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Relationship between the change in pacing threshold and the myocardial injury

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Abstract. – OBJECTIVE: To investigate the effects of general conditions and past medical history of patients on the short-term pacing threshold after the implantation of active electrodes of a cardiac pacemaker, and to understand whether the application of active electrodes will cause muscle injury and the severity of the injury.

PATIENTS AND METHODS: A total of 156 patients who were treated with a cardiac pacemaker and implanted with single or double active electrodes in Cardiovascular Department of Central Hospital of Jiangjin District were enrolled, including 96 patients treated with a single active electrode and 60 patients treated with double active electrodes. Their clinical data were collected. During operation, the short-term atrial and ventricular pacing thresholds were monitored and recorded after the implantation of the active fixed electrode lead.

RESULTS: Multivariate Logistic regression analysis showed that female sex, age, smoking, drinking, coronary heart disease, diabetes mellitus and hyperlipidemia had effects on pacing thresholds in a short-term to some extent after the implantation of active fixed electrode lead of cardiac pacemaker. The levels of myocardial enzymes, myoglobin (MYO), creatine kinase (CK), creatine kinase-MB (CK-MB), lactate dehydrogenase (LDH) and cardiac troponin I (cTNI), in patients were gradually increased after the implantation of active fixed electrode lead and reached the peak at 24 h, which were higher than those before operation ($p < 0.05$). The levels were decreased at 72 h with statistically significant differences.

CONCLUSIONS: Female sex, age, smoking, drinking, coronary heart disease, diabetes mellitus and hyperlipidemia are independent influencing factors of pacing thresholds in a short term after the implantation of active fixed electrode lead of cardiac pacemaker. The implantation of active electrodes can raise the myocardial enzyme indexes compared with those before operation, but will fall to the preoperative levels or normal, near normal levels at 72 h after operation. The implantation of active electrodes is safe and feasible for myocardial tissues.

Key Words:
Pacing threshold, Myocardial enzyme, Myocardial injury.

Introduction

The pacing threshold is the most important functional parameter in the cardiac pacing system. Lower thresholds can not only greatly save the pacemaker's battery power and extend the pacemaker's service life, but also reduce or eliminate the possible discomfort of patients caused by the larger pacing stimulation. Since the pacing electrode lead was clinically implanted via the right ventricular endocardium for the first time in 1958[1], muscle trabecula in the right ventricular apex (RVA) has been conveniently determined as the clinically conventional classic pacing site. The passive wing-shaped electrode lead is the product of implantation into this site, which is characterized by the simple operation, easy positioning, low dislocation rate and fewer complications, so it is widely used in clinical practice. However, with the announcement of research results of CTOPP and UKPACE[2], people have gradually recognized the drawbacks of non-physiological pacing, and RVA has been treated as the worst pacing site due to its worst haemodynamics. The above conclusion was proved by DANISH study, PASE test, UKPACE study, MOST test, etc.[3-5]. Nowadays, people try to avoid these problems caused by non-physiological pacing using different pacing methods and sites, obtaining the physiological pacing effect as far as possible. The right ventricular septal

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pacing is the closest to the His bundle, and the pacing near the His bundle can maximize the transmission of physiology with double-ventricle synchronism. Unlike the wing-shaped electrode lead, the active fixed electrode lead will make the myocardial damage under endocardium. Additionally, the myocardial tissues at the implantation site can suffer from congestion, edema, and necrosis, thus increasing the immediate pacing threshold after implantation. The results of existing studies[6] show that complex conditions, such as the pulse generator, pacing lead, surrounding environment and human body, can interfere with the stability of pacing threshold and cause the dynamic changes. However, there is little research on the changes in short-term threshold after the implantation of active fixed electrode lead of cardiac pacemaker, and its influencing factors. Moreover, the severity of myocardial damage after the lead is unscrewed from the screwed end, and screwed into the cardiac muscles remains unknown. In this paper, the pacing threshold immediately and within 5-10 min after the implantation of active electrodes was defined as the short-term pacing threshold. The primary purpose of this study was to investigate the effects of general conditions and past medical history of patients on the short-term pacing threshold after the implantation of active fixed electrodes of cardiac pacemaker, and their relationships with myocardial damage.

