient of TVI peak-peak amplitude averaged 0.13 ± 0.04 and 0.18 ± 0.01 during AVRT and AVNRT, respectively.

Atrial Fibrillation

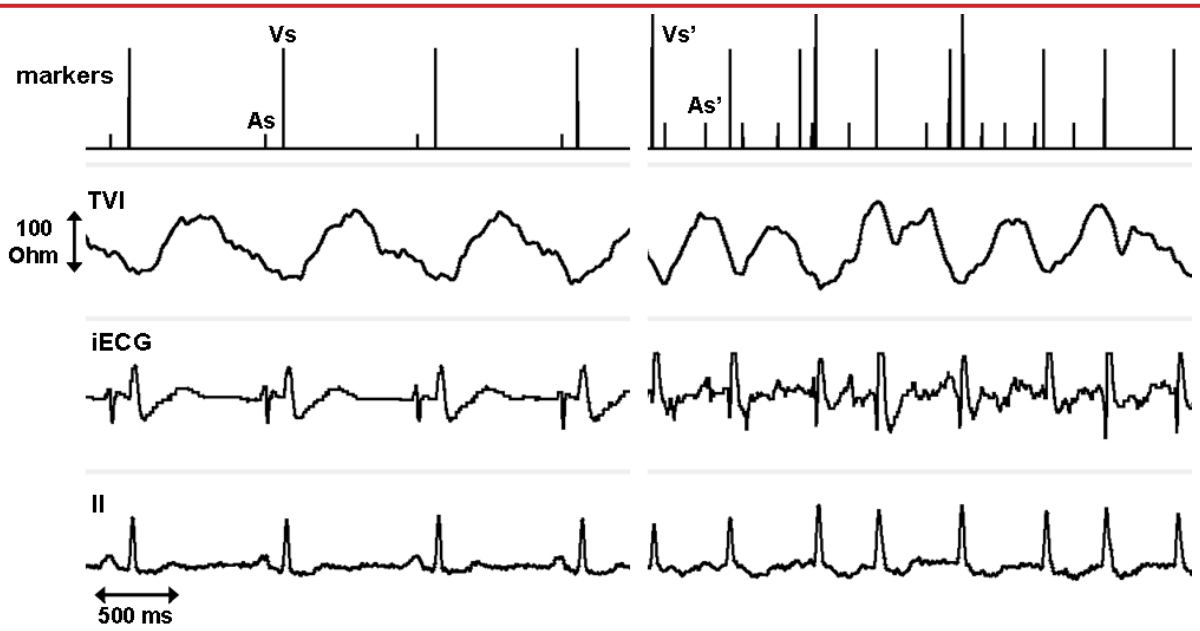

Figure 4: Sinus rhythm (left-hand panel) and AF (right). From top to bottom tracing: event markers (As = atrial sensing; Vs = ventricular sensing; As' = atrial sensing in the pacemaker refractory period; Vs' = ventricular sensing in the pacemaker refractory period) TVI, iECG, and surface ECG lead II. The arrhythmia increased the apparent "noise" of the iECG and the variability of TVI fluctuation.

In the presence of AF with AV conduction along the His-Purkinje system, most of the iQRS signals were narrow and similar to the sinus rhythm waveform. In contrast, no signal corresponded to the P-wave as characterized in sinus rhythm. Disorganized electrical activity was detected instead and made the tracing look noisy (Fig. 4). Such heterogeneous input to the iECG channel might occasionally result in iQRS fusion with signals of atrial origin, with apparent widening and morphologic modification of the ventricular complex. The presence of a polymorphic waveform on the iECG associated with the ventricular sensing markers (with iQRS resembling the sinus rhythm signal in the majority of the cycles), and the increased variability in R-R interval and apparent iQRS duration (with minimum values equal to the iQRS length measured in sinus rhythm), were all AF indicators.

TVI fluctuation was well preserved, though the increase from diastole to end-systole showed higher variability than with reentrant SVTs, due to the irregular R-R interval and related sudden changes in ventricular filling time (Fig. 4). Indeed, the amplitude of TVI systolic increase measured at every beat was correlated with the length of the previous cardiac cycle (Fig 5).

Ventricular Tachycardia

While the iQRS observed on SVT resembled the reference waveform recorded in sinus rhythm, the iECG was deeply modified in the event of a VT (Fig. 6). The iQRS was wider and showed an altered morphology in all cycles. The P-wave was not visible, unless retrograde conduction occurred.

Hemodynamically tolerated VTs were characterized by reduced but stable TVI fluctuation (Fig. 6). In the presence of an average cardiac rate of 154 ± 36 bpm, the TVI peak-peak amplitude decreased to 74 ± 6 % of the individual reference in sinus rhythm, with variation coefficient of 0.21 ± 0.04. In contrast, when the arrhythmia entailed a dangerous impairment of the pump function, clinically evidenced by fainting, symptomatic hypotension, or lack of arterial pressure pulses demonstrated by invasive monitoring, TVI fluctuation was unstable and strongly depressed. In the 10 s preceding the release of a defibrillation shock, non-tolerated VTs with cycle length ≥ 300 ms could still show small TVI waveforms, with average and median normalized amplitude never exceeding 40%. In case of VF or fast VTs with cycle length < 300 ms, the TVI signal was virtually abolished. The absence of TVI fluctuation indicated a heavy impairment of the pump function with resulting arterial pressure drop, as shown in Fig. 7. Effective defibrillation simultaneously restored the electrical rhythm, the arterial pulse pressure and the cyclic TVI excursion.

