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Characteristics of ventricular electrophysiology in a right ventricular rapid pacing - induced canine heart failure model

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[**ABSTRACT**]　**AIM**: To research the characteristics of ventricular electrophysiology in right ventricular rapid pacing - induced congestive heart failure (CHF) dogs. **METHODS**: Dogs ($n = 16$) were randomly divided into 2 groups: the control ($n = 7$) and the CHF group ($n = 9$) induced by rapid right ventricular pacing at 240 pulse · min^{-1} for 4 to 5 weeks. The electrophysiologic parameters were evaluated by the technique of standard electric stimulation and monophasic action potential (MAP) recording. **RESULTS**: (1) Ventricular effective refractory period (VERP), ventricular MAP duration ($MAPD_{90}$), ventricular late repolarization duration (VLRD) and intra - ventricular conduction time (IVCT) were prolonged by 26% ($P < 0.01$), 43% ($P < 0.01$), 318% ($P < 0.05$), and 19% ($P < 0.01$), respectively in CHF group. (2) The ratio of VERP to $MAPD_{90}$ (VERP/$MAPD_{90}$) was decreased by 13% ($P < 0.05$) in CHF group. (3) The dispersion of ventricular recovery time (VRT - D) was increased by 185% ($P < 0.01$) in CHF group. (4) The ventricular fibrillation threshold (VFT) was decreased by 48% ($P < 0.01$) in CHF group. **CONCLUSION**: The abnormal electrophysiological changes in the CHF condition may be contributing factors of lethal ventricular arrhythmias and sudden cardiac deaths in CHF.

[**KEY WORDS**]　Heart failure, congestive; Electrophysiology; Action potentials; Ventricular fibrillation; Hemodynamics

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Congestive heart failure (CHF) is a complex pathophysiologic syndrome. Complex ventricular arrhythmias are noted in approximately 90% of patients with CHF. Sudden cardiac death (SCD) as the result of lethal ventricular arrhythmias is one of the main causes of death in CHF patients. The changes of ventricular electrophysiologic characters in the developing course of CHF are the possible mechanisms of genesis of ventricular arrhythmias[1]. The ventricular electrophysiologic abnormalities specifically related to CHF are not clear. Recent studies have demonstrated that CHF model induced by ventricular rapid pacing in dogs is an ideal one which is very similar to non - ischemic dilated cardiomyopathy in many aspects, including clinical features, hemodynamics, changes of cardiac function, neurohumoral compensations, and pathological changes of the heart[2-4]. The objectives of this study were to conduct a systematic *in vivo* study on the ventricular electrophysiologic changes associated with the development of CHF in this model.

MATERIALS AND METHODS

1　Materials

Sixteen adult mongrel dogs of either sex (from the Experimental Animal Center of Sun Yat - sen University, Grade Ⅰ), weighing (13.7 ± 1.8) kg, were randomly divided into two groups: the control ($n = 7$) and the CHF group ($n = 9$) induced by right ventricular rapid pacing at 240 pulse · min^{-1} for 4 to 5 weeks before electrophysiologic studies. The experimental protocols were carried out complying with the guideline of the local ethics committee.

2　CHF model

The dogs were anesthetized with ip 3% sodium pentobarbital 30 mg · kg^{-1}. The right femoral vein was cannulated for infusing 5% glucose in normal saline 500 mL

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with sodium benzylpenicillin 4.8 million units. A unipolar pacemaker lead (from Guangdong Kuangming Pacemaker Co.) was placed in the right ventricular apex under fluroscopy (Gentle Merate Co.) via the left external jugular vein. The pacing threshold was 0.3 – 1.5 V, the amplitude of R wave was 4 – 10 mV and the resistance was 0.3 – 1.0 kΩ. A small subcutaneous pocket was created between the scapulae for the implantation of the pacemaker generator (Guangzhou Radio Research Institute). The proximal end of the pacemaker lead was connected to pacemaker generator through a subcutaneous canal. The pacemaker generator was set at 240 pulse · min^{-1}, 5.0 V, and pulse width 0.5 ms. The electrophysiologic study was made 4 – 5 weeks after right ventricular rapid pacing. Serum electrolytes (K^+, Na^+, Cl^-, Ca^{2+}, Mg^{2+}) concentrations were monitored postoperatively. The surface ECG was checked weekly to assure constant pacing. The animals were kept under close monitoring daily in general appearance, appetite, activity and respiratory rate.

