定脂质过氧化产物. **结果**: *Gb*E 减轻 LPC 对 ACh 舒血管效应的抑制和防止主动脉内 MDA 含量的增加以及对培养的内皮细胞的损伤. *Gb*E 还明显增加培养的内皮细胞的前列环素的水平. **结论**: *Gb*E 保护内皮细胞免受 LPC 损伤是由于降低脂质过氧化和促进前列环素的合成或释放.

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Effects of amiodarone on cardiac electrophysiology in right ventricular rapid pacing-induced heart failure dogs

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KEY WORDS amiodarone; congestive heart failure; electrophysiology; action potentials; ventricular fibrillation; hemodynamics

AIM: To study the effects of amiodarone (Ami) on cardiac electrophysiologic properties and ventricular fibrillation threshold (VFT) in right ventricular rapid pacing-induced congestive heart failure (CHF) dogs. **METHODS**: Dogs (n = 25) were randomly allocated into 3 groups: A) control group; B) CHF group induced by right ventricular rapid pacing (4 pulses·s^{-1}) for 4 - 5 wk; C) CHF models *po* Ami 300 mg·d^{-1} for 4 - 5 wk. The electrophysiologic parameters and VFT were evaluated by electric stimulation and monophasic action potential (MAP) recording. **RESULTS**: In CHF models, ventricular MAP duration ($MAPD_{90}$), ventricular late repolarization duration (VLRD), and intraventricular conduction time (IVCT) were prolonged by 43 %, 318 %, and 19 %, respectively; the ratio of ventricular effective refractory period (VERP) to $MAPD_{90}$ (VERP/$MAPD_{90}$) and VFT were decreased by 13 % and 48 % respectively; the dispersion of ventricular recovery time (RT-D) was increased by 185 %. In CHF models, Ami had no effects on ventricular $MAPD_{90}$, but increased VERP/$MAPD_{90}$, IVCT, and VFT by 15 %, 10 %, and 67 %, respectively, shortened VLRD by 87 %; and decreased RT-D by 87 %. Ami had no significant influences on the hemodynamic parameters of the CHF dogs. **CONCLUSION**: Ami normalizes the cardiac electrophysiologic properties in CHF dogs.

One of the focuses on the treatment of congestive heart failure (CHF) is to search for effective measures in preventing sudden cardiac death (SCD). Amiodarone (Ami), a class Ⅲ anti-arrhythmic agent, is attracting attention in the prevention of SCD in patients with CHF because of its highly effective and wide-spetrum anti-arrhythmic actions, low incidence of proarrhythmia, mild or no negative inotropic properties, and low incidence of side effects given in lower doses. Low-dose Ami is effective in suppressing ventricular arrhythmias, improving ventricular function, and reducing the mortality of patients with CHF[1,2]. The effects of Ami on cardiac electrophysiologic properties during CHF have not been thoroughly investigated. Recent studies have demonstrated that the CHF model induced by ventricular rapid pacing in dogs is an ideal one which is very similar to the clinical non-ischemic dilated cardiomyopathy in many aspects, including hemodynamics, changes of cardiac function, neurohumoral compensations, and pathological changes in heart[3,4]. This model has been applied widely in researches. The objectives of this study were to conduct a systematic *in vivo* study on the effects of low-dose Ami on cardiac electrophysiologic properties and ventricular fibrillation threshold (VFT) in the CHF model.

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MATERIALS AND METHODS

Materials Ami hydrochloride was kindly provided by Sanofi Winthrop Inc (batch No E-31). Twenty-five adult mongrel dogs of either sex (from the Experimental Animal Center of Sun Yat-Sen University of Medical Sciences), weighing 13.2 ± 1.6 kg, were randomly allocated into 3 groups: A) normal controls (n = 7); B) CHF models induced by right ventricular rapid pacing (4 pulses · s^{-1}) for 4 – 5 wk before electrophysiologic studies (n = 9); C) CHF models *po* (fed the dogs with meatball with Ami tablets inside before each breakfast) Ami 300 mg · d^{-1} for 4 – 5 wk, beginning on d 1 after pacemaker being implanted (n = 9).

