Reviewer's report

Title: Ventricular-arterial uncoupling after myocardial infarction in dogs - invasive versus echocardiographic evaluation

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Reviewer: Steven Lavine

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The authors in this manuscript which to determine the extent of systolic and diastolic functional abnormalities in a model of heart failure with preserved LV ejection fraction. In this study, the authors used a healed model of MI induced by coronary ligation 2 months post myocardial infarction utilizing echocardiography and conductance catheter measurements of left ventricular pressure-volume relationships were obtained in 17 beagle dogs 2 months after a coronary artery ligation, and in 6 controls. At 2 months post ligation, healed myocardial infarction was associated with a well preserved echocardiographic left ventricular ejection fraction (57%) and altered Doppler mitral indices of diastolic function. Invasive measurements showed a markedly decreased end-systolic elastance and end-systolic elastance to effective arterial elastance ratio with altered active relaxation (reduced dP/dt-min but not tau) and preserved left ventricular capacitance and chamber stiffness stiffness constant. Among echocardiographic variables, the wall motion score index was the most reliable indicator of cardiac contractility while E', E/A and E'/A' were correlated to dP/dt-min. They concluded that in a canine model of healed myocardial infarction induced by coronary ligation, heart failure is essentially characterized by an altered contractility with left ventricular-arterial uncoupling despite vascular compensation rather than by an intrinsic diastolic failure.

The presentation of the data make an interesting argument for the importance of greater LV volume, reduced relaxation parameters, nonsignificantly elevated chamber stiffness constant, and ventricular vascular uncoupling as important in explaining this syndrome. The authors chose to concentrate primarily on ventricular vascular uncoupling primarily due to reduced end systolic elastance and slightly reduced arterial elastance. Unfortunately, arterial elastance was calculated from single plane echo volumes (need to be biplane with regional abnormalities) and end systolic elastance from conductance catheter. This is not an issue unless the volumes are different. Referral to the tables indicates substantial difference in end diastolic volumes. End systolic volumes from conductance catheter are not obvious but must be larger as is the stroke volume and filling volume. I would not argue about either calculation but the ratios are an issue. Also, there are no control measurements prior to induction of myocardial infarction.

Another important issue is the pericardium might have been left open. Although it
may not be an issue perhaps in data interpretation unless LV volumes changed from baseline to post MI. It should be so noted in the text in both the methods and as a limitation. Also, the LVEDP of control dogs is 14 mmHg?? This is high as is the 23 mm Hg for post MI dogs. Referral to reference 7 will indicate that their LVEDP’s were lower. Why??

Also, the rationale for reduced aldosterone levels was unclear.

Finally, heart failure with preserved EF is a heterogeneous disease. Data post MI with preserved function is thye relevant subset of this entity not hypertensive heart disease. This needs to be established in the introduction and discussion.

**Level of interest:** An article of importance in its field

**Quality of written English:** Acceptable

**Statistical review:** No, the manuscript does not need to be seen by a statistician.

**Declaration of competing interests:**

I declare that I have no competing interests.