Patients and Methods

Patients

A total of 156 symptomatic bradycardia patients who were treated with cardiac pacemaker in Cardiovascular Department of Central Hospital of Jiangjin District from September 2014 to May 2017 were collected, including 83 males and 73 females with an average age of (60.81 ± 19.39) years old. There were 122 cases of sick sinus syndrome, 8 cases of Degree II atrioventricular block, 18 cases of Degree III atrioventricular block and 8 cases of chronotropic incompetence. 96 patients were treated with single active fixed electrode (ventricular active electrode) and 60 patients were treated with double active fixed electrodes (atrial and ventricular active electrodes). All patients satisfied the type I and IIa indications in ACC/AMS Treatment Guidelines of Abnormal Cardiac Rhythm in 2008. All patients underwent preoperative electrocardiogram, cardiac ultrasound and biochemical examinations (including high-sensitivity C-reactive protein and renal function, etc.), etc.; the brain natriuretic peptide (BNP) and myocardial enzymes were also detected. The antiplatelet drugs were withdrawn. The general conditions of all patients were recorded, including the gender, age, histories of smoking and drinking and accompanying diseases, such as coronary heart disease, hypertension, diabetes mellitus and hyperlipidemia. All patients signed the informed consent. This study was approved by the Ethics Committee of Central Hospital of Jiangjin District.

Materials

Active fixed electrode model: Medtronic RED R015076 or St. Jude Tend Riltms 7120/1888TC or Biotronik Talos D selox S 60 (Medtronic, Inc., Minneapolis, MN, USA). (Report Town and Country)**.** The active electrode leads were implanted into all patients successfully in one time during operation, and there were no complications during and after operation; the pacemaker worked normally.

Operation Methods

Before operation, patients signed the informed consent. Under a supine position, the skin at the surgical site was preserved and disinfected; under the local anesthesia, the left/right subclavian/axillary vein was punctured using the Seldinger technique and the guide wire was guided into the inferior caval vein, which was confirmed under the perspective view. Then, a 3 cm-long incision was made on the skin and bluntly separated to the deep fascia, and the capsular bag was made for full hemostasis. It was sent by the guide wire, and the inner sheath and guide wire were removed. Next, the right atrial and (or) right ventricular spiral electrode lead with straight guide wire was placed into the right atrium and (or) right ventricle along the outer sheath and then the straight guide wire was removed. The shaped guide wire was used for right atrial/ventricular electrode, and the electrode lead was sent into the fixation sites in outflow tract of right atrium/right ventricle through the rotation and pushing method. Under the perspective view, the active electrode lead screw was completely screwed out and the guide wire was removed. The pacing parameters were detected. After the satisfactory test, patients were required to take a deep breath and cough to observe whether there was electrode dislocation; if not, the wire tension might be appropriately adjusted, the right atrial/ventricular electrode was

fixed, the electrode and pulse generator were connected and the capsular bag was implanted. The subcutaneous tissues and skin were sutured layer by layer, and whether there were complications was observed after operation. The surgical area was pressed by sand bag for 6 h, and patients could get out of bed at 12 h.

Observational Indexes

After the right atrial/ventricular active fixed electrode was in place, the spiral electrode lead was screwed out. The pacing thresholds were detected immediately and within 5-10 min after the screwing out using Medtronic5318, respectively. The levels of myocardial enzymes were detected before operation, at 24 h, 48 h and even 72 h after operation.

Statistical Analysis

Multivariate Logistic regression analysis was performed for the influencing factors of atrial and ventricular short-term pacing thresholds. $p < 0.05$ suggested that the difference was statistically significant. The measurement data in normal distribution were presented as mean ± standard deviation, while the enumeration data were presented as %. The repeated measurement of analysis of variance followed by Least Significant Difference (LSD) as the post hoc test was used for the comparison of myocardial enzymes in different time and different groups. All data were analyzed by Statistical Product and Service Solutions (SPSS) 16.0 software (SPSS Inc., Chicago, IL, USA).

