

Pacemaker Function Assessed by External Holter ECG Recording: A High Rate of Mild Malfunction

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Summary

Basic pacemaker functions, including pacing capture and sensing, are regularly checked by trained technicians on an outpatient basis. In this study, an additional assessment of pacemaker function was performed in 169 patients using ambulatory 24h Holter ECG recordings. Forty-one patients with mild symptoms, such as palpitations or dizziness, and no established pacemaker malfunction formed the SYMPT group (DDD: $n = 5$; AAI: $n = 2$, VVI: $n = 34$). Severe symptoms - syncope or obvious pacemaker malfunction - were excluded. The ASYMP group was composed of 128 patients who did not exhibit symptoms (DDD: $n = 7$; AAI: $n = 5$; VVI: $n = 116$). Short periods of loss of capture were observed in 8 out of 41 SYMPT patients (i.e., 20 %, $P = 0.0001$ versus ASYMP group). Asymptomatic oversensing was present in 12 SYMPT patients (28 %) and in 28 ASYMP patients (22 %, $P = ns$). In two patients, oversensing was related to the "thunder" contacts in bipolar screw-in leads. In all other cases, it concerned myopotential oversensing in unipolar leads. The pauses ranged from 1.0 to 3.3 seconds, with a median value of 1.3 s. Undersensing occurred in 20 leads in the SYMPT group (i.e., 43 %), which was mainly related to arrhythmia, and in 21 leads from the ASYMP group (16 %, $P = 0.0001$). Undersensing occurred more frequently in the atrial lead: 10/19 (53 %), than in the ventricular lead: 33/162 (20 %, $P = 0.005$). Normal pacemaker function was seen in 10 SYMPT patients (24 %) and in 82 ASYMP patients (64 %, $P = 0.001$). In conclusion, Holter ECG recordings revealed a high rate of mild pacemaker malfunction, especially in SYMPT patients. The observed malfunction consisted mostly of myopotential oversensing in unipolar leads and of undersensing in patients with arrhythmia. 24h Holter ECG recording proved to be a valuable tool for evaluation of pacemaker troubleshooting. The high incidence of mild pacemaker malfunction should be taken into account in the design, selection, and set-up of future pacemakers, with the preference for bipolar sensing configuration and high sensitivities as well as optimal characteristics of the sensing amplifiers.

Key Words

Pacing capture, malsensing, ambulatory ECG, pacemaker follow-up, troubleshooting

Introduction

Implanted pacemaker systems are routinely checked at pacemaker clinics by a trained technician supervised by a cardiologist. Regular pacemaker checks usually focus on the functional integrity of the implanted pacing system and on the evaluation of the underlying heart rhythm, either sinus rhythm or arrhythmias. The increased complexity of modern pacemakers pose problems for the physician and technician charged with implanting the pacemaker. This is especially true when the patient's symptoms are intermittent or pacemaker

malfunction occurs only under specific conditions that are not easy to provoke in a clinical setting [1-4].

We consider ambulatory 24h Holter ECG recording a valuable and easy-to-use supplemental tool for the evaluation of pacemaker function. In the present study, we focused on the evaluation of basic pacemaker functions, including pacing capture and sensing. Over years 1994 - 1997, a selected series of patients was monitored using Holter ECG and a number of indicative cases are described in this summary report.

Materials and Methods

The study included 169 patients, of whom 41 reported mild symptoms of palpitations, dizziness, or discomfort. These patients formed the symptomatic group (SYMPT), where five patients had a DDD pacemaker, two had an AAI pacemaker, and 34 had a VVI pacemaker. The ASYMP group included 128 patients, seven with a DDD pacemaker, five with an AAI pacemaker, and 116 with a VVI pacemaker. The Holter ECG recordings in the ASYMP group were performed to assess, among other things, sensing performance of the pacemakers. Patients with severe symptoms such as syncope, dizziness with secondary trauma, or obvious pacemaker malfunction were excluded from the study and admitted directly to the hospital for separate analysis and treatment. Patients with a VDD/R system were also excluded from the study.