Discussion

Tachycardia evaluation should begin with ascertaining the nature of the arrhythmia (whether ventricular or supraventricular) and its hemodynamic implications. The acute tests described in the present report confirm that the new diagnostic system now available in an

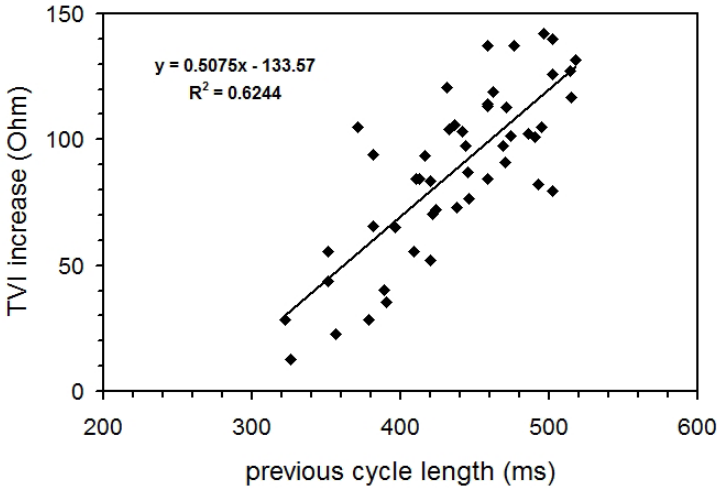

Figure 5: Linear correlation between R-R interval in the previous cycle and amplitude of TVI excursion from end-diastole to end-systole, in the AF episode illustrated in Fig. 4. The cardiac rate ranged from 116 to 186 bpm, averaging 139 ± 18.

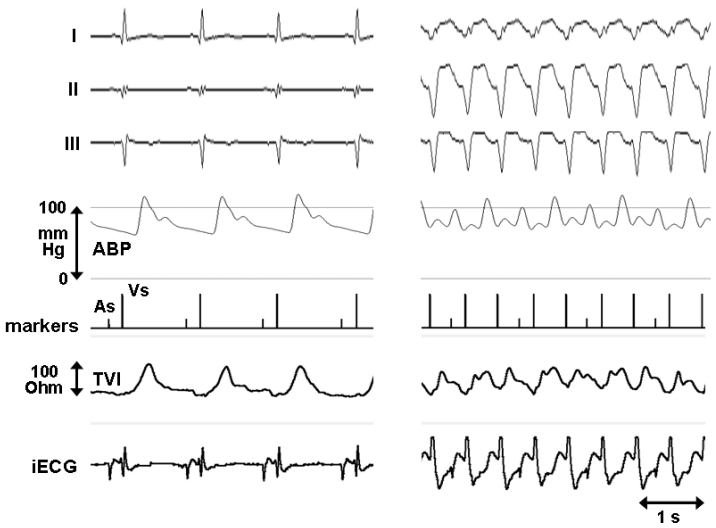

Figure 6: Sinus rhythm (left-hand panel) and an episode of tolerated VT with rate below 120 bpm (right). From top to bottom tracing: surface ECG lead I, II, III, blood pressure in the radial artery (ABP), event markers (As = atrial sensing; Vs = ventricular sensing), TVI and iECG. Both iECG and event markers demonstrate 2:1 retroconduction during the VT.

implantable pacing device could help address these questions in the clinical setting.

The system relies on simultaneous recording of iECG and TVI. The former is an integrated electric signal derived by the electrodes normally present in a dual-chamber stimulator; the latter is the electric impedance measured between right atrium and ventricle throughout a cardiac cycle. As the ventricular component of the iECG is affected by the pattern of ventricular activation, the same iQRS morphology is expected in sinus rhythm and SVT as well, while iQRS modification and widening should characterize a VT. Our experience supports this concept, although some caution was required in the interpretation of apparent changes in the iQRS waveform, which could actually result from the fusion of atrial and ventricular signals. This occurred with AF and especially with AVNRT, where the same altered morphology was present in all cardiac cycles. A trained observer can easily recognize the overlap of the two components in the iECG tracing; nevertheless, this task could result more challenging for an automatic algorithm of arrhythmia classification based on the correspondence with the sinus rhythm template. Aberrant conduction is another possible source of error, as it would result in iQRS alteration even in the presence of SVT. The absence of a stable relationship between atrial and ventricular sensing events might support the VT diagnosis, but it would remain very difficult discriminating a SVT with aberrant antegrade conduction from a VT with 1:1 retrograde conduction. In such cases, it could be helpful to check whether the patient is liable to aberrant conduction by incremental atrial pacing. Provided that aberrant conduction or antidromic AVRT (which is supposed to be a rare finding) can be excluded, striking changes in the iQRS morphology were always noticed in case of a VT, making quite easy the arrhythmia discrimination from any type of SVT.