Results

Analysis of Influencing Factors of Atrial and Ventricular Short-Term Pacing Thresholds

The effects of general conditions on pacing threshold in a short term after the implantation of active electrode of cardiac pacemaker were analyzed *via* the multivariate Logistic regression. The gender, age, hypertension, coronary heart disease, diabetes mellitus, hyperlipidemia, smoking, and drinking as independent variables and ventricular pacing thresholds immediately and at 5-10 min after implantation were defined as dependent variables. The results showed that the coronary heart disease, diabetes mellitus, hyperlipidemia, smoking and drinking were influencing factors of immediate ventricular pacing threshold (Table I); the age, diabetes mellitus and drinking were influencing factors of ventricular pacing threshold at 5-10 min (Table II). Similarly, the logistic regression analysis was performed with the gender, age, hypertension, coronary heart disease, diabetes mellitus, hyperlipidemia, smoking and drinking as independent variables and with atrial pacing thresholds immediately and at 5-10 min after implantation as dependent variables. The results revealed that the coronary heart disease and smoking were influencing factors of immediate atrial pacing threshold (Table III); the diabetes mellitus and smoking were influencing factors of atrial pacing threshold at 5-10 min (Table IV).

Comparison of Each Myocardial Enzyme Level in Different Time

The myocardial enzymes before operation and at 24 h, 48 h and 72 h after operation were detected. The above myocardial enzyme levels in different time were used to analyze its change rule and understand the severity of myocardial damage. Before operation, the serum myoglobin (MYO) level was (57.37 ± 22.78) ng/mL, the creatine kinase (CK) concentration was (67.41 ± 30.56) U/L, the creatine kinase-MB (CK-MB) concentration was (17.01 ± 8.24) U/L, the lactate dehydrogenase (LDH) concentration was (187.34 ± 39.43) U/L, and the cardiac troponin I (cTnI) concentration was 0.01±0.01 ng/mL.

Table I. Influencing factors of immediate ventricular pacing threshold.

	B	S.E	Wald	*p*	OR (95% CI)
Gender	0.424	0.487	0.831	0.289	1.532 (0.627, 3.273)
Age	0.051	0.028	3.284	0.042	1.073 (0.986, 1.248)
Hypertension	0.796	0.528	1.964	0.164	2.763 (0.731, 4.683)
Coronary heart disease	1.384	0.610	5.284	0.021	3.863 (1.183, 9.382)
Diabetes mellitus	3.976	0.631	21.482	< 0.001	21.847 (7.927, 63.483)
Hyperlipemia	1.473	0.701	7.084	0.005	5.872 (1.473, 16.973)
Smoking	1.635	0.697	5.987	0.015	5.027 (1.294, 17.498)
Drinking	2.546	0.681	11.842	0.001	11.384 (2.489, 42.482)

Table II. Influencing factors of ventricular pacing threshold at 5-10 min.

	B	S.E	Wald	p	OR (95% CI)
Gender	1.729	0.726	5.786	0.115	5.219 (1.482, 21.876)
Age	0.120	0.038	6.698	0.014	1.492 (1.749, 1.294)
Hypertension	0.583	0.518	0.649	0.487	1.497 (0.385, 6.524)
Coronary heart disease	0.621	0.698	6.608	0.424	1.794 (0.369, 9.372)
Diabetes mellitus	1.834	0.643	0.974	0.002	6.075 (1.972, 21.639)
Hyperlipemia	-1.074	1.478	0.864	0.387	0.369 (0.046, 2.944)
Smoking	-1.264	1.686	0.796	0.321	0.258 (0.029, 3.694)
Drinking	3.764	1.542	7.523	0.003	40.274 (4.294, 194.382)

Table III. Influencing factors of immediate atrial pacing threshold.

	B	S.E	Wald	p	OR (95% CI)
Gender	0.124	0.782	0.018	0.892	1.145 (0.218, 5.808)
Age	-0.02	0.05	0.047	0.749	0.963 (0.746,1.372)
Hypertension	0.784	1.624	0.780	0.361	2.394 (0.318, 16.384)
Coronary heart disease	2.593	1.174	4.184	0.041	12.485 (1.084, 64.01)
Diabetes mellitus	1.184	0.739	3.075	0.198	3.937 (0.783, 12.042)
Hyperlipemia	-0.295	0.972	0.087	0.740	0.724 (0.132, 3.483)
Smoking	2.964	1.458	1.394	0.016	21.484 (1.972, 143.92)
Drinking	-1.424	1.395	1.296	0.108	0.174 (0.074, 2.472)

