

An Experimental Porcine Model for Left Ventricular Hypertrophy

S. Lunde¹, M. Smerup¹, E. Sloth² and J.M. Hasenkam¹

¹Department of Cardiothoracic & Vascular Surgery, ²Department of Anesthesiology and Intensive Care, Skejby Sygehus, Aarhus University Hospital, Aarhus, Denmark

Introduction. Severe left ventricular hypertrophy (LVH) is commonly caused by acquired valvular aortic stenosis (AS). After aortic valve replacement the left ventricle remains hypertrophic with impaired compliance and relaxation, leaving abnormal haemodynamics as a challenge in the immediate postoperative period. In addition, many LVH patients are overly sensitive to pericardial effusion and therefore readily develop symptoms of tamponade at an earlier stage than non-LVH patients. The haemodynamic management of LVH patients is poorly understood, and many aspects remain unknown. Some features could be disclosed by means of a long term animal LVH model in which the difficulties of the haemodynamic management could be recreated and investigated systematically. The aim of this study was to develop a long term porcine LVH model using supracoronary aortic banding.

Method. Sixteen female 5 kg domestic piglets were randomly divided into two groups. In the first group (n=12) we performed the actual banding procedure and in the second group (n=4) we performed a sham-operation to obtain baseline values. The ascending aorta was exposed via a left lateral thoracotomy and a suture was passed through a silicone tube individually fitted to the circumference of aorta with no stenosis. The ends of the suture were tied together and the pericardial sack and the layers of muscle and skin were closed.

Left ventricular mid-septal and free wall thickness at papillary muscle level were assessed at follow-up examinations after four, six and eight weeks by echocardiographic measurements through a right transthoracic acoustic window using a 3.5 MHz Matrix transducer. At eight weeks follow-up examination, each animal was euthanized and the post-mortem mass of the dissected heart was measured in order to calculate the Heart / body weight ratio, and the diameter of 100 randomly selected cardiac myocytes were measured in each specimen. Results are displayed as mean \pm standard deviation. An unpaired t test was used and $P < 0.05$ was considered statistically significant.*

Results. Please refer to figure 1

Intervention Control

Free wall thickness / cm $0,77 \pm 0,013$ $0,60 \pm 0,006$ $P = 0,015^*$

Mid-septal thickness / cm $0,79 \pm 0,015$ $0,58 \pm 0,010$ $P = 0,012^*$

Heart / Body weight ratio $\times 10^3$ $7,73 \pm 0,970$ $6,23 \pm 0,430$ $P = 0,003^*$

Diameter of the cardiac myocytes / μm 19.6 ± 4.9 11.0 ± 1.9 $P = 0.000^*$

Fig. 1: Results from the eight weeks follow-up examination and post mortem examination.

Conclusion. A practical, feasible long-term porcine model of LVH has been established. Due to the nature of the gradual increase in LV afterload the model may mimic the pathogenic progression of LVH as seen in acquired AS.