Electroacupuncture improves cardiac function and remodeling by inhibition of sympatheexcitation in chronic heart failure rats.

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Running head: The effects of electroacupuncture on chronic heart failure.

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Abstract

Chronic heart failure (CHF) is responsible for significant morbidity and mortality worldwide, mainly as a result of neurohumoral activation. Acupuncture has been used to treat a wide range of diseases and conditions. In this study, we investigated the effects of electroacupuncture (EA) on the sympathetic nerve activity, heart function and remodeling in CHF rats after ligation of the left anterior descending coronary artery. CHF rats were randomly selected to EA and control groups for acute and chronic experiments. In the acute experiment, both the renal sympathetic nerve activity (RSNA) and cardiac sympathetic afferent reflex (CSAR) elicited by epicardial application of capsaicin were recorded. In the chronic experiment, we performed EA for 30 minutes once a day for 1 week to test the long-term EA effects on heart function, remodeling as well as infarct size in CHF rats. The results show EA significantly decreased the RSNA effectively, inhibited CSAR and lowered the blood pressure of CHF rats. Treating CHF rats with EA for 1 week dramatically increased left ventricular ejection fraction (LVEF) and left ventricular fraction shortening (LVFS); and reversed the enlargement of left ventricular end-systolic dimension (LVESD) and left ventricular end-diastolic dimension (LVEDD); and shrunk the infarct size. In this experiment, we demonstrated EA attenuates sympathetic overactivity. Additionally, long-term EA improves cardiac function and remodeling and reduces infarct size in CHF rats. EA is a novel and potentially useful therapy for treating CHF.

Key words: heart failure, electroacupuncture, renal sympathetic nerve activity, cardiac sympathetic afferent reflex
Non-standard Abbreviations and Acronyms

CHF: Chronic heart failure
RSNA: Renal sympathetic nerve activity
CASR: Cardiac afferent sympathetic reflex
EF: Ejection fraction
LVFS: Left ventricular fraction shortening
LVESD: Left ventricular end-systolic dimension
LVEDD: Left ventricular end-diastolic dimension
MAP: Mean arterial pressure
rVLM: Rostral ventrolateral medulla
Introduction

Chronic heart failure (CHF) is a serious, debilitating and increasingly prevalent condition with a poor survival rate. CHF was originally considered to be a hemodynamic disorder, but there is increasing evidence to indicate that CHF is characterized by overwhelmingly compensatory neurohumoral activation including sympathetic overactivity (6, 20, 33, 44, 47). The renal sympathetic nerve activity poses significant contribution to the increased sympathetic drive and altered neurohumoral responses in CHF (12). In addition, the sympathetic overactivity is closely related to abnormalities in cardiovascular reflexes in CHF. It is well known that enhanced cardiac sympathetic afferent reflex is contributory to sustained higher sympathetic outflow activity in the CHF state (45, 46). Prolonged increase of sympathetic tone and withdrawal of parasympathetic activity have been associated with the progression of heart failure, increased sudden death risk, and increased mortality (13, 38). As this has been proven to be detrimental, reduction of sympathetic overactivity has become the cornerstone of CHF treatment.

In traditional Chinese medicine, acupuncture, including the more effective electroacupuncture, has been used to treat a variety of diseases and disorders for at least a thousand years and increasingly acupuncture is being accepted as an alternative medical therapy worldwide. Both clinical evidence and animals studies indicate that acupuncture may have therapeautic and modulatory effects on some types of hypertension, arrhythmias and coronary heart disease (27, 31, 37, 52). It has been demonstrated that EA could lower sympathetic activity (34, 48) and significantly inhibit the cardiovascular sympathoexcitatory reflex responses triggered by chemical, mechanical and electrical stimulations in rats (4, 28,
30, 51). It has been proved that EA at the neiguan-jianshi acupoint [pericardial meridian (P)
5-6] located on the median nerve is most effective in reducing cardiovascular sympathoexcitatory reflex-induced pressor response (27, 52). Therefore, we chose the neiguan-jianshi acupoint for the conduct of this experiment.