There were statistically significant differences in the comparisons of serum MYO level before operation and in different time after operation ($F = 36.196$, $p < 0.001$). The further pairwise comparison revealed that the serum MYO level at 24 h after operation was increased compared with that before operation ($p < 0.05$), and it fell to the preoperative level at 48 h and 72 h after operation ($p < 0.05$). Besides, there were statistically significant differences in comparisons of CK-MB level before operation and in different time after operation ($F=11.469$, $p < 0.001$). The further pairwise comparison revealed that the CK-MB level at 24 h after operation was increased compared with that before operation ($p < 0.05$), decreased at 48 h after operation compared with that at 24 h after operation, and also decreased at 72 h after operation compared with that at 48 h after operation. Also, it fell to the preoperative level ($p < 0.05$). The variation trends of CK, LDH and cTnI were consistent, and there were statistically significant differences in the comparisons before operation and in different time after operation ($p < 0.001$). The further pairwise comparison revealed that the levels at 24 h and 48 h after operation were increased compared with those before operation ($p < 0.05$), and CK fell to the preoperative level at 72 h after operation ($p < 0.05$) (Table V).

Comparison of Each Myocardial Enzyme Level in Different Groups

There was statistically significant difference in each myocardial enzyme (MYO, CK-MB, CK, LDH, cTnI) level before operation and in dif-

Table IV. Influencing factors of atrial pacing threshold at 5-10 min.

	B	S.E	Wald	p	OR (95% CI)
Gender	1.374	0.964	1.693	0.164	2.495 (0.853, 21.494)
Age	-0.021	0.069	0.183	0.836	0.972 (0.842, 1.742)
Hypertension	-1.353	1.482	1.837	0.184	0.294 (0.018, 2.492)
Coronary heart disease	-0.371	1.359	0.074	0.749	0.684 (0.041, 14.824)
Diabetes mellitus	2.087	1.485	9.245	0.005	24.482 (3.592,130.242)
Hyperlipemia	0.594	1.185	0.285	0.824	1.429 (0.279, 13.593)
Smoking	3.795	1.482	9.976	0.002	47.294 (3.593,149.920)
Drinking	-1.532	1.794	1.620	0.294	0.164 (0.017, 2.837)

Table V. Myocardial enzyme levels in different time.

Time point	MYO (ng/mL)	CK (U/L)	CK-MB (U/L)	LDH (U/L)	cTnI (ng/mL))
Before operation	60.37 ± 22.78	67.41 ± 30.56	17.01 ± 8.24	187.34 ± 39.43	0.01 ± 0.01
24 h	79.34 ± 41.86*	101.74 ± 71.56*	19.63 ± 7.40*	222.67 ± 42.69*	0.06 ± 0.04*
48 h	56.10 ± 23.77♦	93.82 ± 63.97*	18.42 ± 6.84*♦	218.54 ± 40.41*	0.04 ± 0.03*
72 h	55.98 ± 19.97♦	70.09 ± 32.57♦#	17.19 ± 5.03♦#	190.32 ± 37.83♦#	0.01 ± 0.02♦#
F	34.578	37.619	12.624	23.684	19.463
p	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001

*: Compared with that before operation ($p < 0.05$). ♦: Compared with that at 24 h after operation ($p < 0.05$). #: Compared with that at 48 h after operation ($p < 0.05$).

ferent time after operation, and there was no statistically significant difference between single group and double group. The time and interaction between the two groups had no statistically significant differences (Table VI).

Discussion

In 1958, the first ventricular fixed frequency pacemaker (VOO) was invented and the four-chamber pacemaker (DDTAV) has been used clinically nowadays, so the cardiac pacemaker has been developing for more than half a century. During this course, the risks of right auricle and RVA pacing have been gradually recognized by people, but the benefits of the special parts in heart (high atrial septum, low atrial septum, right ventricular outflow tract septum and right ventricular inlet septum, etc.) are advocated by the majority of clinicians. In this context, these special pacing parts are particularly important. Active fixed (spiral) electrodes are born at the right moment, making tremendous contributions to clinical research on multi-site cardiac pacing. The implantation of pacing electrodes during the implantation of cardiac pacemaker is a critical step in the entire operation, which not only plays an important role in the life quality of patients, but also has a critical influence on the service life of pacemaker. Pacing threshold, perceived P/R wave amplitude and electrode impedance, are three parameters in testing whether the pacing electrode is in place in the pacemaker implantation, among which the pacing threshold is the most important. The implantation site directly affects the pacing threshold, which is also the most

Table VI. Myocardial enzyme levels in the two groups.