The cardiologist saw the patients on a routine basis, while the technician evaluated the ECG recordings for sensing and capture. As a minimum, the stimulation threshold, sensing threshold, lead impedance, and magnet response were studied. All diagnostic counters from the pacemaker memory were printed out. A 12-lead ECG recording was taken and, in cases where malsensing or malpacing was present, a chest X-ray was usually made to assess the lead position.

The average age of the studied patients was 70 ± 9 years (range 27 - 92; 101 male and 68 female). Indications for pacing were intermittent or permanent AV block ($n = 64$), intermittent second-degree AV block ($n = 18$), bi- and trifascicular block ($n = 24$), sick sinus syndrome ($n = 55$), and atrial fibrillation with low ventricular rate ($n = 10$). There were no significant differences between the SYMPT and ASYMP groups.

The pacemakers used in this study were of different brands, including Medtronic, Vitatron, CPI, and Biotronik. The leads were from Pacesetter, CPI, Vitatron, Oscor, Medtronic, and Biotronik.

Ambulatory 24h Holter ECG Recording

Holter ECG recordings were made over 24h using special pacemaker recorders (Oxford Medilog, UK) equipped with a third channel for identification of pacemaker stimuli. Using the audiovisual method, a skilled and trained technician scanned the ECG recordings. Automatic quantitative printouts contained data

on heart rate, pauses, and ventricular and supraventricular arrhythmias. The percentage of paced beats was determined. Changes in rhythm, examples of suspected malfunction of the pacemaker, or unusual sequences were printed out at 25 mm/s paper speed for detailed analysis. A pacemaker specialist reviewed all printed ECGs. Special notice was given to the recording of artifacts and the analysis was repeated in some instances without the pacemaker module of the Holter equipment. Whenever possible, the malfunctioning of the pacemaker was provoked, repeated, or otherwise confirmed at the clinic through an extensive evaluation of the pacemaker function.

Definitions

Loss of capture was defined as one or more pacemaker stimuli without capture in the ventricle or in the atrium. The pacemaker output was set to at least 3x pulse width threshold or at least 2x voltage threshold, depending on the threshold measuring system of the pacemaker. Oversensing was defined as any unwanted pacemaker response to a stimulus, i.e., inhibition in the case of VVI and AAI pacing or triggered AV sequential pacing when no event actually occurred in the respective chamber. Myopotential oversensing was assumed in the absence of any event on the ECG recording or any other exterior cause, when myopotentials were present on one of the leads of the Holter ECG recording. Undersensing was defined as a lack of response of the pacemaker in the presence of a physiologic signal in the atrium or ventricle, whichever applicable. Atrial and ventricular sensitivity values were nominally programmed to 0.5 and 2.5 mV, respectively. All atrial leads were bipolar and more than 60 % of the ventricular leads were unipolar, especially in the VVI group.

Results

24h Holter ECG recordings were performed at a median of 2 years after pacemaker implantation (range: 1 week to 6 years). In the SYMPT group, Holter monitoring was taken as an adjunct to pacemaker evaluations conducted in the outpatient clinic. In the ASYMP group, Holter monitoring was performed to assess heart rate for the optimization of pacemaker sensor function. In the latter group, patients did not complain on any discomfort related to the pacemaker system.