None of the studies have been directed at recording the effects of EA on renal sympathetic nerve activity and especially cardiac sympathetic afferent reflex in CHF state and so far there are only a few studies evaluating the effects of acupuncture in patients with CHF. These studies have inconsistently reported whether or not there was an increase in cardiac function (18, 32, 49). The exact effects of EA on CHF remains unclear. Therefore, in the present study, for the first time, we tested the hypothesis that EA attenuates renal sympathetic nerve activity, cardiac sympathetic afferent reflex and improves cardiac function and remodeling through downsizing infarct size in chronic heart failure rats, which may provide experimental evidence that EA might be used as an alternative method to alter progression of heart failure.

Methods

Experimental Animals

Procedures involving rats were approved by the Experimental Animal Care and Use Committee of Nanjing Medical University and the studies conformed to the Guide for the Care and Use of Laboratory Animal published by the US National Institutes of Health (NIH Publication No. 85-23, revised 1996). These procedures were approved by the Institutional Animal Care and Use Committee of Nanjing Medical University. Male Sprague–Dawley rats weighing between 250 and 300g were used in the experiments.

Creation of CHF rats
CHF was induced by the coronary artery ligation technique. All rats were anesthetized with 10% chloral hydrate (0.03ml/kg ip). Surgery was carried out with sterile techniques. The left anterior descending coronary artery was ligated with a 6-0 suture near its branch point from the aorta, between the pulmonary artery outflow tract and the left atrium. Mortality was nearly 40% in these experiments, and death occurred primarily during the first day after ligation. The rats were caged in an environment with ambient temperature maintained at 22°C and humidity at 30–40%. Laboratory chow and tap water were available ad libitum. The sham rats were operated the same as the heart failure rats except ligation of their coronary arteries. The terminal experiment was carried out 4 weeks after coronary ligation or sham surgery. CHF model rats were all confirmed by specific technician using animal echocardiography (Vevo 2100, Detector MS250, FUJIFILM VisualSonics, Inc. Toronto, Canada), which was used to evaluate the heart rate, EF, FS, LVEDD and LVESD through M-mode recordings.

Terminal experiments and measurements

In acute experiments, each rat was anesthetized with urethane (800mg/kg ip) and α-chloralose (40mg/kg ip). Supplemental doses of anaesthetic were administered at 1/10th of the initial dose per hour. A midline incision in the neck was made where the carotid artery was cannulated for measurement of mean arterial pressure (MAP) and heart rate (HR). The external jugular vein was also cannulated for drug infusion.

Renal sympathetic nerve activity recording.

In the rats, a left flank incision was made and a retroperitoneal dissection was used to expose the renal artery and nerves. The renal sympathetic nerves were identified and dissected free of the surrounding connective tissue. The signal was amplified with a Grass direct-current
preamplifier (model P18D; Astro-Med, West Warwick, RI) with the low-frequency cutoff set at 30 Hz and the high-frequency cutoff set at 3 kHz. The amplified and filtered signals were integrated at the time constant of 100 ms. This value was subtracted from all the integrated values of RSNA. The raw nerve activity, integrated nerve activity, MAP, and HR were recorded on a PowerLab data acquisition system (model 16S; ADInstruments, Mountain View, CA) and stored on disk until being analyzed.

**Evaluation of cardiac sympathetic afferent reflex**

The rats were placed on positive-pressure ventilation. The chest was opened through the fourth intercostal space on the left side. The pericardium was removed to fully expose the LV. This preparation was used for anterior LV epicardial application of a piece of filter paper (3x3 mm) containing capsaicin (0.4µg in 2 µl) (M2028, Sigma-Aldrich, St. Louis, MO) for 1 min to induce CSAR. Then, the filter paper was removed and the epicardium was rinsed three times with 10 ml of warm normal saline (37°C). The CSAR was evaluated by change of the RSNA and MAP responses to epicardial application of capsaicin.

