Clinical impact of surface electrocardiography of cardiac arrhythmias in pacemaker-ICD patients

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ABSTRACT

Cardiologists often are called to explain electrocardiograms of pacemaker/implantable cardioverter (PMK/ICD) patients during arrhythmic events. The most frequent arrhythmia is atrial fibrillation (AF) whether in PMK or in ICD patients. Generally, it is not difficult to diagnose, it can affect the quality of life of this subgroup because it can generate inappropriate and painful therapies. Arhythmic as atrioventricular block or other bradyarrhythmias can show a particular way of presentation especially for the device's intervention using specific algorithms that cardiologists should know to adequately interpret the phenomenon. For example, Rate Drop Response algorithm (to prevent syncose) or other antiarrhythmic functions for atrial tachyarrhythmias (Post Mode Switching Overdrive Pacing (POMP) or Atrial Rate Stabilization) can alter surface electrocardiogram after AF or atrial ectopic beats. Ventricular arrhythmias in ICD patients are frequent. Burst, ramp and shock are therapies with a high percentage of efficacy. However, sometimes, supraventricular arrhythmias (SVT) can induce inappropriate interventions. In other cases appropriate burst, ramp or shock (during a ventricular tachycardia (VT)) can degenerate it in a fast ventricular tachycardia or in ventricular fibrillation with consequent shocks. Wavelet, onset, stability, and other algorithms, specifically in dual chamber ICDs, are used to discriminate SVT from VT assuring more specific interventions. (Anadolu Kardiyol Derg 2007: 7 Suppl 1; 60-3)

Key words: pacemaker, implantable cardioverter defibrillator, arrhythmia, electrocardiography

Introduction

The number of patients with pacemaker/implantable cardioverter (PMK/ICD) is increasing and frequently cardiologists are called to decide about some arrhythmic events happened in this subgroup of subjects. We have also to consider that often, previous arrhythmias justified the implant of these devices so we can expect a lot of electrocardiograms (ECG) to interpret with accuracy.

ECG analysis in pacemaker patients

Regarding bradyarrhythmias the diagnosis of these and therapies of the device are not so difficult to analyze (sino-atrial blocks, atrioventricular (AV) blocks, etc.). Of major interest for the clinician appears the ability to understand some modalities to treat arrhythmic events by the PMK/ICD that could make someone thinking to a malfunction (1-4). For example there are PMKs that can activate some algorithms for the treatment of neuromediated cardioinhibitory syncope (for example the function RDR - Rate Drop Response) or to look for the spontaneous AV conduction in order to decrease the percentage of right ventricular pacing (Medtronic - MVP Minimal Ventricular Pacing). In the former example (Fig. 1) the integrated diagnostic system of PMKs is able to recognize a rapid decrease of heart rate during a vasovagal reflex. A specific planning allows to detect a drop in heart rate so that the device will start to stimulate at 110-120 bpm to avoid the syncopal phenomenon. In the latter the PMK senses the pathological rhythm, rapid ventricular back up pacing to avoid symptomatic pauses and if the AV conduction is not restored the PMK switches in DDD/R modality. Periodically conduction tests are performed to verify if AV conduction is restored and in this case the PMK automatically turns back to AAI/R modality. During this mode switching we could see on the ECG some phases of AV block, and so we could think of a malfunction. The analysis of the beat following the blocked atrial beat, that is stimulated with a short AV interval, allows us to give a correct interpretation of the phenomenon (Fig. 2). Search AV is another algorithm to minimize right ventricular pacing. It automatically analyzes conduction sequences and gradually increases the paced and sensed AV delays until intrinsic ventricular activation is uncovered. A programmable maximum offset allows the titration of search limits.

Automatic mode switching (Fig. 3) is the ability of the pacemaker to automatically change from one mode to another in response to an inappropriately rapid atrial rhythm. In the DDD or DDDR pacing modes, if a supraventricular tachyarrhythmia occurs and the PMK senses the pathological rhythm, rapid ventricular pacing can occur. Mode switching avoids this limitation by switching from DDD or DDDR during sinus rhythm to a nontracking mode, such as DDI or DDR, during the pathological atrial rhythm.