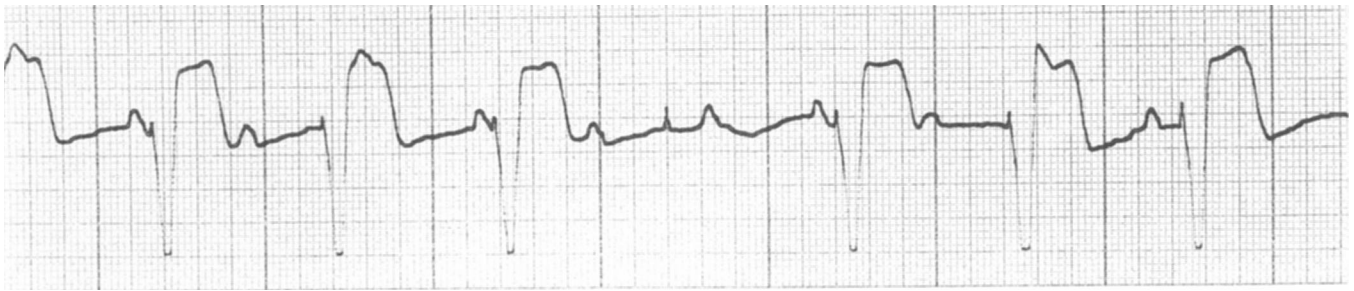


Figure 1. Three weeks after pacemaker implantation, a 53 year-old female patient with a VVI pacemaker and a tined steroid lead (Medtronic 4004) complained on sensations like a "missed beat". Ventricular threshold was excellent (0.8 V / 0.2 ms), pacemaker output was 2.5 V / 0.5 ms, and lead impedance about 400 Ω . Intermittent loss of capture was observed repeatedly. Lead microdislocation was confirmed by fluoroscopy and invasive measurements.

Intermittent loss of capture was observed in 8 out of 41 patients in the SYMPT group and in no patient in the ASYMP group (20 % versus 0 %, $P = 0.0001$) (an illustration is given in Figure 1). Lead dislocation or microdislocation was the major cause of loss of capture. In two screw-in leads, the observed increase in threshold surpassed the maximum pacemaker output, especially during the night.

Oversensing occurred in both groups. In the ASYMP group, oversensing was present in 28 recordings (22 %) compared to 12 positive recordings in the SYMPT group (28 %, $P = ns$). In two patients, oversensing was related to the "thunder" contact in a bipolar screw-in lead (Oscor, see Figure 2). In all other cases, it concerned myopotential oversensing in unipolar leads (Figure 3). Complaints on palpitations or dizziness in the SYMPT group were not related to the pauses. The pauses ranged from 1.0 to 3.3 s, with a median value of 1.3 s. Reprogramming to a lower ventricular sensitivity was necessary only in a few

patients. Myopotential oversensing in the atrium, where all leads were bipolar, was not observed.

Undersensing occurred more frequently in the SYMPT group (in 20 out of 46 leads, i.e., 43 %), which was mainly related to arrhythmia, in comparison to 21 cases of intermittent or isolated undersensing in the ASYMP group (16 %, $P = 0.0001$). Figures 4 and 5 depict undersensing of sinus beats and atrial extrasystoles, respectively. In both cases, the ensuing pause was readily detected through Holter ECG analysis. In general, undersensing occurred more frequently in the atrium (10/19, i.e., 53 %) than in the ventricle (33/162, i.e., 20 %, $P = 0.005$). Undersensing of normally conducted ventricular beats has been observed in a few patients and a hardware failure was suspected to be the underlying cause (Figure 6). In the majority of cases, undersensing in the ventricle concerned extrasystolic beats (Figures 7 and 8). Undersensing of a ventricular extrasystole is potentially more dangerous than undersensing of an atrial extrasystole. The



Figure 2. Episodic intermittent pauses were seen in a 62 year-old male patient with a VVI pacemaker and a bipolar screw-in lead (Oscor). Sensed R-wave amplitude was 10 mV. The duration of the observed pauses were random and independent from the programmed sensitivity. This excluded the possibility of T-wave oversensing. The screw mechanism induced spurious "thunder contact" signals and P-waves showed remarkable spike-like configuration at a total of 560 intervals.

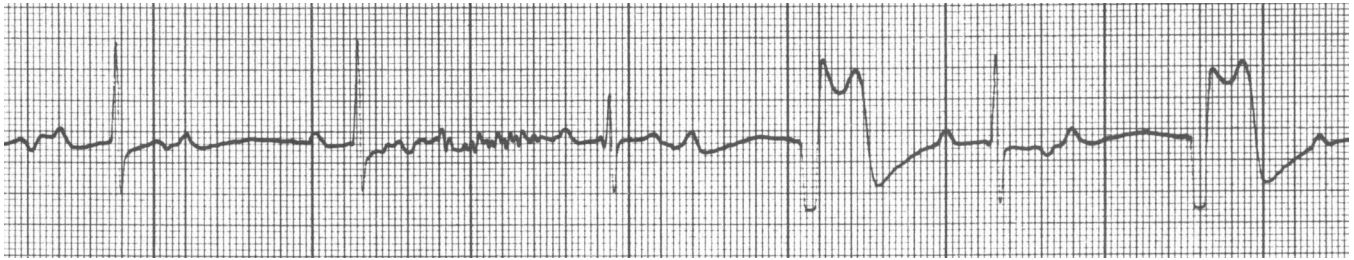


Figure 3. A typical example of myopotential oversensing. The rate hysteresis was programmed to 50 bpm. The first escape interval was not properly timed, which might be caused by pacemaker switching to the "reversion (noise) mode" (Medtronic 8420). Sensitivity value was set to 2.5 mV.