**Selection of acupoint and performance of electroacupuncture**

The acupoints of the rats are quite analogous to those in humans. Both neiguan and jianshi acupoint [pericardial meridian (P) 5-6] are located on the median nerve. In the rat, stainless needles were placed at the Neiguan and Jiangshi acupoint located 1.5–2.0 mm and 2.5–3.0 mm above the wrist between the ligaments of the flexor carpi radialis and the palmaris longus respectively. The criterion for accurate needle positioning was relied on the observation of a slight repetitive flexion of the paw during stimulation (30). The stimulation was set at low-current, low-frequency (0.5-ms pulses, 2 Hz and 2 mA) by a Hans-200A electrostimulator.
(Beijing Sheng Da medical instrument center, Beijing, China) for 30 minutes throughout the study. In order to exclude the effect of EA current, for the control EA used in the experiment, Pianli-Wenliu acupoints [large intestine (LI) 6-7] in the forelimb were selected because they are near the neiguan-jianshi acupoint yet are located along another meridian (large intestine meridian) (51).

**Measurement of infarct size**

The left ventricles were cut from apex to base into four transverse slices, which were processed in a routine manner for histological study. Sections 5um thick were cut, stained with Masson's trichrome stain. ImageJ 1.34 software was used to measure lengths and areas of infarct and LV. In each slice, the infarct ratio was defined as the ratio of the length of the endocardial circumference made up by the infarct area to the entire endocardial circumference. Infarct area in each slice was calculated by multiplying the infarct ratio by the LV area of the whole slice. Infarct ratio of the entire LV was obtained by calculating the ratio of the total infarct area to the sum of LV areas in all slices analyzed (5,36).

**Experiment protocol**

**Acute experiments**

Acute experiments have been consisted of two cohorts. In the first cohort, three groups were prepared for the acute experiments including CHF rats-EA (n=6), CHF rats-control EA (n=6) and sham rats-EA (n=6). After the blood pressure, heart rate and baseline RSNA were stable for at least 10mins during recording, we performed bilateral EA at neiguan and jianshi acupoint in CHF and sham rats group at 2Hz and 2mA for 30mins. The control EA were performed at same intensity for 30mins in the other CHF rats group. We tried to discern
whether the EA at the neiguan-jianshi acupoint could reduce the renal sympathetic nerve activity among CHF rats compared with sham rats and control EA treated CHF rats. In second cohort, three groups including CHF rats-EA (n=6), CHF rats-control EA (n=6) and sham rats-EA (n=6) were used to record CSAR. After the blood pressure, heart rate and baseline RSNA were stable for at least 10mins, the CSAR elicited by epicardial application of capsaicin was recorded before and 30min after EA and control EA. We evaluated whether EA at the neiguan-jianshi acupoint could weaken the CSAR among CHF rats compared with sham rats and control EA treated CHF rats.

**Chronic experiments**

The rats were divided into three groups: CHF rats-EA (n=10), CHF rats-metoprolol (n=6), CHF rats-control EA (n=7) as well as CHF rats without EA (n=6). In the chronic experiment, EA was initiated 4 weeks after ligation of left anterior descending coronary artery. All rats were treated under anesthesia when EA stimulation was given. We performed bilateral EA at the neiguan-jianshi acupoint in CHF rats. Meanwhile, control EA were performed in another CHF rats group using the Pianli-Wenliu acupoint. The stimulation was given at 2Hz and 2mA for 30min once per day for a total duration of 1 week. After 1 week of stimulation, the cardiac structure and function of rats within all groups was assessed by animal echocardiography (Vevo 2100, Detector MS250, FUJIFILM VisualSonics, Inc. Toronto, Canada). For comparison with the effects of EA on heart performance, the effective β1-selective blocker, metoprolol (MP; Sigma, St.Louis, U.S.A.), was used as a control. The CHF rats orally administered the metoprolol on the dose basis of 60mg/kg once per day for 7 days by direct gastric lavage (42). After the examination of animal echocardiography, the CHF rats were
sacrificed for heart excision, the ratio of heart weight to body weight and infarct size were measured.