CHF model The dogs (Experimental Animal Center, Sun Yat-Sen University of Medical Sciences, Grade 1) were anesthetized with ip 3 % pentobarbital sodium 30 mg · kg^{-1}[5]. The right femoral vein was cannulated for infusing 5 % glucose in normal saline 500 mL + benzylpenicillin sodium 4.8 MU. A unipolar pacemaker lead (from Guangdong Xinhui Kuangming Pacemaker Co) was placed in the right ventricular apex under a fluroscope (Gentle Merate Co) via the left external jugular vein. The pacing threshold was 0.3 – 0.5 V, the amplitude of R wave was 4 – 10 mV and the resistance was 0.3 – 1 kΩ. A small subcutaneous pocket was created between the scapulae for the implantation of the pacemaker generator (Guangzhou Radio Res Inst). The pacemaker lead was connected to pacemaker generator through a subcutaneous canal. The pacemaker generator was set at 4 pulses · s^{-1}, 5.0 V, and pulse width 0.5 ms[3,4]. The electrophysiologic study was made 4 – 5 wk after right ventricular rapid pacing.

Hemodynamics A Swan-Ganz catheter was passed through right atrium and right ventricle into pulmonary artery via the external jugular vein. The following hemodynamic parameters were measured with a Spectramed P23XL transducer on Marquette Transcope 12 monitor: 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[3]. The hemodynamic parameters were measured under closed chest before the implantation of pacemaker and electrophysiologic study, 30 min after cessation of pacing in CHF and CHF + Ami groups separately.

Electrophysiologic study

Dogs After being anesthetized with ip 3 % pentobarbital sodium 30 mg · kg^{-1}, the dogs were intubated and mechanically ventilated (Shanghai Medical Instrumenu Factory) with humidified air. A 6F catheter was placed in the right femoral vein for infusion (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) at a paper speed of 100 mm · s^{-1} (Fig 1).

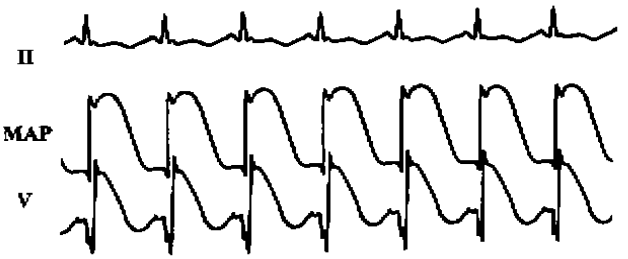

Fig 1. Simultaneous recordings of surface ECG, ventricular bipolar electrograms (V), and monophasic action potentials (MAP).

Ventricular bipolar electrograms were subjected to band-pass filtering (50 – 500 Hz). MAP were recorded using a nonpolarizable contact electrode in conjunction with a DC-coupled differential preamplifier. The electro-

physiologic studies were begun 30 min after cessation of pacing in CHF and CHF + Ami groups. Each experiment lasted < 5 h.

Electrophysiologic measurements (1) Sinus cycle length (SCL). (2) Intra-ventricular conduction time (IVCT). It was represented by QRS duration of ECG. (3) The ventricular refractory effective period (VERP). VERP was measured by programmed stimulation (Medtronic 5325) at twice diastolic pacing threshold, with a duration of 1.8 ms during basic ventricular drive. The longest S_1S_2 interval that did not evoke a ventricular depolarization was defined as the VERP[6]. (4) Ventricular activation time (AT)[7]. (5) Ventricular recovery time (RT)[7]. (6) Dispersion of ventricular RT (RT-D). RT-D was defined as the difference between the earliest and latest ventricular RT (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.

Ventricular MAP duration (MAPD)[8] (1) $MAPD_{90}$ was the interval, along a line horizontal to the diastolic baseline, from the onset of activation to the 90 % repolarization level. (2) Ventricular 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.

Ventricular fibrillation threshold (VFT) VFT was measured by a train of constant current pulses that scanned the T wave at a stable atrial paced cycle length of 400 ms[5]. VFT was defined as the least amount of current required to elicit ventricular fibrillation.

Pathologic evaluation and heart morphology Pericardial effusion, pleural effusion and the change of the lungs were evaluated qualitatively after sternotomy. Postmortem examination: 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[3]. Fresh left and right ventricular tissue was fixed in formalin and paraffin sections were stained with hematoxylin and eosin (HE).