coupling interval of the ventricular extrasystole is related to the pacing interval in VVI and DDD pacemakers, which is guided by tracking or sensor rate. Figure 8 provides an example of pacing in the vulnerable phase of a ventricular extrasystole.

Meticulous pacing and sensing of the pacemakers (defined as 100 % correct) was observed in 10 patients in the SYMPT group (24 %) and in 82 patients in the ASYMP group (64 %, $P = 0.001$).

Discussion

Our study demonstrated that the use of 24h Holter ECG recordings is an effective method for detection of pacemaker malfunction. The observed oversensing malfunction could be in many cases corrected by adjusting the sensitivity value, especially in patients exhibiting myopotential oversensing via unipolar pacemaker leads. A lower sensitivity setting than 2.5 mV is justified in case of unipolar lead systems as long as it allows proper sensing in the ventricle and atrium. In some patients, myopotentials can be sensed at a sensi-

tivity of 7 mV. Adequate sensing of both conducted beats and regular ventricular extrasystolic beats must be assured.

Pauses induced by myopotential oversensing were limited in duration (maximum 3.3 seconds) and occurred without symptoms. The literature on the topic suggests that pacemaker inhibition caused by myopotentials occurs in 17 % to 38 % of patients [5-9]. Gaita et al. report a higher incidence in patients with positive provocative muscle tests and complaints of dizziness [10]. Myopotential oversensing in bipolar lead systems has not been observed in this series as it has in other series [9]. The muscle provocation test can be used for a proper reprogramming of the sensitivity values [9]. Pacemaker behavior should be taken into account during interpretation of myopotential oversensing - this is supported by the example in Figure 3 and has been reported elsewhere [11,12]. A "reversion mode" or "noise mode" has been built into some pacemakers, which in fact means switching to an SOO mode in the event of multiple fast sensed signals. This can be potentially dangerous because it risks ventricular pac-

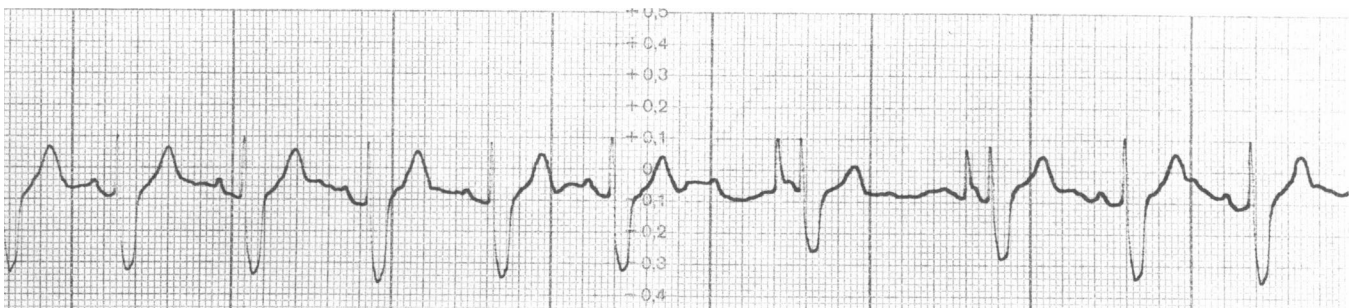


Figure 4. Intermittent undersensing of sinus beats in the atrium, leading to atrial stimulation at a lower rate followed by atrial capture and subsequent ventricular stimulation and capture. The sinus node was reset with the second atrial stimulus at the lower rate. Some patients may experience such episodes as "dropped" beats, due to sudden drop in heart rate.

