Dynamic Patch Artificial Myocardium; Effects on the Residual Myocardial Function

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Dynamic patch artificial myocardium (D-PATCH) has been developed to replace the damaged left ventricular (LV) wall in severe cardiogenic shock patients with a massive myocardial infarction. This study was undertaken to demonstrate the effects of (1) single D-PATCH support on the global cardiac function (group 1, 12 dogs, whole heart model), (2) single D-PATCH support on the LV function and myocardial metabolism (group 2, 6 dogs, right heart bypass model), and (3) the concomitant support of D-PATCH and aortic counterpulsation (CP) on the regional myocardial function of the residual LV wall (group 3, 6 dogs, right heart bypass model with ultrasonic piezoelectric crystals). In group 1, mean aortic pressure and cardiac output were increased 20–36% ($p < 0.01$) and 17–50% ($p < 0.01$) respectively, and mean left atrial pressure was decreased 16% ($p < 0.01$) by D-PATCH assist. In group 2, under constant preload volume, afterload pressure and heart rate, tension time index (TTI) was decreased 21–26% ($p < 0.01$) and myocardial oxygen consumption was also decreased 24–29% ($p < 0.001$) by D-PATCH assist. In group 3, although peak LVP was increased by single D-PATCH assist, when heart was assisted concomitantly by D-PATCH and CP peak LVP was significantly decreased (18%, $p < 0.01$). Percent LV segmental shortening was not increased by CP assist, but increased 143% ($p < 0.02$) by D-PATCH assist. In conclusion, D-PATCH can improve the global LV function and myocardial metabolism of the ischemic failing heart, also improve the regional myocardial function of the residual LV wall. Thus, D-PATCH is effective for the salvage of ischemic myocardium while maintaining the global cardiac function.

FOR the recovery of the patients in severe cardiogenic shock caused by acute myocardial infarction, excision of the infarcted area concomitantly with complete revascularization can be beneficial by preventing paradoxical

systolic expansion!

Successful infarctectomies which involved no more than 7% to 15% of the left ventricular wall has been reported,² a rapid left ventricular failure will develop in larger infarctectomies due to a severe reduction of the left ventricular volume.^{3,4} Therefore, a synthetic material patch has been inserted for these patients in order to maintain the left ventricular volume, but this akinetic patch also led to a severe reduction in cardiac output.⁵

Key Words:

Artificial myocardium
Left ventricular assist device
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Fig.1. AVCO-MGH Dynamic Patch Artificial Myocardium (D-PATCH).

Fig.2. Dynamic Patch Artificial Myocardium anastomosed to left ventricular wall of dog.

For the purpose of maintaining the left ventricular volume while mechanically supporting the failing heart, the dynamic patch artificial myocardium (Dynamic Patch) has been developed.^{6,7}

The assist mechanism of Dynamic Patch is quite different from that of conventional IABP or bypass type left ventricular assist devices. It is still not known whether Dynamic Patch can contribute to the recovery of the damaged ischemic myocardium. Because the direct assist mechanism of Dynamic Patch on the ischemic left ventricle may increase left ventricular pressure and left ventricular pressure work, also myocardial oxygen demand.⁸

The purpose of the study is to demonstrate

- (1) the effects of Dynamic Patch Artificial Myocardium on the left ventricular function and myocardial metabolism.

Fig.3. Right Heart Bypass Model with Dynamic Patch Artificial Myocardium and Extra Aortic Counterpulsation Device.

- (2) the hemodynamic effects of the concomitant assist of Dynamic Patch and Aortic Counterpulsation on the failing heart.
- (3) the effect of Dynamic Patch on the regional myocardial function of residual left ventricular wall.

MATERIALS AND METHODS

AVCO-MGH Dynamic Patch Artificial Myocardium (D-PATCH) is wedge-shaped and consists of an outer plastic shell with an inner inflatable diaphragm made of AVCOTHANE, and Dacron sewing ring for suturing to the left ventricular free wall as previously described (Fig. 1).⁷⁻⁹ The size of D-PATCH is 4.5 x 6.5 cm in the diaphragm portion and generates a 25 ml maximum stroke volume by pneumatic drive. The Extra-aortic Counterpulsation Device (CP) is tube-shaped and consists of an AVCO balloon (20 ml) mounted inside of a femoral arterial cannula (22ft. USCI) and polyvinyl chrolide tube (30 cm in length and 3/8 inch in diameter). As driving systems, we used an AVCO PULASTILE PUMP MODEL 20 for D-PATCH and an AVCO

Fig.4. Representative hemodynamic effect of D-PATCH in severe heart failure (group 1, whole heart model).

AP = aortic pressure; LVP = left ventricular pressure; LAP = left atrial pressure; LAD FLOW = left anterior descending arterial flow; CO = cardiac output; dp/dt = rate of rise of left ventricular pressure

BALLOON PUMP MODEL 7 for CP. The driving pneumatic pressure were adjusted to 200 mmHg respectively. Inflation timing of pneumatic drive of D-PATCH was adjusted to the left ventricular systole and that of CP was adjusted to diastole in order to achieve maximal reduction of the mean left atrial pressure (LAP). 24 mongrel dogs weighing 15 to 36 kg were anesthetized intravenously and maintained with sodium pentobarbital (initial dose 25 mg/kg BW). Ventilation was controlled by a volume respirator (Harvard Apparatus) and ECG was monitored continuously. The chest was opened through a median sternotomy and catheters were placed in the aortic root and right superior pulmonary vein for continuously monitoring central aortic and left atrial pressure. After systemic heparinization (3 mg/kg BW), the animals were placed on cardiopulmonary bypass with aortic perfusion through left subclavian artery and venous return through separate caval cannulas, and azygos vein was divided. A blood priming was used to maintain HCT > 30%.