Serum Ami concentrations Serum Ami concentrations were measured using HPLC[9].

Statistical analysis Data were expressed as $\bar{x} \pm s$. Paired t test and one-way ANOVA were used.

RESULTS

Clinic, hemodynamics, and pathology in CHF dogs

Clinic All dogs had clinical characteristics of CHF, such as anorexia, hypokinetics, tachypnea, and pedal edema 4 - 5 wk 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 (from 13.7 ± 1.8 kg to 13.6 ± 1.0 kg, $P > 0.05$).

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 wk after right ventricular rapid pacing (Tab 1). TPR was increased in the CHF dogs *vs* controls (10 133 ± 3 733 kPa · min · kg · L^{-1} *vs* 4800 ± 1333 kPa · min · kg · L^{-1}, $P < 0.01$).

Pathology All CHF dogs had pulmonary congestion and edema. Pericardial effusion, pleural effusion, and ascites were seen in most CHF dogs. There were increases of HW, HW/BW, LVLD, RVTD, and LVV ($P < 0.05$, $P < 0.01$) with decrease of RVFWT ($P < 0.05$). There was a tendency of decreasing in LVFWT ($P > 0.05$) (Tab 2). Histologic examination of ventricle revealed cardiac cell edema, fat degeneration, focal necrotic cardiac myofibers, interstitial edema, neutrophils and lymphocytes

Tab 1. Effects of right ventricular rapid pacing (4 pulses·s^{-1} for 4 – 5 wk) on hemodynamic parameters in dogs and effects of amiodarone (300 mg·d^{-1} *po* for 4 – 5 wk) on haemodynamic parameters in CHF dogs. $n = 9$, $\bar{x} \pm s$.
$^{c}P < 0.01$ *vs* before pacing. $^{d}P > 0.05$, $^{e}P < 0.05$ CHF + Ami group after pacing *vs* CHF group after pacing.

	Group	Before	After
HR/bpm	CHF	178 ± 18	136 ± 21^{c}
	CHF + Ami	170 ± 20	132 ± 20^{cd}
mRAP/kPa	CHF	0.19 ± 0.20	1.1 ± 0.4^{c}
	CHF + Ami	0.15 ± 0.29	0.75 ± 0.23^{ce}
mRVP/kPa	CHF	0.9 ± 0.7	2.0 ± 1.0^{c}
	CHF + Ami	1.1 ± 0.6	2.0 ± 0.7^{cd}
mPAP/kPa	CHF	1.1 ± 0.8	2.3 ± 1.1^{c}
	CHF + Ami	1.8 ± 0.7	2.7 ± 0.7^{cd}
mPCWP/kPa	CHF	0.2 ± 0.2	1.2 ± 0.6^{c}
	CHF + Ami	0.4 ± 0.3	1.08 ± 0.17^{cd}
CO/L·min^{-1}	CHF	3.9 ± 0.7	1.5 ± 0.3^{c}
	CHF + Ami	4.7 ± 0.7	1.7 ± 0.5^{cd}
CI/L·min^{-1}·kg^{-1}	CHF	0.29 ± 0.06	0.109 ± 0.027^{c}
	CHF + Ami	0.34 ± 0.06	0.14 ± 0.04^{cd}
SV/L·beat^{-1}	CHF	0.023 ± 0.005	0.0112 ± 0.0024^{c}
	CHF + Ami	0.027 ± 0.005	0.013 ± 0.003^{cd}
TPR/kPa·min·kg·L^{-1}	CHF	–	10 133 ± 3 733
	CHF + Ami	–	8 533 ± 2 000^{d}
SBP/kPa	CHF	–	14.0 ± 2.7
	CHF + Ami	–	15.8 ± 1.9^{d}
DBP/kPa	CHF	–	9.3 ± 2.1
	CHF + Ami	–	10.0 ± 2.4^{d}

Tab 2. Effects of right ventricular rapid pacing (4 pulses ·s^{-1} for 4 – 5 wk) on heart anatomy in dogs. $\bar{x} \pm s$.
$^{a}P > 0.05$, $^{b}P < 0.05$, $^{c}P < 0.01$ *vs* control. $^{d}P > 0.05$, $^{e}P < 0.05$ *vs* CHF.