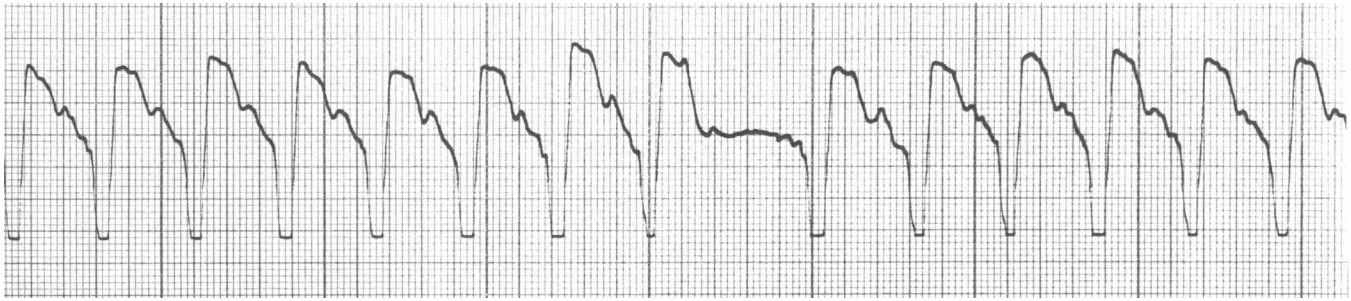


Figure 5. Undersensing of atrial extrasystoles during tachycardia. The reason for failed P-wave sensing could be superimposition of the P-wave on the T-wave falling early during the refractory period, or the P-wave following the T-wave was actually undersensed. In any case, the outcome was generation of pacing rate lower than 60 bpm.

ing in the vulnerable phase of the preceded beat and should be avoided whenever possible [5].

Oversensing of other signals, like "thunder contact", have been observed only rarely (Figure 2). However, it poses an important problem in regard to interpretation and troubleshooting. In these cases, T-wave sensing has to be included, but it also occurs in non-paced events and at random with respect to the cardiac cycle. An analysis in VVT or DDT pacing modes may help solve the problem [13], in addition to the invasive measurements with the lead (Oscor Medical, PY 51 and PY 52). Furthermore, reprogramming of the sensitivity value may help to distinguish T-wave sensing as spurious signals generated by the screw mechanism can be as large as 14 mV [14].

Undersensing of normal beats, either atrial or ventricular, has been described as being similar to our observations (Figures 4 and 6). Berg et al. has also reported undersensing of normal beats in a comparative study [6]. In our case, undersensing of normally conducted beats could not be traced to an earlier event and was

merely due to improper timing of the sensed event in a faulty pacemaker design. Normally, improper sensing despite adequate implantation signals clearly signifies dislocation of the lead. Undersensing of arrhythmias is the major cause, with a frequency falling between 16 % and 35 % [7,9,15,16]. Wiegand et al. [9] observed more undersensing in unipolar systems (35 %) than in bipolar systems (22 %), despite a slightly higher P-wave amplitude in the unipolar systems. In some patients, several repetitive episodes were undersensed. The reasons for this can be diverse, but the vector of an extrasystolic beat can at first be perpendicular to the bipolar dipole, which renders a near zero potential. During implantation, little or no attention is usually given to extrasystolic beats.

Furthermore, P-waves diminish during exercise and further reduce in cases of diseased myocardium as presented in sick sinus syndrome. The P-wave amplitude is inversely related to increasing age [6,17]. P-waves varied over time due to respiration and body position in the range from 0 to 1.7 mV, while the average P-



Figure 6. Undersensing of normally conducted ventricular beats for the reasons not obvious from the ECG-recording. Extensive pacemaker evaluation was unable to reveal the reason for ventricular sensing malfunction. The pacemaker was replaced before the scheduled elective replacement point.