Sometimes PMKs could generate themselves arrhythmias. For example in patient with AAI/R PMK previously implanted for sick sinus syndrome we can observe AV delays when the atrial rate is increased by rate responsive function. This may be due to a mismatch between the actual heart rate and the autonomic nervous tone (overstimulation) and may lead to a pacemaker syndrome (Fig. 4).
4). Sometimes we can observe an atrial retrograde conduction (PMK VVI) that generates a PMK syndrome. This phenomenon can also bring to atrial arrhythmias (atrial flutter or atrial fibrillation (AF)). Arrhythmias on surface ECG can be induced by undersensing, oversensing or failure to capture (Fig. 5, 6) (5).

Another interesting example could happen when we have an atrial stimulus sensed by the ventricle. In this case, we get a failure to pace with consequent asystolic pause (cross talk) if there is not a spontaneous ventricular depolarization (Fig. 7).

Sometimes, and it is very important especially in PMK dependent patients, we can see on ECG a ventricular spike within 110 msec of an atrial stimulus. This algorithm (ventricular safety pacing) avoids an asystolic pause in case of ventricular oversensing. In case of appropriate sensing the stimulus, falling during the absolute ventricular refractory period, will not give a ventricular depolarization (Fig. 8).

**ECG analysis in ICD patients**

The ECG analysis can lead ourselves to the diagnosis of ventricular tachyarrhythmias based on the morphology of wide QRS tachycardia, but not with certainty. Obviously, this is a relevant problem in ICD patients. In single chamber ICD it could be difficult to discriminate a supraventricular (SVT) from ventricular tachyarrhythmias so the device can use some algorithms that analyze R wave morphology and the onset of the tachycardia.

- **Wavelet** discriminates ventricular tachycardia (VT) from SVT with rapid conduction
- **Onset** discriminates VT from sinus tachycardia
- **Stability** discriminates atrial fibrillation with rapid conduction from VT

**Wavelet algorithm**

It is based on the principle that rhythms of ventricular origin such as ventricular fibrillation (VF) and VT have normally QRS morphology different from rhythms of supraventricular origin.

Wavelet is an electrogram morphology algorithm with simple on-off programming (when it is programmed on, ICD inhibits the identification if a sufficient number of QRS during tachycardia are similar to those of stored sample) (6, 7). It automatically compares the electrogram (EGM) morphology during high rate rhythms to a stored template of normal (baseline) rhythm. Wavelet delivers fewer inappropriate therapies for SVT (compared to EGM width) and has been shown to increase specificity without decreasing sensitivity. Wavelet adapts to changes in electrogram morphology and updates it automatically to adapt to changes in the morphology of normal beats.

**Onset algorithm**

Onset permits to prevent identification of a sinus tachycardia instead of VT examining the acceleration of ventricular rate. If ventricular rate gradually increases, for instance during sinus tachycardia, the device inhibits the counting of VT events. If the ventricular rate rapidly increases, so as during a true tachyarrhythmias, then the device activates counting of VT events.

**Stability algorithm**

It permits to discriminate atrial fibrillation with a rapid conduction from VT because during AF the ventricular rate is unstable, in
fact there is a high variability of RR interval. An RR interval is considered unstable if the difference between its value and one of three previous intervals is greater than the programmed stability interval.

For dual-chamber and biventricular devices utilization of other algorithms (Medtronic - PR Logic; Guidant- Rhythm ID; St'Jude Medical - A-V Rate Branch; Biotronik - Smart Detection) makes possible to discriminate VT from SVT (8, 9).

These algorithms logically integrate rate detection with information about conduction patterns, regularity, and AV dissociation. They are designed to maintain high sensitivity for ventricular tachyarrhythmias while improving discrimination for SVTs. Distinction criteria inhibit the inappropriate ventricular recognition during SVT with rapid conduction. The device analyzes activation patterns and temporization in both chambers analyzing P wave and R wave sequences and rates. These data allows to detect AF, atrial flutter, sinus tachycardia and other SVT with 1:1 conduction. When testing shows presence of one of these rhythms, recognition is inhibited.