	Control ($n = 7$)	CHF ($n = 9$)	CHF + Ami ($n = 9$)
HW/g	121 ± 28	146 ± 13^{c}	159 ± 14^{cd}
HW/BW/g·kg^{-1}	9.4 ± 1.5	11.5 ± 1.4^{b}	11.0 ± 1.2^{bd}
LVFWT/mm	13.3 ± 2.0	10.7 ± 2.3^{a}	11.2 ± 2.5^{ad}
RVFWT/mm	7.4 ± 0.9	5.4 ± 1.5^{b}	6.3 ± 1.5^{ad}
LVLD/mm	42 ± 6	55 ± 4^{c}	50 ± 4^{ce}
RVTD/mm	43 ± 4	52 ± 5^{c}	51 ± 3^{cd}
LVV/mL	39 ± 11	78 ± 17^{c}	68 ± 10^{cd}

infiltration, and vascular congestion.

Cardiac electrophysiologic parameters in CHF dogs

SCL and IVCT SCL and IVCT were prolonged by 31 % and 19 %, respectively in the CHF dogs *vs* controls ($P < 0.01$). There were insignificant changes in SCL in the treated group *vs* the CHF group ($P > 0.05$). IVCT was prolonged by 10 % ($P < 0.05$) (Tab 3).

Tab 3. Effects of amiodarone (300 mg·d^{-1} *po* for 4 – 5 wk) on cardiac electrophysiologic properties at paced cycle length of 375 ms except VERP/MAPD$_{90}$ and VFT at 400 ms and SCL at sinus rhythm in CHF dogs. $\bar{x} \pm s$.
$^{a}P > 0.05$, $^{b}P < 0.05$, $^{c}P < 0.01$ *vs* control.
$^{d}P > 0.05$, $^{e}P < 0.05$, $^{f}P < 0.01$ *vs* CHF.

	Control ($n = 7$)	CHF ($n = 9$)	CHF + Ami ($n = 9$)
SCL/ms	337 ± 38	440 ± 81^{b}	453 ± 81^{bd}
IVCT/ms	59 ± 6	70 ± 4^{c}	77 ± 9^{ce}
VERP/ms	169 ± 12	213 ± 38^{c}	206 ± 13^{cd}
AT/ms	10 ± 5	20 ± 10^{b}	18 ± 6^{bd}
RT/ms	178 ± 15	234 ± 44^{c}	224 ± 15^{cd}
RT-D/ms	13 ± 4	37 ± 15^{c}	18 ± 5^{af}
MAPD$_{90}$/ms	169 ± 24	242 ± 37^{c}	214 ± 11^{cd}
VLRD/ms	11 ± 15	46 ± 41^{b}	6 ± 19^{ae}
VERP/MAPD$_{90}$	0.95 ± 0.08	0.83 ± 0.17^{b}	0.95 ± 0.08^{ae}
VFT/mA	32 ± 5	18 ± 10^{c}	30 ± 8^{af}

VERP, AT, RT, and RT-D VERP, AT, and RT were prolonged by 26 %, 100 %, and 32 %, respectively and RT-D was increased by 185 % in the CHF group *vs* controls ($P <$

0.01). There were insignificant changes in VERP, AT, and RT ($P > 0.05$) with a decrease (87 %) in RT-D in the treated group *vs* the CHF group ($P < 0.01$). There was no marked difference in RT-D between the treated groups and controls ($P > 0.05$) (Tab 3).

$MAPD_{90}$, VLRD, and $VERP/MAPD_{90}$ $MAPD_{90}$ 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 *vs* controls. In the treated group, there was little prolongation in $MAPD_{90}$ ($P > 0.05$) with shortening (87 %) in VLRD ($P < 0.05$) and increase (15 %) in $VERP/MAPD_{90}$ ($P < 0.05$) *vs* the CHF group. There were little differences in VLRD and $VERP/MAPD_{90}$ between the treated groups and controls ($P > 0.05$) (Tab 3).