3 Hemodynamics

A Swan – Ganz catheter was passed through right atrium, right ventricle and into pulmonary artery via the external jugular vein. The following hemodynamic parameters were measured with a Spectramed P23XL transducer on Marquette Transcope 12 monitor (Marpuette Co.): right atrial pressure (RAP), right ventricular pressure (RVP), pulmonary arterial pressure (PAP), and pulmonary capillary wedge pressure (PCWP) by balloon occlusion. Cardiac output (CO) was determined by the thermodilution technique. A 6F catheter was inserted into the right femoral artery to measure the arterial blood pressure (ABP). Stroke volume (SV) was calculated as CO/heart rate (HR), cardiac index (CI) as CO/body weight (BW), and total peripheral resistance (TPR) as mean ABP/CI × 100[4]. The hemodynamic parameters were measured under closed chest before surgery and electrophysiologic study, 30 min after cessation of pacing in CHF groups.

4 Electrophysiologic study

4.1 Animal preparation After being anesthetized with the method used in making CHF model, the dogs were intubated and mechanically ventilated (Shanghai Medical Instrument Factory, China) with humidified air at a rate of 10 – 12 breath · min^{-1} and tidal volume of 12 to 13 mL · kg^{-1}. A 6F catheter was placed in the right femoral vein for infusion during the operation (about 1 L). The heart was exposed through a median sternotomy and cradled in the pericardium. A pair of stainless steel – wire electrodes (diameter 125 μm, 5 mm apart) were inserted into the right atrial appendage for cardiac pacing. Two pairs of electrodes were inserted into right ventricular outflow tract, right ventricular anterior wall, left ventricular anterior wall, left ventricular lateral wall and the apex, respectively for cardiac pacing and recording. Standard lead Ⅱ ECG, ventricular bipolar electrograms, together with ventricular epicardial MAP were recorded simultaneously using a 7 – channel, ink – jet recorder (Mingograf 7, Siemens, Sweden) at a paper speed of 100 mm · s^{-1}. Ventricular bipolar electrograms were subjected to band – pass filtering (50 – 500 Hz). MAP was recorded using an unipolarizable contact electrode in conjunction with a DC – coupled differential preamplifier. The electrophysiologic studies were begun 30 min after cessation of pacing in CHF groups. Each experiment lasted no more than 5 h.

4.2 Electrophysiologic measures ① Sinus cycle length (SCL). ② Intra – ventricular conduction time (IVCT). It was represented by QRS duration of ECG. ③ The ventricular effective refractory period (VERP). VERP was measured by programmed stimulation (Medtronic 5325, USA) at twice diastolic pacing threshold, with duration of 1.8 ms during basic ventricular drive. The longest S1 S2 interval that did not evoke a ventricular depolarization was defined as the VERP[5]. ④ Ventricular activation time (VAT). VAT was the interval from the onset of the standard lead Ⅱ ECG QRS complex to the point where the rapid deflection of the largest deflection of the local ventricular bipolar electrograms crossed the baseline[6]. ⑤ Ventricular recovery time (VRT). VRT was the sum of local VAT and local VERP at each ventricular site at the same cycle length[6]. ⑥ Dispersion of VRT (VRT – D). VRT – D was defined as the difference between the earliest and latest VRT (right ventricular outflow tract, right ventricular anterior wall, left ventricular anterior wall, left ventricular lateral wall, and apex) at the same cycle length. Parameters ② – ⑥ were measured during atrial or ventricular pacing at cycle lengths of

375 ms and 400 ms, respectively.

