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Elevation Of Ventricular Pacing Thresholds: About 30 Cases

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Abstract:

Ventricular pacing threshold elevation is a complication that we frequently encounter in patients with pacemakers, which can be responsible for a recurrence of life-threatening conduction disorders. This is why it is very important to explain to implanted patients the importance of regular follow-up, and to encourage them to consult a specialist at the slightest recurrence of clinical symptoms. Our work included 30 implanted patients who raised their threshold during follow-up. It is a retrospective observational and a descriptive study carried out in the Cardiology B department at the Souissi Maternity Hospital in Rabat over a period of two years, from 2020 to 2022. The aim of the study is to characterize chronic pacing lead performance during long-term follow-up and to identify clinical risk factors associated with increased threshold in this population.

Keywords:

ventricular pacing threshold elevation, pacemaker complications

Introduction:

Pacemaker is a medical device delivering electrical impulses to stimulate the heart muscle. It is the first-line treatment for symptomatic bradyarrhythmias, and its implantation has an important role in interventional cardiology practices worldwide.

When implanting a permanent pacemaker, three important parameters must be carefully tested: detection, lead impedance and pacing threshold. (1)

The stimulation threshold is defined as the minimum amount of energy required to capture myocardial tissue electrically. It can be measured in voltage (volts) or pulse width (milliseconds) and must be less than 1 volt, but it depends on the characteristics of the myocardium, the lead and the quality of contact between the lead and the myocardium. It varies over the implantation period, with chronicity estimated at 3 months post-implantation. During follow-up, the implantation threshold may undergo variations influenced by physiological, pharmacological, pathological or mechanical factors related to lead lesions. If a progressive or abrupt rise in stimulation threshold is noted during regular monitoring, or if it is discovered by a clinical or electrical event, the causes and contributing factors need to be investigated.

Systematic data on the permanent increase in pacing thresholds after pacemaker implantation are scarce, and data on the preoperative, periprocedural and postoperative characteristics associated with threshold increase have not been fully clarified, particularly in patients with no lead fracture or dislodgement.

Patients and Methods:

1 - Study population:
Our work is a retrospective study with a descriptive aim based on the exploitation of patients' files followed in consultation of pacemaker interrogation at the rhythmology unit of the cardiology department B at the Souissi Maternity Hospital in Rabat during a period of 2 years, from the year 2020 to 2022. Out of a total number of 612 pacemakers implanted during this period, we were able to collate 30 patients with a high threshold in our study.

Inclusion criteria: patients implanted with a single or dual chamber pacemaker in our department who had a normal ventricular threshold after implantation and in whom a ventricular threshold elevation was discovered during follow-up.

Exclusion criteria: patients lost to follow-up, irregular consultation follow-up and lack of data or unusable files.

2- Patient follow-up and data collection:
We set up an operating form on which we reported the demographic data of our population, as well as cardiovascular risk factors, cardiac and extracardiac comorbidities and medications taken. We specified the date and indication of primary implantation and whether it was a single or dual chamber pacemaker.
Discharge parameters were specified, specifically lead stimulation thresholds, impedances and lead positions. The cutoffs respected in our study were; a stimulation threshold below 1 V, and impedance between 200 and 1000 ohms.
Post-operative complications were noted, including pneumothorax, haematoma and infection. Our patients were regularly monitored.
The first check-up is carried out 3 months after implantation, providing standard follow-up by collecting measured data (stimulation and detection thresholds, stimulation impedance and battery status), with particular emphasis on the stimulation threshold. Subsequently, on a yearly basis, depending on the length of time the patient has been monitored.
When an elevated threshold is detected, an interview is repeated carefully, along with an electrocardiographic, biological and radiological check to verify lead integrity. This is followed by a specialized discussion in the rhythmology unit, for a therapeutic decision.

3- Statistical analysis:
This was a descriptive observational study.
The quantitative variable (age) was described in median ± standard deviation. The normality of its distribution was decided on the basis of the Shapirowilk test. All other variables studied were qualitative and were described in terms of numbers and percentages.
All statistical analyses were performed using Jamovi software. (2)

4- Ethical conditions:
The ethics committee was not consulted, as this was a retrospective study whose data were collected anonymously.