Number of active electrode		MYO (ng/mL)	CK (U/L)	CK-MB (U/L)	LDH (U/L)	cTnI (ng/mL))
Before operation	Single group	58.02 ± 21.99	62.69 ± 33.68	16.58 ± 9.28	185.97 ± 49.64	0.01 ± 0.01
	Double group	59.21 ± 23.65	74.13 ± 30.50	16.97 ± 8.39	210 ± 52.54	0.01 ± 0.01
24 h	Single group	71.46 ± 37.60	94.70 ± 47.28	18.06 ± 7.69	214.97 ± 41.39	0.06 ± 0.04
	Double group	80.69 ± 36.19	103.43 ± 49.34	19.37 ± 9.86	215.35 ± 52.59	0.05 ± 0.05
48 h	Single group	55.49 ± 21.47	94.26 ± 51.48	18.35 ± 6.48	213.70 ± 31.98	0.04 ± 0.16
	Double group	58.92 ± 18.37	93.53 ± 48.42	17.59 ± 8.27	218.96 ± 42.96	0.04 ± 0.09
72 h	Single group	56.64 ± 16.35	63.58 ± 25.58	16.52 ± 3.89	193.59 ± 31.69	0.02 ± 0.37
	Double group	56.08 ± 17.23	64.24 ± 20.83	16.34 ± 2.42	190.64 ± 28.05	0.01 ± 0.18
	F time	33.273	30.269	11.495	17.758	18.483
	p time	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001
	F group	0.723	0.628	4.091	0.831	0.037
	p group	0.289	0.390	0.051	0.274	0.892
	F interaction	1.042	1.83	1.638	3.984	0.538
	p interaction	0.47	0.429	0.158	0.134	0.629

important reason for the longer operation time[7]. The modern view is that the pacing threshold is affected by the following factors: (a) *physiological factors*: experiments[8] show that sympathetic tension is inversely proportional to the pacemaker threshold; in other words, the decreased sympathetic tension can increase the pacing threshold by about 33%. (b) *Antiarrhythmic drugs (AAD)*: clinical studies[9] have confirmed that Class-I AAD can significantly increase the pacing threshold; in addition, the pacing threshold can also be affected by the body's response to drugs and electrophysiological effect, etc. (c) *Myocardial factors*: it used to be reported that patients with acute myocardial infarction have small response to the electrical stimulation due to local necrosis of myocardial tissues and no good electrophysiological characteristics, so higher pacing energy is needed to make the stimulation site receive the electrical pulse, causing pacemaker reaction[10,11]. Therefore, there is an increase in atrial or ventricular pacing threshold in different degrees after the implantation of pacemaker. It is reported[12] that the pacing threshold in patients with cardiac pacemaker is significantly decreased after hyperbaric oxygen therapy compared with that before treatment. (d) *Electrolyte imbalance:* elevated potassium in cells can raise the threshold temporarily, and the elevated extracellular sodium can also increase the threshold, but more significantly than the former; besides, increasing the carbon dioxide partial pressure and mildly decreasing the oxygen partial pressure can increase the pacing threshold. (e) *Electrical conversion*: it used to be reported that single-polar pacing threshold will be increased to varying degrees in patients with tachyarrhythmia after the *in vitro* high-energy electrical conversion, while the double-polar threshold is not increased[13].

This study showed that female sex, age, smoking, drinking, coronary heart disease, diabetes mellitus and hyperlipidemia have a certain impact on pacing threshold in a short-term after the implantation of active fixed electrode lead of cardiac pacemaker. After the implantation of active electrode lead, the levels of myocardial enzymes (MYO, CK, CK-MB, LDH and cTNI) in patients were increased gradually and reached the peak at 24 h, which were significantly higher than those before operation. The levels were decreased at 72 h with statistically significant differences. So, the damage of the implantation of active fixed electrode lead to the myocardium remains unclear, there were no statistically significant differences in the myocardial enzyme levels between single group and double group, and myocardial damage had no correlation with the number of active fixed electrode lead. Due to the smaller sample size, the effect of local myocardial tissues at the implanted site of active electrode on the results cannot be excluded, and the above factors are not analyzed and studied.

Conclusions

Female sex, age, smoking, drinking, coronary heart disease, diabetes mellitus and hyperlipidemia are independent influencing factors of pacing thresholds in a short term after the implantation of active fixed electrode lead of cardiac pacemaker. The implantation of active electrodes can raise the myocardial enzyme indexes compared with those before operation, but will fall to the preoperative or normal, near normal levels at 72 h after operation. The implantation of active electrodes is safe and feasible for myocardial tissues.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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