4.3 Ventricular MAP duration (MAPD)[7] ① $MAPD_{90}$ was the interval, along a line horizontal to the diastolic baseline, from the onset of activation to the 90% repolarization level. ②Ventricular late repolarization duration (VLRD, the difference of local ventricular $MAPD_{90}$ and VERP at the same cycle length). ③ Ratio of VERP to $MAPD_{90}$ (VERP/$MAPD_{90}$). The above parameters were measured during atrial or ventricular pacing at cycle length of 375 ms and 400 ms.

4.4 Experimental ventricular tachyarrhythmias (VTA) Inducibility of VTA (including non-sustained ventricular tachycardia, sustained ventricular tachycardia and ventricular fibrillation) was determined using programmed stimulation at basic pacing cycle lengths of 400 ms, 375 ms, 300 ms and 250 ms and up to three extrastimuli. The second extrastimulus (S3) was started with the S1S2 interval fixed at 30 ms longer than VERP. The S2S3 interval was set at 80% of basic pacing cycle length, with use of 5 ms scanning decrements until S3 failed to induce activation. If double extrastimulation failed to induce arrhythmia, a third extrastimulus (S4) was added with a similar scanning procedure[5].

4.5 Ventricular fibrillation threshold (VFT) VFT was measured by a train of constant current pulses that scanned the T wave at a stable right atrial pulses cycle length of 400 ms[8]. VFT was defined as the least amount of current required to elicit ventricular fibrillation. Ventricular fibrillation was terminated 15s after its onset by DC shock [(5-15) W·S] delivered directly to the surface of the heart.

5 Pathologic evaluation and heart morphology

Pericardial effusion, pleural effusion and the change of the lungs were evaluated qualitatively after sternotomy. A postmortem cardiac examination was conducted and the following indexes were measured: heart weight (HW), ratio of HW to BW (HW/BW), free wall thickness of the left and right ventricles (LVFWT and RVFWT), left ventricular longitudinal diameter (LVLD, the left ventricular diameter from the atrioventricular valvular ring to the apex), right ventricular transverse diameter (RVTD, the right ventricular diameter at the halfway from the atrioventricular valvular ring to the apex), and left ventricular volume[$LVV = (\pi \times LVLD \times RVTD)^2 \div 6$]. LVFWT and RVFWT were measured at the point on the free ventricular wall halfway from the atrioventricular valvular ring to the apex[2]. Fresh left and right ventricular tissue was fixed in formalin and paraffin sections were stained with hematoxylin and eosin (HE).

6 Statistical analysis

Data were expressed as $\bar{x} \pm s$. The difference between baseline study and restudy was analyzed with the paired t test. The student t test was used to compare mean values between the control and the paced group. The relation between electrophysiologic variables and hemodynamic, heart morphologic parameters was evaluated by analysis of stepwise regression. Two-sided tests were used.

RESULTS

1 Clinic, hemodynamics, and pathology in CHF dogs

1.1 Clinic All dogs had clinical characteristics of CHF, such as anorexia, hypokinetics, tachypnea, and pedal edema 4-5 weeks after right ventricular rapid pacing. Respiratory rate increased from (18 ± 1) times · min^{-1} to (40 ± 3) times · min^{-1} ($P < 0.01$) with many moist rales. BW did not change much [(13.7 ± 1.8) kg *vs* (13.6 ± 1.0) kg, $P > 0.05$].

1.2 Hemodynamics There was an increase in the mean RAP (mRAP), mean RVP (mRVP), mean PAP (mPAP), and mean PCWP (mPCWP) ($P < 0.01$) with decrease of CO, CI, and SV ($P < 0.01$) 4-5 weeks after right ventricular rapid pacing (Tab 1). TPR was increased in the CHF dogs *vs* controls [(10 133 ± 3 733) kPa · min^{-1} · kg^{-1} · L^{-1} *vs* (4 800 ± 1 333) kPa · min^{-1} · kg^{-1} · L^{-1}, $P < 0.01$].