During either SVTs or tolerated VTs, stable TVI fluctuation with average amplitude larger than 50% of the sinus rhythm waveform was detected. In contrast, the average TVI peak-peak amplitude never exceeded 40% in non-tolerated tachyarrhythmias with relative slow rate (cycle length ≥ 300 ms), and the signal vanished during faster non-tolerated VTs. These results are consistent with previous studies, which demonstrated that TVI can reflect the hemodynamic performance and the reliability of the pump function.[12-19] Other impedance recording systems, based upon unipolar or transventricular electrode configuration, have been tested for the capacity to discriminate stable from unstable arrhythmias.[20-21] Although the amplitude of the impedance excursion was significantly lower with unstable than stable VTs and a correlation between stroke impedance and arterial pulse pressure was reported, the range of signal reduction for the two conditions was partially superimposed, so that a cut-off value suitable to distinguish one from the other could not be determined.

The first application of the paired TVI-iECG system is purely diagnostic and is intended to supply the physician with advanced

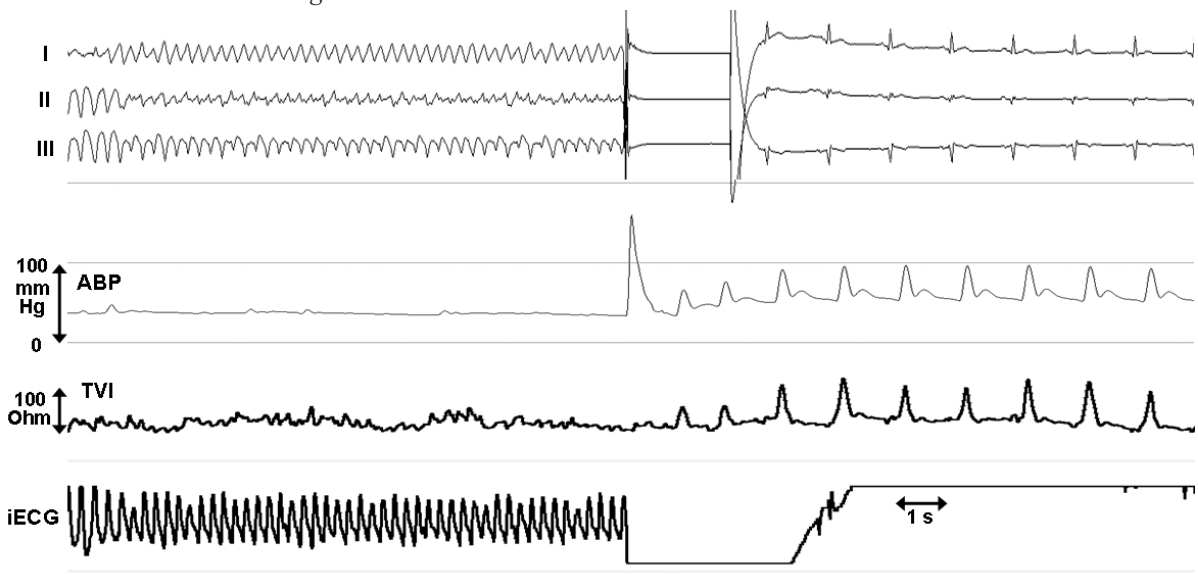

Figure 7: Another VT episode in the same patient as in Fig. 6, reaching a higher cardiac rate (above 200 bpm). From top to bottom tracing: surface ECG lead I, II, III, blood pressure in the radial artery (ABP), TVI and iECG. During the VT, the pump function was deeply depressed and the TVI fluctuation was absent. A defibrillation shock restored the sinus rhythm and the arterial pressure pulses, with the associated TVI excursion.

information on the tachyarrhythmia episodes recorded by the implanted pacemaker in daily life. The evaluation of the iECG tracing would allow precise discrimination between VT and SVT, and the analysis of TVI changes with respect to sinus rhythm can give insight into the hemodynamic effects of the arrhythmia. Both TVI and iECG recordings performed in sinus rhythm and tachycardia can be downloaded at the follow-up check, and are transmitted by remote monitoring after each high-rate episode to timely ensure the appropriate medical care, especially in the event of AF, which might require prompt antithrombotic treatment. Further studies are needed to establish whether these signals are also suitable to drive automatic algorithms of arrhythmia discrimination, which might be implemented in ICDs to regulate the administration of the electrical therapy. A reliable recognition of tolerated VTs based upon hemodynamic sensing, instead of rate assessment alone, could substantially reduce the incidence of shock release, improving the patient's quality of life as well as the effectiveness of the therapy.

Conclusion:

The combined evaluation of TVI and iECG can provide valuable information on the nature of a tachycardia and the associated hemodynamic risk, with potential benefits in the follow-up of pacemaker patients.

Limitations

The reported results have been obtained using temporary leads designed for EP studies. It cannot be excluded that TVI or iECG response to arrhythmia could be influenced by the lead properties and might be different in permanent implants, though the main features of both signals are independent of the recording tools in basal conditions.

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