Statistical analysis

The effects of EA on RSNA are expressed and calculated as the percentage change from the baseline. The percentage changes of the RSNA induced by cardiac sympathetic afferent nerve stimulation were used as an indicator of the sensitivity of the CSAR. The improvement of cardiac function and structure in the CHF rats was evaluated by the change of animal echocardiography results after 1-week EA treatment. Two-way ANOVA associated with the Newman-Keuls test for post hoc analysis was used when multiple comparisons were made. Other Comparisons were assessed by Student’s paired t-test. All statistical analysis were done using computer software (SPSS; Chicago, IL). All data were expressed as mean ± SE. A value of P<0.05 was considered statistically significant.

Results

Effects of coronary ligation on baseline hemodynamics and heart weight

The measurements of cardiac function and mean arterial pressure (MAP) in sham rats (n=17) and CHF rats (n=29) are shown in Table 1. It is readily observed that the CHF rats have a significant decrease in ejection fraction (EF) and fraction shortening (FS) as well as MAP compared with sham groups (P<0.05). Additionally, left ventricular end-systolic dimension (LVESD) and left ventricular end-diastolic dimension (LVEDD) were significantly enlarged in CHF rats compared with sham rats (P<0.05). The table-1 also demonstrates that heart weight and the ratio of heart weight to body weight were significantly greater in CHF group rats than in sham group rats (P<0.05). Overall, this data indicates the presence of myocardial
damage and suggests a decreased contractile function and cardiac remodeling in CHF rats.

**Effects of EA on renal sympathetic nerve activity (RSNA) and mean arterial pressure (MAP)**

A representative recording of effects of EA on RSNA and MAP is shown in Figure-1. As can be seen, EA significantly lowered the MAP and RSNA in CHF rats compared with sham rats and control EA treated CHF rats. As shown in Figure-2, EA significantly reduced RSNA and MAP in CHF rats. In contrast, control EA had no significant effects on RSNA and MAP in CHF rats (p<0.05). However, the sham rats group that received EA could show decrease in RSNA and MAP as well. Importantly, EA reduced RSNA and MAP in CHF rats more significantly than sham rats (p<0.05).

**Effects of EA on cardiac sympathetic afferent reflex (CSAR) elicited by epicardial application of capsaicin**

The representative recording of effects of EA on CSAR before-and-after EA is shown in Figure-3, which demonstrates that there is significant inhibition of CSAR in CHF rats after EA. Figure-4 demonstrates that RSNA and MAP responses to epicardial application of capsaicin is significantly enhanced in CHF rats than in Sham rats before EA (P<0.05) (Figure-4A and B). After the treatment of EA, the increase in RSNA and MAP induced by capsaicin among CHF rats were significantly inhibited compared with sham rats and control EA treated CHF rats (P<0.05). The control EA had no significant effect on RSNA and MAP by capsaicin in CHF rats.

**Effects of long-term EA on the cardiac function, remodeling and infarct size**

Figure-5 demonstrates that one week of EA treatment could significantly improve the heart
function and structure in CHF rats. One week long EA dramatically increases the EF (Figure-5A) and FS (Figure-5B) and reverses the enlargement of LVEDD (Figure-5C) and LVESD (Figure-5D) in CHF rats compared with control EA treated CHF rats and CHF rats without treatment (P<0.05). The effects of EA stimulation are comparable to the effects of \( \beta_1 \)-selective blocker, metoprolol on heart function and structure. Additionally, Figure-6 shows the EA could as well shrink the infarct size in CHF rats compared with non-EA treated CHF rats (P<0.05), which is consistent with effects of metoprolol, but the effects of EA are weaker than that of metoprolol in reducing the infarct size (P<0.05). However, there was no significant difference in ratio of heart weight to body weight among EA treated CHF rats and control EA treated CHF rats and CHF rats without treatment.