Tab 1 Hemodynamic parameters in dogs before and after right ventricular rapid pacing (240 pulses · min^{-1} for 4-5 weeks. $\bar{x} \pm s$. $n = 9$)

	Before pacing	After pacing
mRAP(kPa)	0.187 ± 0.200	1.120 ± 0.413*
mRVP(kPa)	0.880 ± 0.680	1.960 ± 0.960*
mPAP(kPa)	1.093 ± 0.773	2.307 ± 1.093*
mPCWP(kPa)	0.227 ± 0.227	1.173 ± 0.627*
CO(L · min^{-1})	3.900 ± 0.700	1.500 ± 0.300*
CI(L · min^{-1} · kg^{-1})	0.289 ± 0.057	0.109 ± 0.027*
SV(mL · $beat^{-1}$)	23.200 ± 4.900	10.700 ± 2.100*

*$P < 0.01$ *vs* before pacing.

1.3 Pathology All CHF dogs had pulmonary congestion and edema. Following abnormal findings were found in CHF group: pericardial effusion and pleural effusion in all dogs, ascites in 6 dogs. There were increases of HW ($P<0.01$), HW/BW ($P<0.05$), LVLD ($P<0.01$), RVTD ($P<0.01$), and LVV ($P<0.01$) with decrease of RVFWT ($P<0.05$) in CHF dogs. There was a tendency of decreasing in LVFWT ($P>0.05$) in CHF dogs (Tab 2). Histologic examination of ventricle revealed cardiac cell edema, fat degeneration, focal myocardial necrosis, interstitial edema, neutrophils and lymphocytes infiltration, and small vascular congestion.

Tab 2 Effects of right ventricular rapid pacing (240 pulses · min^{-1} for 4 – 5 weeks) on heart anatomy in dogs ($\bar{x} \pm s$)

	Control($n=7$)	CHF($n=9$)
HW(g)	121.0 ± 28.0	145.0 ± 13.0**
HW/W(g · kg^{-1})	9.4 ± 1.5	11.5 ± 1.4*
LVFWT(mm)	13.3 ± 2.0	10.7 ± 2.3
RVFWT(mm)	7.4 ± 0.9	5.4 ± 1.5*
LVLD(mm)	42.3 ± 5.6	55.3 ± 4.0**
RVTD(mm)	43.0 ± 3.5	51.6 ± 4.8**
LVV(mL)	38.6 ± 11.2	77.8 ± 16.5**

* $P<0.05$, ** $P<0.01$ *vs* control.

2 Cardiac electrophysiologic parameters in CHF dogs

2.1 SCL and IVCT As compared with that before pacing, SCL and IVCT were prolonged by 31% [(440 ± 81) ms *vs* (337 ± 38) ms, $P<0.05$] and 19% [(69 ± 4) ms *vs* (59 ± 6) ms, $P<0.01$] respectively in the CHF dogs (Tab 3).

2.2 VERP, VAT, VRT, and VRT – D As compared with the controls, VERP, VAT, and VRT were prolonged by 26% ($P<0.01$), 100% ($P<0.05$), and 32% ($P<0.01$), respectively and VRT – D was increased by 185% in the CHF group ($P<0.01$) (Tab 3).

2.3 $MAPD_{90}$, VLRD, and $VERP/MAPD_{90}$ As compared with the controls, $MAPD_{90}$ and VLRD were prolonged by 43% ($P<0.01$) and 318% ($P<0.05$), respectively and $VERP/MAPD_{90}$ was decreased by 13% in the CHF dogs (Tab 3).

2.4 Incidence of experimental VTA In control group, ventricular fibrillation was only induced in one dog. Three dogs were induced ventricular fibrillation in CHF group. There was no incidence of other VTAs in either group. There was no significant difference in the incidence of experimental VTA between control and CHF group ($P>0.05$).