Results:

30 patients were included in our study. Table 1 reports the results of our descriptive study, summarizing all demographic data, cardiovascular risk profile, antecedents and comorbidities of patients, as well as data related to primary implantation and follow-up.

	Variables	Total (n= 30)
Demographic data	Age (years)	62,9 ± 12,2
	Female	11 (36,6)
	Male	19 (63,4)
Cardiovascular risk factors and comorbidities	Arterial hypertension	6 (30)
	Diabetes	18 (60)
	Ischemic cardiopathy	12 (40)
	Other cardiopathies	8 (26,7)
	Antiarrythmic drugs	0
Primo implantation	Pacemaker: Dual chamber Single chamber	 25 (83,3) 5 (16,7)
	Lead positions : Apex Low septum Mid-septum	 15 (50) 4 (13,3) 11 (36,7)
	Post implantation complications : Pneumothorax Hematoma Infection	 0 0 0
Follow up	Delay between first implantation and high threshold: After 3 months After 1 year After 2 years After 4 years After 5 years After 7 years	 1 (3,3) 7 (23,3) 4 (13,3) 5 (16,7) 10 (33,3) 3 (10)
	Threshold values: Between 1 and 2 V Strictly between 2 and 3 V Greater than or equal to 3 V	 9 (30) 13 (43,3) 8 (26,7)
	Brutal threshold elevation Progressive threshold elevation Impedance elevation	17 (56,7) 13 (43,3) 0
	Radiological abnormalities: Lead displacement Lead fracture	 17 (56,7) 0
	EKG: Electro-stimulated rhythm Conduction disorder	 27 (90) 3 (10)
	Biological abnormalities: Dyskaliemia High level of HbA1C High level of troponin	 7 (23,3) 6 (20) 0
Therapeutic decisions	Old lead repositionning	8 (26,7)
	New lead implantation in RV	9 (30)
	Surveillance	13 (43,3)

Discussion:

A/ Generalities:

1- Epidemiological data:

Implantation of a permanent pacemaker is the only established treatment for bradyarrhythmia that improves prognosis and quality of life. (3) Over the past ten years, 50,000 implantable pacemakers have been introduced in Japan every year. In the United States, more than 350,000 pacemakers are implanted every year, and this figure has doubled over the past 30 years. (4)

The number of pacemakers implanted in the rhythmology unit of our Cardiology B department is also set to rise, with 272 implanted in 2022, compared with 204 in 2021 and 136 in 2020. The number of lead repositionings has also risen, from 4 in 2020 to 9 in 2021 and 7 in 2022.

The elderly are more prone to bradyarrhythmia, and it is estimated that more patients will require pacemaker implantation as society ages. Hence the importance of understanding these vital devices and preventing the various potential complications that could jeopardize their vital prognosis.

2- Pacemaker implantation procedure followed in our department:

All patients included in the study were implanted in our department for high-grade conductive disorder under local anesthesia with 2% Xylocaine.

After 3 cm incision two fingerbreadths from the right clavicle near the deltopectoral groove, the subcutaneous plane was dissected and the pacemaker pocket was made behind the aponeurosis of the pectoralis major muscle, with meticulous attention paid to haemostasis.

If the cephalic vein cannot be used (very deep or absent), the subclavian vein is punctured.

This is followed by the insertion of two leads, one ventricular, the other atrial, which are tested using a programmer, then screwed into the myocardium once the correct parameters have been obtained, and finally attached by their olives to the pectoralis major muscle.

The leads are then connected to the box, which is inserted into the lodge after meticulous asepsis to obtain an electro-trained rhythm. Lastly, the skin is closed plane by plane using separate stitches or overlocking, depending on the state of hemostasis.

A temporary stimulation was used to patients who required it, and removed at the end of the procedure.