**Discussion**

Chronic heart failure originates from an impairment of the systolic and the diastolic functions of the heart. The main causes of CHF today are ischemic heart disease and hypertension as well as cardiomyopathy (11). Despite optimised heart failure medication, chronic heart failure continues to cause significant morbidity and mortality rates worldwide, demonstrated by a 5-year survival rate of 25% in men and 38% in women (17), making it highly desirable to discover another effective way to treat CHF. This paper is the first animal study to directly examine the effects of EA on sympathetic activity and progression of CHF after left anterior descending coronary artery ligation in CHF rats. We found direct evidence EA reduces the renal sympathetic nerve activity and inhibit cardiac sympathetic afferent reflex which is sensitized in the CHF state. Additionally, EA plays a potentially useful role in improving cardiac function and remodeling and reducing infarct size. The main objective of the present
study was to test the hypothesis that EA can be beneficial to modulate sympathetic activity in the CHF state and improve the function and structure of the debilitating CHF as well as reducing the infarct size, which could be comparable to effects of the beta-blockade drugs like metoprolol. We provide the evidence that EA could be usefully alternative way, at least in the complementary way, to treat CHF.

The mechanisms underlying CHF are partially understood. Mounting evidence indicates that neurohumoral activation, especially autonomic dysfunction including sympathetic overactivity, contributes to the progression of CHF (23, 33, 44, 47). In fact, sympathetic activation is even present early in the course of left ventricular dysfunction (3, 14). In an animal model of early left ventricular dysfunction without overt heart failure, among which it has shown that sympathetically influenced low-frequency heart rate variability was significantly increased and parasympathetically mediated high-frequency variability was significantly decreased (10). Initially, the increased sympathetic nervous system activity during the early phase of CHF helps the heart compensate for deteriorating pumping function. However, excessively sustained activation has a devastating impact on cardiac function and remodeling and therefore, the maladaptive process is a key factor in the pathophysiology of CHF (44). Increased sympathetic drive and reduced parasympathetic activity have been highly relevant to both disease severity and the prognosis in CHF (6). Therefore, reduction of sympathetic overactivity has become the cornerstone of CHF treatment. Most recent strategies have focused on preventing organs from high sympathetic drive by reducing the sympathetic overactivity such as β-adrenergic blockers (9).

Sympathetic overactivity may also directly and indirectly exert adverse effects on the
structure and function of heart. Reportedly, increased norepinephrine release is relevant to the development of left ventricular hypertrophy and exerts toxical effects on cardiac fibroblasts and myocytes (39, 41). In addition, high level norepinephrine acts through the $\beta$-adrenergic pathway to stimulate apoptosis in adult rat ventricular myocytes in vitro (7) and apoptosis plays an important role in the left ventricular remodeling progression process (35). Clinical and basic studies have demonstrated that sympathoexcitation was relevant to blunted arterial baroreceptor and cardiopulmonary receptor reflexes as well as enhanced cardiac afferent sympathetic reflex which is a sympathoexcitatory reflex (46, 47). In the brainstem, rostral ventrolateral medulla (rVLM) is well known as the regulation center of sympathetic activity (8). The central abnormalities of regulation for sympathetic nervous system overactivity in chronic heart failure have been reported to play a significant role (21). It is well established that brain renin angiotensin system, oxidative stress, the dysfunction of nitrogen oxide production in the rVLM contribute to the sympathoexcitation and enhanced cardiac afferent sympathetic reflex in chronic heart failure (16, 45, 54). Therefore, rVLM is a potentially powerful therapeutic target to suppress the sympathetic overactivation. With the current various therapeutic approaches utilizing $\beta$-adrenergic blockers, the prognosis is still not good enough and it is necessary to avoid the side effects of medication. From the available data, conservative pharmacological therapy is likely to be insufficient.