2.5 VFT VFT was decreased by 44% in the CHF group *vs* controls ($P<0.01$) (Tab 3).

Tab 3 Changes of electrophysiologic properties in CHF dogs ($\bar{x} \pm s$)

	Control($n=7$)	CHF($n=9$)
SCL(ms)	337 ± 38	440 ± 81*
IVCT(ms)	59 ± 6	69 ± 4**
VERP(ms)	169 ± 12	213 ± 38**
AT(ms)	10 ± 5	20 ± 10*
RT(ms)	178 ± 15	234 ± 44**
RT – D(ms)	13 ± 4	37 ± 15**
$MAPD_{90}$(ms)	169 ± 24	242 ± 37**
VLRD(ms)	11 ± 15	46 ± 41*
$VERP/MAPD_{90}$	0.945 ± 0.084	0.831 ± 0.169*
VFT(mA)	31.8 ± 4.9	18.0 ± 10.4**

* $P<0.05$, ** $P<0.01$ *vs* control. All parameters were measured at paced cycle length of 375 ms except SCL at sinus rhythum and $VERP/MAPD_{90}$ and VFT at 400 ms.

3 Correlation with hemodynamics and heart morphology

VERP positively correlated with mRVP ($r=0.3340$, $P<0.05$) and RVTD ($r=0.4055$, $P<0.05$). VRT positively correlated with mRVP ($r=0.4733$, $P<0.01$). VRT – D correlated inversely with CI ($r=-0.4739$, $P<0.01$) and LVFWT ($r=-0.5149$, $P<0.01$).

4 Serum electrolytes concentration

There were no marked changes in serum electrolytes concentrations between baseline study and restudy in two groups. There were no significant differences between control and CHF groups in electrolytes concentrations.

DISCUSSION

$MAPD_{90}$ and VERP were obviously prolonged in CHF in this study. This is well consistent with previous experimental study and clinical study[9,10]. Over load of intracellular Ca^{2+} resulting from the reduction of intracellular Ca^{2+} transport in CHF may be one of the main causes of ventricular repolarization prolongation[11].

Our study showed that $VERP/MAPD_{90}$ was decreased in CHF dogs. This suggested that $MAPD_{90}$ prolonged much more significantly than VERP in CHF dogs. Marked prolongation of VLRD was found in CHF

dogs in our study. Prolongation of VLRD facilitates the occurrence of reentry which results from prolongation of phase Ⅲ block period, and early after depolarization (EAD). It has been reported that EAD was recorded with prolongation of action potential duration in CHF models and patients[12].

We noted that VRT - D was increased and IVCT were prolonged in CHF dogs. The decreased resting membrane potential and slowed V_{max} of phase 0 of action potential may contribute to conduction slowing[9,13]. It has been established that reentry is facilitated when VRT - D was increased. In addition, prolongation of IVCT results in extension of excitable gap, which facilitates the occurrence of reentry.

Li et al[9] reported that the incidence of ventricular fibrillation was increased in CHF dogs. We also noted that VFT in CHF dogs was decreased. Overexcitation of sympathetic nerve and heterogeneity of sympathetic innervation in myocardium may contribute to the decrease of VFT.

In this study, the incidence of experimental VTA was similar in both groups. It has been reported that the inducibilities of VTA by programmed stimulation in non - ischemic cardiomyopathy patients were lower than that in ischemic cardiomyopathy . It has been established that reentry is the common initiating mechanism of ventricular arrhythmia in both cardiomyopathy, but the reentry substrates are different. Reentry circuits in patients with ischemic cardiomyopathy are often relatively large and stable, but in patients with non - ischemic cardiomyopathy, the electrophysiologic characters are affected by many variable factors that produce non - fixed reentry circuit. The ventricular tachycardia is not inducible easily by programmed ventricular electrical stimulation.

Some electrophysiologic parameters correlated with hemodynamics and heart morphology parameters in our study. It has been demonstrated that there is contraction - excitation feedback in heart. This could be an important potential cause of arrhythmias in patients with CHF since a direct electrophysiological change may be induced by alterations in ventricular size, pressure, or function, factors common to all types of CHF regardless of aetiology[14].

