Perfusion of an Empty-Beating Heart, a New Good Technique to Protect Hypertrophied Hearts for Valve Surgery

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Introduction: Conventional heart protection techniques (Cardioplegia) may result in postoperative heart dysfunction or even heart failure, especially in the patients with hypertrophied hearts. We believe that the postoperative cardiac dysfunction is related to myocardial edema and expansion of cellular compartments caused by cardioplegia. To improve protection of hypertrophied hearts we have recently developed a new technique, so called "perfusion of an empty-beating heart" (PEBH). Unlike cardioplegia, the new technique maintains normal cardiac electromechanical activities throughout protection period. Therefore we hypothesized that use of PEBH could completely abolish cardioplegia-associated myocardial edema and enlargement of cellular compartments, and also maintains normal myocardial energy metabolism. The present study was to test our hypothesis.

Materials and Methods: A pig model of the left ventricular hypertrophy was induced by banding the ascending aorta for 10 weeks. The hypertrophied pig hearts were then excised and mounted on a Langendorff apparatus that was placed in the center of a 7T magnet. All the hearts were subjected to a protocol consisting of a 20-min control perfusion and 80-min myocardial protection with either PEBH (group I, n=10) or cardioplegia (group II, n=10). PEBH and cardioplegia were conducted with normokalemic and hyperkalemic pig blood, respectively. Heart was perfused through the aorta during control perfusion and through both aorta and coronary sinus during myocardial protection. Throughout protocol, interstitial hydrostatic pressure (IHP) was monitored with a microtip pressure transducer. Volumes of the intra- and extra-cellular compartments and myocardial energy metabolism were measured using ^{31}P MR spectroscopy in conjunction with chemical markers for total water compartment (dimethyl methylphosphonate) and extracellular compartment (phenylphosphonic acid). Volume of the intracellular compartment was calculated by subtracting the volume of the extracellular compartment from the total water compartment.

Results: We found that PEBH maintained IHP at a normal level (13.0 ± 0.6 mmHg) throughout heart protection period while cardioplegia increased IHP significantly (23.3 ± 1.2 mmHg) (Fig. 1). PEBH also caused a much less increase in the volume of extracellular compartment than did cardioplegia (Fig. 2). More importantly, the volume of the intracellular compartment remained unchanged during 80- min PEBH whereas it increased significantly during cardioplegia (Fig. 3). ^{31}P MR spectra showed that PEBH maintained normal levels of myocardial ATP, PCr, and Pi when its blood flow was greater than 50% of its control value (flow set with perfusion pressure at 60 mmHg) (Figs. 4-6). A significant decrease in PCr and ATP was observed only when blood flow of PEBH was lower than 50% of its control (Figs. 4-6). In contrast, cardioplegia maintained normal levels of ATP, PCr, and Pi until its blood flow lower than 10% of its control value.

Conclusion: PEBH prevents myocardial edema and enlargement of intracellular compartment and is able to sustain normal myocardial energy metabolism even in presence of a moderate coronary stenosis. It is expected that the new technique will improve postoperative patient recovery.

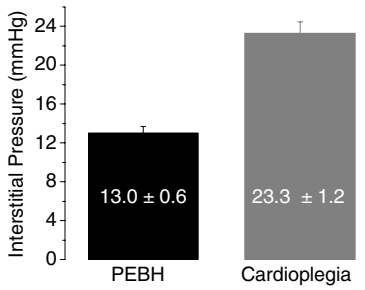

Fig 1. Interstitial hydrostatic pressure (IHP) obtained from pig hearts at the end of myocardial protection. Note that IHP was significantly higher in cardioplegia group than in PEBH group.

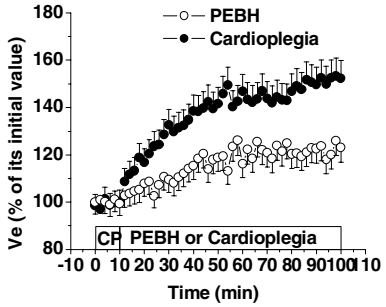

Fig 2. Comparison of extracellular space measured during PEBH and cardioplegia. Note that the increased extent of extracellular space was greater during cardioplegia than during PEBH.

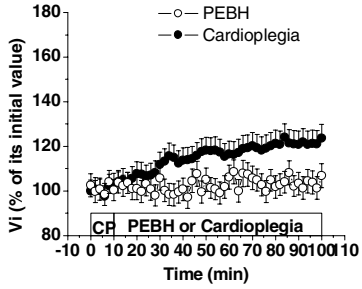

Fig 3. Comparison of intracellular space measured during PEBH and cardioplegia. Note that intracellular space had almost unchanged during PEBH, while intracellular space increased by 20% at the end of cardioplegia.

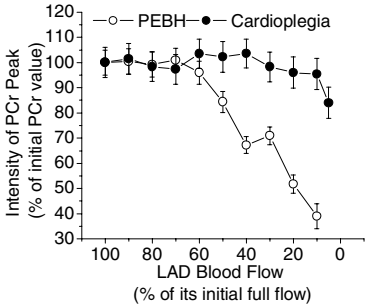

Fig 4. Changes in PCr levels with dif LAD blood flow reduced during PEBH or cardioplegia. Note that PCr level began to decrease when LAD blood flow was less than 50% of its initial flow during PEBH.

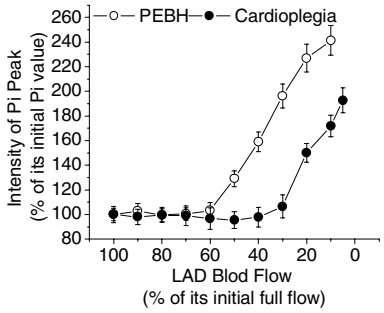

Fig 5. Changes in Pi levels with LAD blood flow reduced during PEBH or cardioplegia. Note that Pi level started to rise when blood flow was reduced by more than 50% of its initial flow during PEBH.

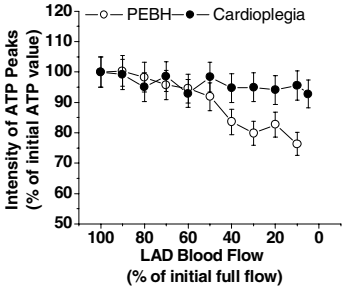

Fig 6. Changes in ATP levels with LAD blood flow reduced during PEBH and cardioplegia. Note that ATP level of beating heart drop at more than 50% coronary stemosis; however, the difference did not reach statistical significance.