3- Prevalence of increased stimulation threshold:

Although there have been technological improvements in the manufacture of pacemakers and pacing leads (5), the ventricular pacing threshold can increase over time, reducing battery longevity and increasing the risk of a potentially fatal cardiac event secondary to pacing abnormalities (6)(7). The prevalence of increased pacing threshold varies from 4% to 25% depending on the type of study. (8)

In a study that retrospectively recruited a total of 1033 patients who underwent initial pacemaker implantation between January 2008 and June 2016 and had a long-term follow-up visit lasting 5.4 ± 2.1 years, a permanent increase in ventricular pacing threshold was found in 8.4% (or 87/1033) of patients. (9)

Previous studies have shown that standard steroid-eluting leads effectively reduce the increase in stimulation threshold caused by inflammatory reactions at the tissue-lead interface after implantation (10). This effect was stable and maintained during 5 or 10 years of follow-up (11)(12). Nevertheless, Biffi et al (13) reported a permanent increase in stimulation threshold > 3 V in 4 of 126 patients (3%) beyond one year in a single-center experiment. Kistler (14) and Medi (8) reported an increase in stimulation threshold > 3 V in 4% of patients and 2% of patients over a follow-up period of 24 months and 6 months respectively.

These short-term observational studies confirm that the ventricular pacing threshold can increase in patients with modern leads too, and that a permanent increase in the latter ≥ 2.5 V occurred in around 2-4% of patients.

Furthermore, the frequency of pacing threshold increase was similar over the first 6 years of follow-up, suggesting that this increase can occur at any time during follow-up, and that patients may be vulnerable to this event at any time. Regular follow-up is therefore essential to detect life-threatening disturbances. In our patients, all the stimulation leads used are steroid-eluting.

4- Measures to be taken upon discovery of a high threshold:

Once an increase in pacing threshold is detected, pulse width and/or output can be increased to improve the margin of safety by automatic verification of pacemaker capture. (9) Lead integrity and position should be checked in the first instance by impedance measurement and X-ray inspection.

If the lead is displaced, repositioning is necessary; if it is fractured or damaged, it must be replaced.

On the other hand, if the fracture or displacement of the lead is eliminated, a new follow-up visit should be scheduled every month until the threshold shows fluctuations ≤ 1 V. In this case, the parameters influencing the stimulation threshold should be checked and corrected if necessary.

If no improvement is obtained during follow-up, a new ventricular lead is required after extraction of the old lead if this is possible, or after its fixation in the pectoralis major muscle if it is old or difficult to remove.

B/ Demographic and clinical characteristics of patients with elevated stimulation thresholds

1- Demographic parameters:

In a study that carried out long-term follow-up in 1033 patients (9), the mean age was 68.98 ± 8.68 in patients with pacing threshold elevation compared with 62.9 ± 12.2 years in our study, which probably suggests that the elderly are more likely to raise the pacing threshold in contrast to younger subjects. This may be explained by the higher incidence of pacemakers in elderly subjects and the higher frequency of comorbidities in this group of patients.

The sex ratio in our population was 1.7, with males predominating at 63.4%.

In the same comparative study cited above, more men than women had raised their pacing thresholds, which is probably due to the greater frequency of cardiovascular events in men than in women, which may be responsible for a higher pacing threshold.

2- Influence of cardiovascular risk factors:

Cardiovascular risk factors are associated with a higher incidence of stimulation threshold.

Diabetes is considered to be the main factor behind the permanent and chronic increase in pacing threshold (9). In our study, 60% of patients were diabetic. Another study showed that patients with an increased stimulation threshold had higher blood glucose and HbA1c levels than those without an increased threshold (9). Zdarska et al (15) revealed subtle changes in the heart's electric field by electrocardiogram with significantly reduced R-wave amplitudes in diabetic patients. This suggests that hyperglycemia may influence the excitability of cardiac tissue.

Further analysis revealed that diabetic patients with elevated pacing thresholds exhibited increased oxidative stress. Aging and death of human myocardial cells mediated by oxidative stress activation leads to an accumulation of interstitial fibrotic tissue deposits and changes in myofibrillar proteins, leading to fibrosis, increase of myocardial stiffness and tension (16) that could theoretically influence myocardial excitability threshold, making diabetic myocardium more prone to negatively affect pacing threshold. (17)

Hypertension is a cardiovascular risk factor found in 30% of our patients. Its direct association with increased threshold is not well elucidated in the literature. However, its correlation with increased cardiovascular risk and the occurrence of ischemic events, as in 40% of cases in our study, may explain the relationship with increased pacing threshold.