Acupuncture as a therapeutic intervention is widely accepted and practiced in the United States (2). Acupuncture can effectively treat postoperative and chemotherapy nausea and vomiting as well as postoperative pain. Acupuncture can also be useful as an effective method to treat other situations, such as addiction, headache, tennis elbow, myofascial pain,
osteoarthritis, low back pain, and asthma. The Neiguan-jianshi acupoint is most effective
point to treat cardiovascular diseases (24, 27, 52). In previous studies, EA at the
neiguan-jianshi could inhibit cardiovascular sympathoexcitatory pressor reflex responses
triggered by chemical, mechanical and electrical stimulation in rats (4, 28, 30, 51). For
example, stimulation of the median nerve to mimic EA at the Neiguan acupoint diminishes
regional myocardial ischemia triggered by a sympathetically mediated increase in cardiac
oxygen demand in cats with the partial coronary artery occluded (28). However, the exact
effects of EA on CHF are still elusive, due to ambiguous outcomes reported by different
clinical studies. Therefore, in this paper, we tried to examine if EA could also attenuate the
renal sympathetic nerve activity and cardiac sympathetic afferent reflex and improve cardiac
function and remodeling in CHF rats. The present study confirms our hypothesis that EA
improves cardiac function and cardiac remodeling, reduces infarct size by inhibition of
sympathetic activity in CHF rats, but the exact mechanisms to explain these changes remain
unknown. In previous studies, it has been demonstrated that group III and IV afferent
pathways in the median nerves were activated during EA, which provide input to a number of
regions in the hypothalamus, midbrain, and brain stem that are associated with cardiovascular
regulated sympathetic activity (28, 29, 32, 43). Specifically, resection of the median nerve
eliminated the therapeutic effect of EA at the Jianshi-Neiguan acupoint (53). From previous
available studies, EA at neiguan-jianshi could activate μ- and δ-opioid receptors to inhibit
sympathetic activity, specifically receptors located in the rostral ventrolateral medulla (rVLM)
(4, 29, 53). Additionally, EA can induce preproenkephalin mRNA expression in rVLM which
accounts for reductions in sympathetic outflow (25). EA could result in modulation of the
nNOS activity in the hypothalamus to regulate sympathetic outflow in spontaneously hypertensive rats (19). EA could also modulate cardiac autonomic imbalance by increasing the cardiac vagal component of heart rate variability (15, 22). There are studies reporting that vagal nerve stimulation markedly improved the long-term survival of CHF rats through the prevention of pumping failure and cardiac remodeling (26, 50). These effects could explain our finding that the cardiac function and remodeling as well as infarct size reduction in CHF rats could be improved by long-term EA stimulation via the possibility of enhanced vagal drive and inhibited sympathetic activity.

Clinical Implication and Study Limitations

This present study shows for the first time that EA attenuates renal sympathetic nerve activity and cardiac sympathetic afferent reflex in CHF rats and suggests that EA improves cardiac function and remodeling as well as reducing infarct size to prevent cardiac progression in chronic heart failure rats, which may provide evidence that EA could be a useful alternative treatment method to heart failure. In addition to CHF, the sympathetic activity reduction also plays a pivotal role in the treatment of other diseases, such as diabetes and chronic renal failure (40), so it is conceivable that EA is a potentially promising and valuable way to manage the category of these disorders. In this study, the usefulness of EA as a complementary or alternative therapy in animals has herein been established, but the relevant mechanism has not been well addressed. Therefore, more large-scale, long-term trials of research on its clinical efficacy as well as the underlying mechanisms of its effects need to be further performed.