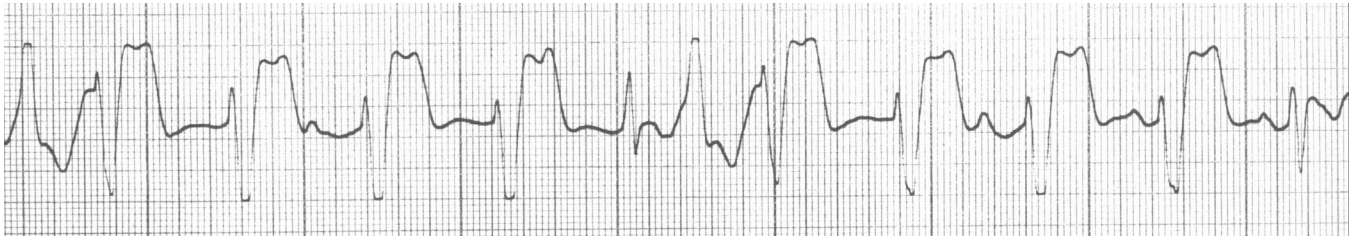


Figure 7. A typical example of undersensing of a ventricular extrasystole. After three paced beats, there were two consecutive extrasystolic beats, the last of which was not sensed.

wave amplitude was 2.30 ± 0.77 mV in that series. Another issue may be also misleading: during implantation and follow-up, the maximum P-values are given and not the minimum measured values. Due to all the aforementioned signal variations, it is quite understandable that atrial arrhythmias as well as normal sinus beats and conducted ventricular beats are occasionally undersensed.

No loss of capture was detected in the ASYMP group, a finding previously reported by other investigators [18-20]. As long as the accepted standard in programming the pacemaker output is maintained, no loss of capture is likely. Still, loss of capture was observed in 8 out of 41 patients in the SYMPT group (20 %). In a previous report on pacemaker patients with syncope after implantation, loss of capture was observed in 4.3 % of patients [21]. Bias in patient selection plays an important role. For example, we have studied several patients in whom steroid leads offered normal impedance and stimulation thresholds despite microdislocation due to a loose distal end, ranging from the ring to the tip of the lead (Figure 1). In some screw-in leads, the stimulation threshold surpassed maximum output of the pacemaker, especially during the night. Dislocation or pacemaker

failure was not detected in this study because patients with obvious dislocation or high threshold were excluded.

Conclusion

Holter ECG recordings revealed a high rate of mild pacemaker malfunction, especially in a subgroup of symptomatic patients. The malfunction was mostly related to unipolar pacemaker systems (myopotential oversensing) and to patients with arrhythmia (undersensing). Atrial arrhythmias were found to be more prone to undersensing. In case of troubleshooting, the Holter ECG is a valuable tool. The high incidence of mild pacemaker malfunction should be taken into account in the design, selection, and set-up of future pacemakers, with the preference for bipolar sensing configuration and high sensitivities as well as optimal characteristics of the sensing amplifiers.

References

- [1] Famularo MA, Kennedy HL. Ambulatory electrocardiography in the assessment of pacemaker function. *Am Heart J.* 1982; 104: 1086-1094.

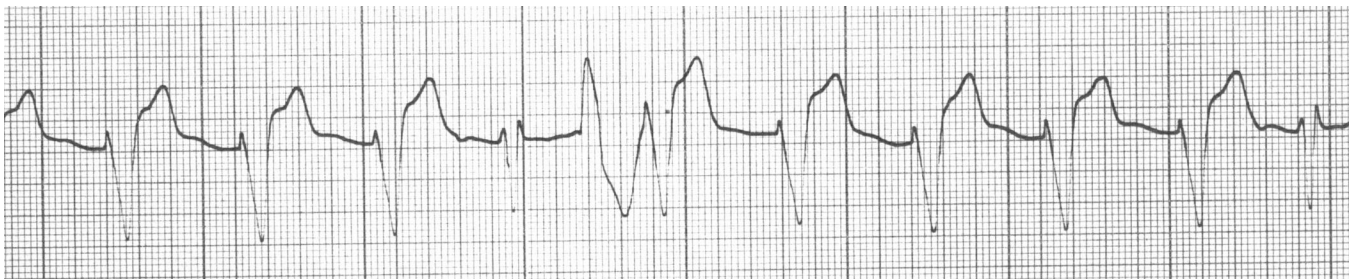


Figure 8. Example of undersensing of a ventricular extrasystole, leading to ventricular pacing according to R-on-T phenomenon.