Our study suggested that abnormal electrophysiological changes might be contributing factors of lethal ventricular arrhythmias and sudden cardiac deaths in CHF.

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右心室快速起搏致心力衰竭犬的心室电生理特性

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［摘　要］ **目的**：研究右心室快速起搏致充血性心力衰竭（CHF）犬心室电生理特性。**方法**：16 只犬随机分为正常对照组（$n=7$）和 CHF 组（$n=9$），应用右心室快速起搏（240 pulse · min^{-1}）4 - 5 周制作 CHF 犬模型，应用心脏电刺激和单相动作电位（MAP）记录技术测定心室生理指标。**结果**：（1）CHF 组心室有效不应期、心室 MAP 时程、复极后期及传导时间均延长，分别延长 26%（$P<0.01$）、43%（$P<0.01$）、318%（$P<0.05$）和 19%（$P<0.01$）；（2）CHF 组心室有效不应期与 MAP 时程的比值减小 13%（$P<0.05$）；（3）CHF 组兴奋恢复时间离散性增加 185%（$P<0.01$）；（4）CHF 组室颤阈值降低 48%（$P<0.01$）。**结论**：CHF 异常的心室电生理特性可能是导致恶性心律失常及心脏性猝死发生的基础。

［关键词］ 心力衰竭，充血性；电生理学；动作电位；心室颤动；血流动力学
［中图分类号］ R363　　［文献标识码］ A

中国病理生理学会炎症发热感染低温专业委员会和中医专业委员会第十一届全国联合学术会议（会议纪要）

中国病理生理学会炎症发热感染低温专业委员会和中医专业委员会第十一届全国联合学术会议于 2007 年 8 月 20 日 - 8 月 24 日在河北承德顺利召开。本次会议共收到科研论文 71 篇，其中参加青年优秀论文评选 13 篇，大会发言 27 篇，参加会议的代表 57 人，来自于 13 个省、市。

大会进行了青年优秀论文的报告会，并评选出 8 篇青年优秀论文予以奖励，其中一等奖 2 篇、二等奖 3 篇、三等奖 3 篇（名单见附件 1）。

会议收到的学术论文涉及以下几方面：1. 炎症、发热及感染病理过程的分子机制；2. 中医药治疗心、脑血管疾病的病理生理的基础研究；3. 肝、肾纤维化发生分子机制以及中药干预作用环节的研究；4. 内毒素休克甘氨酸干预机制，以及育亨宾和小檗碱联合应用抗休克的分子机制；5. 中医药对皮肤溃疡愈合的细胞和分子机制研究；6. 中药冬棱草甲素促进肿瘤细胞和巨噬细胞的吞噬效应的信号通路，以及肿瘤与自噬新领域的研究；7. 中西医结合治疗白内障，从白内障模型的建立到中药治疗机制和临床应用作了系列的研究。以上内容在会上进行交流，与会代表受益很大。

本次会议还对炎症发热感染低温专业委员会的组成人员进行了调整，通过民主推荐和大会无记名投票选举产生了第五届炎症发热感染低温专业委员会，选举陆大祥为主任委员，黄启福、颜亮、黄宁、郝钰为副主任委员（全体委员名单见附件 2）。

经中医专业委员会讨论通过增补天津中医药大学范英昌教授、湖南中医药大学雷久士教授和广州中医药大学杜标炎教授为专业委员会委员。

本次全国学术会议的特点是：1. 提交大会的论文数量较多、水平高，涉及基础研究和临床研究；2. 参与的青年优秀论文较多，水平较高，青年学者正在成长；3. 与会者态度认真，遵守会纪，自始至终参加会议；4. 承德医学院各级领导及病理生理教研室对会议十分重视，对会议的组织工作做出了贡献。

大会对青年学者提出希望：青年学者要增强社会责任感和使命感，医学科学是神圣的事业，希望在座的青年学者继续努力工作，为这一神圣的事业贡献自己的力量。

中国病理生理学会炎症发热感染低温专业委员会和中医专业委员会

2007 年 8 月 24 日于承德

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