3- Ischemic heart disease and threshold elevation

Cui et al (18) demonstrated that acute ischemia lasting more than 30 minutes could increase the pacing threshold almost threefold compared with baseline. Local ischemia lasting several hours can significantly alter myocardial resistivity. The electrical uncoupling of myocardial cells by ischemia leads to a 50-100% increase in intracellular resistance. (19). Recently, Chen et al (20) and Upadhyay et al (21) reported that pacemaker parameters returned to normal after revascularization.

Pivatto et al (22) also described an improvement in pacing threshold after early reperfusion. But in another study (9), myocardial infarction remained associated with a permanent and chronic increase in pacing threshold, despite the fact that 94% of patients with myocardial infarction had been revascularized. This effect could be explained by increased myocardial ischemia involving multiple territories in patients with increased threshold.

It could also be due to the diagnostic difficulties of acute coronary syndrome in pacemaker patients because of atrioventricular block. As the initial electrocardiogram of patients with acute myocardial infarction is very important for invasive treatment in the acute phase, it is not surprising that patients with an electro-trained rhythm are less likely to benefit from primary angioplasty than patients with a spontaneous rhythm. Further research into this question seems warranted. In our series, none of our patients presented a clinical or biological presentation of a coronary syndrome that could explain the chronic or acute evolution of the pacing threshold.

4- Effect of antiarrhythmic treatments

Some studies have found an association between chronic amiodarone treatment and a significant increase in the defibrillation threshold. (23)

Another study also observed greater amiodarone use in the group including patients with a high pacing threshold, but multivariable analysis did not result in a statistical difference (9). Most patients who used amiodarone had underlying heart disease. It is therefore possible that these patients have an intrinsically higher risk of developing an increased pacing threshold. Huang et al (24) also studied the influence of different antiarrhythmic drugs, including amiodarone, on chronic pacing threshold in healthy dogs and found that these drugs did not affect chronic threshold or endocardial R-wave amplitude.

The table below summarizes these effects:

Vaughan Williams classification	Anti-arrhythmic drugs	Effect for pacing threshold
I a	qunidine	↑
	procainamide	↑～↑↑
	ajimaline	↑
	disopyramide	↑
	cibenzoline	↑↑
	pirmenol	↑～↑↑(?)
I b	aprindine	↑
	lidocaine	→
	phenytoin	→
	mexiletine	↑
I c	propafenone	↑↑↑
	pilsicainide	↑↑↑
	flecainide	↑↑↑
Ⅱ	propranolol	→
Ⅲ	amiodarone	→
	sotalol	→
Ⅳ	verapamil	→
—	digitalis	→～↓

↓: decrease, →: no change, ↑: increase

5- Lead position:

The choice of position for implantation of the pacing lead in the right ventricle must take into account the need to preserve interventricular synchrony, and therefore mimic as closely as possible the path of physiological myocardial depolarization. Thus, the septal position is the most recommended. However, it carries a higher risk of lead displacement than the apical position. It is therefore essential to test the stability of the leads and give them sufficient length to ensure secure fixation.

50% of our patients had the septal position more medium than high, and 50% in the apical position.

C/ Delay between first implantation and elevated threshold

One study divided 109 patients with elevated ventricular threshold according to the time between primary implantation and discovery of the elevated threshold. The maximum increase occurred during the first year after implantation in 13.8% (15/109) of patients, during the second year in 19.3% (21/109) of patients, between the third and sixth year in 58.7% (64/109) of patients, and after the sixth year in 8.2% (9/109) of patients. (9)

These results are in line with those found in our population, with a predominance of the high threshold in the first 5 years after implantation. This is probably due to the greater vulnerability of leads and the greater frequency of displacement or fracture accidents in the first years after implantation, and also to the earlier discovery of reversible factors that can increase the threshold.

D/ Anomalies associated with increased stimulation threshold discovered during follow-up

The discovery of an increased ventricular pacing threshold calls for a number of measures to be taken.