- [2] Gaita F, Asteggiano R, Bocchiardo M, et al. Holter monitoring and provocative maneuvers in assessment of unipolar demand pacemaker myopotential inhibition. *Am Heart J*. 1984; 107: 925-928.
- [3] Janosik DL, Redd RM, Buckingham TA, et al. Utility of ambulatory electrocardiography in detecting pacemaker dysfunction in the early postimplantation period. *Am J Cardiol*. 1987; 60: 1030-1035.
- [4] Kennedy HL. Ambulatory (Holter) electrocardiography technology. *Cardiol Clin*. 1992; 10: 341-359.
- [5] Beiras Torrado X, Crespo Carazo N, Amorin Ferreiro F, et al. Sensing anomalies with the VVI pacemaker. Clinical study by Holter. *Rev Esp Cardiol*. 1990; 43 (Suppl. 2): 48-51.
- [6] Berg M, Frohlig G, Schwerdt H, et al. Reliability of an automatic sensing algorithm. *PACE*. 1992; 15: 1880-1885.
- [7] Chiu CC, Gow RM, McCrindle BW, et al. Impact of programmed sensitivity safety factor on atrial sensing in children. *PACE*. 1997; 20: 2163-2170.
- [8] Secemsky SI, Hauser RG, Denes P, et al. Unipolar sensing abnormalities: Incidence and clinical significance of skeletal muscle interference and undersensing in 228 patients. *PACE*. 1982; 5: 10-19.
- [9] Wiegand UK, Schier H, Bode F, et al. Should unipolar leads be implanted in the atrium? A Holter electrocardiographic comparison of threshold adapted unipolar and high sensitive bipolar sensing. *PACE*. 1998; 21:1601-1608.
- [10] Gaita F, Asteggiano R, Bocchiardo M, et al. Holter monitoring and provocative maneuvers in assessment of unipolar demand pacemaker myopotential inhibition. *Am Heart J*. 1984; 107: 925-928.
- [11] Van Gelder LM, el Gamal MI. Undersensing in VVI-pacemakers detected by Holter monitoring. *PACE*. 1988; 11: 1507-1511.
- [12] Erkkila KI, Singh JB. Reversion mode activation by myopotential sensing in a ventricular inhibited demand pacemaker. *PACE*. 1985; 8: 50-51.
- [13] Kamke W, Nitschke M, Gutschker A, et al. Facilitation of DDD follow-up using the DDT mode. *PACE*. 1992; 15: 1836-1840.
- [14] Sarmiento JJ. Clinical utility of telemetered intracardiac electrograms in diagnosing a design dependent lead malfunction. *PACE*. 1990; 13: 188-195.
- [15] Bode F, Wiegand U, Katus HA, et al. Inhibition of ventricular stimulation in patients with dual chamber pacemakers and prolonged AV conduction. *PACE*. 1999; 22: 1425-1431.
- [16] Wiegand UK, Bode F, Schneider R, et al. Diagnosis of atrial undersensing in dual chamber pacemakers: Impact of autodiagnostic features. *PACE*. 1999; 22: 894-902.
- [17] Brandt J, Attewell R, Fahraeus T, et al. Acute atrial endocardial P wave amplitude and chronic pacemaker sensitivity requirements: Relation to patient age and presence of sinus node disease. *PACE*. 1990; 13: 417-424.
- [18] Schwaab B, Schwerdt H, Heisel A, et al. Chronic ventricular pacing using an output amplitude of 1.0 volt. *PACE*. 1997; 20: 2171-2178.
- [19] Brandes A, Bethge KP, Gonska BD, et al. Transient pacemaker dysfunction and spontaneous arrhythmias in symptomatic and asymptomatic patients with ventricular demand pacemakers. *Z Kardiol*. 1996; 85: 237-247.
- [20] Stamato NJ, O'Toole MF, Fetter JG, et al. The safety and efficacy of chronic ventricular pacing at 1.6 volts using a steroid eluting lead. *PACE*. 1992; 15: 248-251.
- [21] Pavlovic SU, Kocovic D, Djordjevic M, et al. The etiology of syncope in pacemaker patients. *PACE*. 1991; 14: 2086-2091.

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