Results Infarct size was slightly reduced in AVE 0991 group compared to control group (42.6±3.6% vs 50.9±4.4%, p<0.01). In addition, AVE 0991 reduced MI-induced hypertrophy as quantified by diameter measurements of cardiomyocytes (vs. control group 25.49±4.37 μm vs 32.06±6.85 μm, p<0.05). The overexpression of TGF-β1 and TNF-α mRNA were inhibited by chronic AVE 0991 treatment alone (vs. control group, TGF-β1: 4.15±1.18 vs 14.25±3.84, p<0.05; TNF-α: 2.21±0.44 vs 3.87±0.55, p<0.05, respectively). AVE 0991 markedly attenuated the increase of the expression of Collagen I (1.79±0.15 vs 4.3±0.75, p<0.001) and III (3.12±0.42 vs 9.55±0.83, p<0.001) mRNA compared to control group.

Conclusion The nonpeptide angiotensin-(1-7) analogue AVE 0991 could attenuate overexpression of inflammatory cytokines and ventricular remodelling induced by myocardial infarction (MI).

Objective To investigate the effects of perindopril and/or spironolactone on atrial structural and functional remodelling in atrial fibrillation (AF) dogs induced by chronic rapid atrial pacing, and research the relations between rennin-angioensin-aldosterone system (RAAS) and atrial interstitial remodelling and atrial fibrillation.

Methods 24 healthy male hybrid dogs aged 15–18 months were paced for 8 weeks and randomly divided into four groups: control group, perindopril group (P), spironolactone group (S), and combination of perindopril and spironolactone group (P+S). The dogs in P group, S group, and P+S group respectively received perindopril (1 mg·kg⁻¹·d⁻¹) and/or spironolactone (10 mg·kg⁻¹·d⁻¹). Plasma Angiotensin II (Ang II) and aldosterone (Ald) were measured before and after 4 and 8 weeks pacing. Transthoracic echocardiographic examinations were performed before and after 8 weeks pacing. The number of dogs maintained AF and duration of AF after stopping of pacing were recorded. Atrial collagen volume fraction (CVF) was analysed by Masson staining after 8 weeks pacing.

Results (1) Plasma Ang II and Ald were no significant differences between four groups before pacing. Compared with the control group, plasma Ang II and Ald after 4 and 8 weeks pacing was significantly higher than that before pacing; in the other groups, there were no significant differences. (2) Compared with the control group, the diameter, end-systolic volume and end-diastolic volume of the left atrium of P group, S group and P+S group were significantly lower. In the control group, plasma Ang II and Ald levels after 4 and 8 weeks pacing was significantly lower than that before pacing; in the other groups, there were no significant differences. (3) Compared with the control group, the rate of dogs maintained atrial fibrillation of three drug treatment groups after stopping of pacing significantly reduced, with a shorter average duration of AF. (4) Compared with the control group, the value of CVF in P group, S group and P+S group was significantly lower.

Conclusion The occurrence and development of atrial fibrillation and atrial structural remodelling is closely related to RAAS activation. The RAAS blockers can inhibit atrial fibrosis, improve the changes of atrial structure and function, and reduce the incidence and duration of atrial fibrillation in the atrial fibrillation dogs induced by chronic rapid atrial pacing.