1 - Radiological abnormalities:
A radiological check of the face, profile and OAG is essential whenever a sudden rise in the pacing threshold is detected. Lead displacement is a complication that causes a rise in stimulation threshold, with variable incidence.
When lead displacement is quite obvious on radiography, with a lead that has changed position in comparison with immediate post-implantation reference radiography, the cause of elevated threshold is mechanical, but the diagnosis remains quite complex in the case of unidentified micro-displacement on control radiology.

2- Electrical abnormalities:
A rise in pacing threshold concomitant with a recurrence of clinical symptomatology after post-implantation improvement calls for an electrical evaluation and interrogation of the pacemaker.
The association of a ventricular capture or detection defect with a change in ventricular lead impedance may be related to a pacing lead fracture.

3- Biological abnormalities:
During routine questioning, patients presenting with threshold elevations not explained with radiography should not hesitate to ask for a biological control.
Indeed, many factors may explain this increase, including hypothyroidism, hyperkalemia and hyperglycemia. (25)
One study even focused on the relationship between serum uric acid levels and ventricular pacing threshold values, showing that patients with high serum uric acid levels at the time of implantation had higher ventricular pacing threshold values than those with low serum uric acid levels. (1) By day 30 post-implantation, median threshold decreases were higher in patients with low serum urea levels. To prolong pacemaker life, therefore, it is worth noting serum uric acid levels among the factors associated with threshold increase.
Reversible factors certainly increase the pacing threshold, but only slightly and gradually. However, a high threshold can lead to a recurrence of conduction disorders associated with rapid battery depletion, or to a potentially fatal pacing anomaly. It is therefore essential to correct electrolyte disorders, specially kalemia and uric acid levels, to control diabetes with regular monitoring of dietary hygiene measures and HbA1C levels, and to combat all other cardiovascular risk factors that may be responsible for ischemic events.

E/ Therapeutic decision

In the event of elevated threshold, the therapeutic decision depends on whether it is sudden or progressive, whether the cause is reversible or not, how long the lead has been implanted, and above all on the clinical and electrical consequences.
A progressive and discreet elevation with no clinical repercussions calls for regular surveillance, with closer monitoring to detect a more serious anomaly requiring urgent action, as in the case of 13 of our patients with a progressive elevation, who were monitored for at least 2 years without requiring any special management.
For patients diagnosed as having a mechanical cause with lead displacement, urgent management with repositioning of the same lead or reimplantation of a new lead was required in 17 of our patients.
When the lead is old, it is difficult to remove, either to reposition it or to remove it for replacement. In such cases, laser therapy is recommended for lead extraction. The general principle of extraction is that the risk of the procedure is lower than the risk of leaving the leads in place. This is the case in the majority of patients with high thresholds, who are at risk of progressing to major life-threatening conductive disorders. Most lead extractions are performed via a percutaneous trans-venous approach. However, there are three main indications for open surgical extraction: failure of percutaneous lead extraction, non-surgical heart disease

(such as pre-existing need for valve replacement) or endocarditis with very large infected vegetations, which would require open extraction to minimize embolic complications.
In our department, because of the unavailability of these therapies, we simply keep the old lead, which we fix in the pectoral muscle, and carefully insert a second new ventricular lead, as in 9 of our patients, with verification of pacing parameters before the patient is discharged from the operating room.
Studies and series on this subject are limited to clinical cases, thus restricting discussion of the appropriate way to proceed.

Conclusion:

As the pacemaker patient population increases in size and longevity, the issue of increased pacing threshold is likely to become more important, as it is likely to affect the longevity of the pacing system, or even pose a life-threatening safety issue.
Our study highlights the demographic and clinical profile of patients with threshold elevation, who should be considered as particularly at-risk patients and taken into account in treatment and management. We have also studied the long-term behavior of this parameter during follow-up in our rhythmology department, as well as the causes involved in this increase and their correction.
All in all, this is a retrospective, descriptive and observational study, and the results of the analysis must be interpreted in the light of this fact. However, this type of study reflects usual clinical practice. An important limitation is the small size of the study population. Larger prospective studies with longer follow-up are therefore needed to better identify the risk factors associated with the permanent increase in stimulation threshold.

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