**Arrhythmias in PMK/ICD patients**

Atrial fibrillation is the most frequent arrhythmia in PMK/ICD patients. The diagnosis from surface ECG generally is not difficult, with rare exceptions. It is of interest, somewhat, analysis of some specific functions of pacemakers that should be known to correctly explain ECG patterns. These algorithms have been introduced whether to prevent AF (Atrial Preference Pacing, Atrial Rate Stabilization, Post Mode Switching Overdrive Pacing (PMOP)) or to interrupt it (ATP - AntiTachycardic Pacing - burst, ramp, shock). For example, PMOP function is aimed to decrease the incidence of premature relapse of AF. This algorithm assures an atrial stimulation at high rate (i.e. 100 bpm) for several minutes, when sinus rhythm is restored after episodes of AF, that is when for mode switch function the PMK stimulates in DDD/R modality from DDI/R. Atrial Rate Stabilization function (Fig. 9) allows the PMK to stimulate after a premature ectopic atrial beat to prevent potentially proarrhythmic long pauses. It gradually extends stimulation interval until a normal and organized intrinsic rhythm could emerge or to the achievement of the lower rate. Atrial Preference Pacing is a function that supports a continuous atrial stimulation instead of a spontaneous atrial rhythm within a programmable maximum heart rate to prevent supraventricular tachyarrhythmias inhibiting the dispersion of atrial refractory period.

The interruption therapies for atrial and ventricular tachyarrhythmias are burst, ramp and shock. Burst stimulation consists of supplying of a determined number of sequences in AOO (for atrial tachyarrhythmia - Fig.10) modality or VOO modality (for ventricular tachy). During ramp stimulation (Fig. 11) the device supplies impulses with intervals gradually shortening with a programmable decrement. The first impulse of each sequence attends during the tachycardia at a programmable percentage of its duration. Antitachycardia pacing is not painful.

Shocks can be synchronized (for AF or VT) or not (for VF) and with different energy. They are painful.

In ICD patients, AF often causes inappropriate interventions of device when ventricular rate is so fast that falls in the range of ventricular tachyarrhythmias. In this case, we should modify pharmacological therapy or device programming to obtain beneficial effects.

Surface ECG sometimes can show AF onset mechanism. For example after nonsustained atrial tachycardia on paced rhythm (Fig.12).

An example of interest could be the possibility (however rare) that an appropriate intervention for a VT as a shock can turn VT in

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![Figure 4. Tachycardia in AAI/R pacemaker with long atrioventricular interval](image1)

![Figure 5. AAI pacemaker. Sensing and pacing defect](image2)

![Figure 6. Oversensing in VVI pacemaker](image3)

![Figure 7. Crosstalk](image4)

![Figure 8. Ventricular Safety Pacing](image5)

![Figure 9. Atrial rate stabilization. A premature atrial complex (PAC) followed by atrial stimulation at high frequency](image6)
VF. In this case, the device will deliver another shock to terminate the arrhythmia (Fig. 13).

Finally, we remind pacemaker mediated tachycardia (PMT) (Fig.14). Pacemaker-mediated tachycardia is a form of reentrant tachycardia that can occur in patients who have a dual-chamber pacemaker. In this setting, the pacemaker forms the anterograde (atrium to ventricle [A→V]) limb of the circuit and the atrioventricular (AV) node is the retrograde limb (ventricle to atrium [V→A]) of the circuit. The AV node conducts the ventricular-paced beat or a premature ventricular contraction (PVC) back (ie, retrograde) to the atrium and depolarizes the atrium before the next atrial-paced beat, the impulse can then trigger the pacemaker to repeatedly pace the ventricle, thereby generating a reentrant arrhythmia circuit known as PMT or endless-loop tachycardia. Hence, the pacemaker forms the antegrade limb of the circuit, and the AV node is the retrograde limb. Another common situation that causes PMT is failure to capture the atrium with a paced beat, which means the atrium then is amenable to depolarization by the impulse conducted retrograde from the ventricle. The tachycardia continues until retrograde conduction is lost or the atrium becomes refractory. Note that as many as one third of patients with antegrade complete AV block have intact retrograde conduction, which may be intermittent. Depending on the retrograde conduction time, the PMT rate may be at or below the programmed upper rate limit of the pacemaker. This form of pacemaker behavior only can occur in a dual-chamber system. We can stop PMT setting a magnet upon the device. It inactivates the sensing circuit and determines a dual chambers asynchronous stimulation. Measures to prevent this phenomenon are PVARP extension, AV interval shortening and maximum heart rate modification. Present PMK and ICDs have some protection algorithms to prevent PMT that usually extend PVARP after premature ventricular beats or after a sequence stimulated ventricle - sensed atrium at high rate with fixed VA interval.

References

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