Acknowledgement
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Disclosures

None

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Danzhong but not to Zhongting increases the cardiac vagal component of heart rate variability.


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Figure Legends

Figure-1 Representative recording of RSNA and MAP change by EA. Original recording of mean arterial pressure (MAP) and renal sympathetic nerve activity (RSNA) is shown in (A) CHF rats-EA; (B) Sham rats-EA; (C) CHF rats-control EA. The arrows indicate EA was started. EA indicates electroacupuncture; RSNA, renal sympathetic nerve activity; MAP, mean arterial pressure; Raw RSNA was defined as renal sympathetic nerve discharge before noise clearance; Intergated RSNA was defined as renal sympathetic nerve discharge after noise clearance.

Figure-2 Effects of 30-min EA on (A) percentage of RSNA change; (B) MAP change in EA treated sham rats(sham-EA), EA treated CHF rats (CHF-EA) and control EA treated CHF rats (CHF-control EA) *P<0.05, compared with CHF-control EA, †P< 0.05, compared with sham-EA. EA indicates electroacupuncture; RSNA, renal sympathetic nerve activity; MAP, mean arterial pressure.

Figure-3 The representative recording of RSNA and MAP change induced by epicardial application of capsaicin before-and-after EA in CHF rats. (A) CSAR before EA in CHF; (B) CSAR after EA in CHF. At arrows, the capsaicin was applied. EA indicates electroacupuncture; RSNA, renal sympathetic nerve activity; MAP, mean arterial pressure; CSAR, cardiac sympathetic afferent reflex.

Figure-4 Effects of 30-min EA on cardiac sympathetic afferent reflex (CSAR). (A) percentage
of RSNA change induced by epicardial application of capsaicin before and after EA; and (B) MAP change induced by epicardial application of capsaicin before and after EA in EA treated sham rats, control EA treated CHF rats and EA treated CHF rats. *P< 0.05, CHF-before EA vs. Sham-before EA, † P< 0.05, CHF-after EA vs. CHF-before EA. EA indicates electroacupuncture; RSNA, renal sympathetic nerve activity; MAP, mean arterial pressure

Figure-5 The 1-week-long effects of EA on (A) the change of ejection fraction; (B) the change of fraction shortening; (C) the change of LVEDD; and (D) the change of LVESD in CHF, compared with CHF- EA and CHF-control EA and CHF-metoprolol. * P<0.05, vs. CHF only, † P< 0.05, vs. CHF-control EA , # P< 0.05, vs. CHF-metoprolol. EA indicates electroacupuncture; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension

Figure-6 The effects of EA on infarct size in CHF rats compared with non-EA treated CHF rats and metoprolol treated CHF rats. (A) CHF only; (B) CHF-EA; (C) CHF-metoprolol; (D) the percentage of infarct size in LV area. * P<0.05, vs. CHF, † P< 0.05, vs. CHF-EA. EA indicates electroacupuncture, LV indicates left ventricle.
Table 1: Effects of coronary ligation on baseline hemodynamics and heart weight in CHF and sham rats

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<td>n</td>
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<td>Baseline MAP (mmHg)</td>
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<td>Heart Rate</td>
<td>435.8±9.3</td>
<td>386.7±6.0*</td>
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<td>FS(%)</td>
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CHF, chronic heart failure; MAP, mean arterial pressure; EF, ejection fraction; FS, fraction shortening; LVESD, left ventricular end-systolic dimension; LVEDD, left ventricular end-diastolic dimension; HW/BW, heart weight/ body weight. Data are given as mean ±SE.

*P<0.05, CHF vs. Sham.
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CHF, chronic heart failure; MAP, mean arterial pressure; EF, ejection fraction; FS, fraction shortening; LVESD, left ventricular end-systolic dimension; LVEDD, left ventricular end-diastolic dimension; HW/BW, heart weight/ body weight. Data are given as mean ±SE.

*P< 0.05, CHF vs. Sham.