VFT VFT was decreased by 44 % in the CHF group *vs* controls ($P < 0.01$). In the treated group, VFT was increased by 67 % ($P < 0.01$) to an extent similar to that in controls ($P > 0.05$) (Tab 3).

Hemodynamic parameters in CHF dogs In the treated group, mRAP was decreased ($P < 0.05$) *vs* the CHF group. There were little changes in mRVP, mPAP, and mPCWP ($P > 0.05$). CO, CI, and SV tended to increase ($P > 0.05$) (Tab 1).

Serum Ami concentration Serum Ami level in the treated group was 1.3 ± 1.1 $mg \cdot L^{-1}$.

DISCUSSION

In this study, we successfully established CHF canine models by right ventricular rapid pacing for 4 – 5 wk. The clinical characteristics, hemodynamics and cardiac pathological features of the models were similar to the findings revealed in previous studies[3,4].

We noted in this study that $MAPD_{90}$ and VERP in CHF dogs were obviously prolonged. This is well consistent with the previous report[10]. Our study also showed that $VERP/MAPD_{90}$ was decreased in CHF dogs although VERP was prolonged. It is suggested that $MAPD_{90}$ prolonged much more than VERP in CHF dogs.

In this study Ami insignificantly prolonged $MAPD_{90}$ and VERP in CHF dogs, but increased $VERP/MAPD_{90}$, which is consistent with the previous report[11]. It suggested that a composite of time-dependent as well as voltage-dependent effects of Ami prolonged of VERP[11]. A mark prolongation in VLRD, which is composed of relative refractory period and suppernormal period, was observed in CHF dogs in this study. Ami shortened VLRD in CHF dogs.

We noted that ventricular RT-D was increased and both IVCT and AT were prolonged in CHF dogs. It has been established that reentry is facilitated by conduction from an area with a short RT to an area with a long RT, that is, a unidirectional block caused by prolonged RT. In addition, prolongation of ventricular conduction time results in extension of excitable gap, which facilitates the occurrence of reentry. In this study Ami significantly diminished RT-D in CHF dogs. It is suggested that Ami has the effect of rendering ventricular repolarization synchronously and homogeneously, which can finally eliminate unidirectional block and suppress the occurrence of reentry.

The incidence of ventricular fibrillation was increased in CHF dogs induced by right ventricular rapid pacing[10]. We also noted that VFT of CHF dogs was decreased while VFT of the treated group was increased. It suggested that Ami had antifibrillation effect.

Ami is a vasodilator with mild or no negative inotropic effect. In this study Ami had no negative influence on hemodynamics. It has been confirmed that low-dose Ami has beneficial effects on cardiac function in CHF patients[1,2].

In conclusion, this study showed that Ami increased $VERP/MAPD_{90}$ and VFT, prolonged IVCT, shortened VLRD, and decreased RT-D in CHF dogs. Ami normolizes the cardiac electrophysiologic properties without negative influence on the hemodynamic parameters in CHF dogs. It suggested that Ami had potential value on the prevention and treatment of ventricular arrhythmias and SCD in CHF.

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胺碘酮对快速右心室起搏致心力衰竭犬心室电生理特性的影响

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关键词 胺碘酮；充血性心力衰竭；电生理学；动作电位；心室纤颤；血液动力学

起搏

目的：研究胺碘酮（Ami）对快速右心室起搏（240 pulses·min^{-1}，4 – 5 wk）致心力衰竭（CHF）犬心室电生理特性及室颤阈值（VFT）的影响. **方法**：应用心脏电刺激及单相动作电位（MAP）技术测定心室电生理参数及 VFT. **结果**：CHF 犬心室电生理特性表现为：MAP 时程（$MAPD_{90}$）及复极后期（VLRD）延长. 有效不应期（VERP）与 $MAPD_{90}$ 的比值（$VERP/MAPD_{90}$）减小，兴奋恢复时间离散性（RT-D）增加，传导时间延长，VFT 降低；Ami 对 CHF 犬的心室电生理作用表现为：无明显延长 $MAPD_{90}$，增加 $VERP/MAPD_{90}$，缩短 VLRD，减小 RT-D，减慢传导速度，提高 VFT. **结论**：Ami 使 CHF 犬异常的心室电生理特性改变大致回复正常.