A small catheter was inserted into the aortic root for injection of the cardioplegic solution. Under systemic hypothermia (20–22°C) the ascending aorta was cross-clamped and cold crystalloid cardioplegic solution (4°C, K⁺ 35 mEq/L, vol. 10 ml/kg BW) was injected through the aortic root cannula, and re-infused every 30 minutes during the aortic cross-clamp. An area of 15 ± 2% of the left ventricular free wall (wet weight) was resected carefully avoiding injury to the papillary muscles. The D-PATCH was implanted with intermittent and continuous suture anastomosis with 3–0 Tevdek or 2–0 Ticon. About 5 minutes before aortic de-clamp, the arterial perfusion temperature was raised to 37°C and maintained in that range throughout the experiment. (Fig. 2).

After aortic declamp defibrillation was performed if necessary. The heart was perfused for 60 minutes and maintained in the empty beating condition by left ventricular venting. Dogs were separated into three groups.

Group 1 Whole Heart Model with D-PATCH

Fig.5. Representative hemodynamic effect of D-PATCH in mild heart failure. (group 2, right heart bypass model) Note the remarkable change of tension time index (TTI, 10.5 mmHg·sec→15.9 mmHg·sec).

assist. (12 dogs)

Group 2 Right Heart Bypass Model with D-PATCH assist. (6 dogs)

Group 3 Right Heart Bypass Model with concomitant assist of D-PATCH and Aortic Counterpulsation. (6 dogs)

In 12 dogs (group 1), after 60 minutes of reperfusion dogs were separated from cardiopulmonary bypass and chest was closed with chest drainage tubes in bilateral thoracic cavities. The anticoagulant effect of heparin was neutralized by protamine. Under mild anesthesia and control ventilation the hemodynamic effects of D-PATCH were observed upto 18 hours.

In other 12 dogs, after 60 minutes of reperfusion, a second perfusion cannula was inserted to the main pulmonary artery and a ligature placed around the pulmonary artery, completely isolating the right heart which then received only coronary venous drainage. Total coronary flow (minus left Thebesian flow) was drained from the cannulated right ventricle. The sinoatrial node was successfully crushed in all 12

dogs, and heart rate was maintained constant at 160 per minute in group 2 (6 dogs) and 120 per minute in group 3 (6 dogs) by atrial pacing.

In group 2, with pulmonary arterial volume loading, two conditions of heart failure were produced by the infusion of propranolol. In the first condition, with a constant preload volume (80 ml/kg BW/min) and a constant afterload pressure (80 mmHg), propranolol was infused until LAP exceeded 15 mmHg (condition-A). Severe heart failure was produced by reducing the preload volume to 40 ml/kg BW/min and keeping the constant afterload pressure at 80 mmHg, propranolol was added until LAP exceeded 15 mmHg (condition B). Serial measurements were carried out with the device off and on in both conditions. Occasional ventricular arrhythmias were effectively controlled by lidocaine injection (50 mg IV), pH was appropriately corrected by sodium bicarbonate, pO_2 , pCO_2 , and electrolytes were controlled within physiological range. No other drugs were given during the experiments.

Fig. 6. Effect of D-PATCH on left atrial pressure (LAP) and left ventricular stroke work (SW) in group 2.

Fig. 7. Effect of D-PATCH on peak left ventricular pressure (LVP), LV systolic period, and tension time index (TTI) in group 2.

After device activation or inactivation, coronary blood flow reached a constant level within 5 minutes. Blood samples were taken at this steady state during each condition from the aortic root cannula and right ventricular cannula. Calculations of myocardial oxygen consumption (MVO_2) were based on $A-VO_2$ difference obtained directly with Lexicon determination, and was expressed as the product of coronary flow and coronary arteriovenous oxygen difference in milliliter/minute/100 gm of wet heart weight.

The tension time index (TTI) was calculated according to the method of Sarnoff and coworkers.¹⁰

In group 3, with a same setting as group 2, an additional Extra-aortic Counterpulsation Device (CP) was inserted to the brachiocephalic artery and the tip of the device was placed inside the aortic arch (Fig. 3). Ultrasonic piezoelectric crystals embedded in epoxy beads 3 mm in diameter were inserted in pairs approximately 15 mm apart in the left ventricular anterior wall. Crystal pairs were placed along the minor equator of the left ventricle, used in conjunction

with a multichannel sonomicrometer (Tohoku Univ. Apparatus). The pairs of ultrasonic crystals provided continuous measurements of chord length of the area which recorded on the oscillograph (Iwasaki SS-5100). Regional left ventricular dimensional measurements were made from the oscillograph recording of the chord as follows: (1) end-diastolic chord length (EDCL), (2) end-systolic chord length (ESCL), and (3) systolic shortening (SS) calculated as $SS = EDCL - ESCL$. To normalize the percent of systolic shortening was calculated as $SS/EDCL \times 100 (\%)$.

After reperfusion period volume loading was started from the pulmonary arterial line. Severe heart failure was produced by selective ligation of coronary artery and continuous infusion of propranolol. Keeping constant preload volume (40 ml/kg BW/min) and afterload pressure (50 mmHg), propranolol was added until LAP exceeded 15 mmHg if necessary. Serial measurements were carried out in the condition of (1) No assist: Control, (2) CP assist, (3) D-PATCH assist, and (4) Concomitant assist of D-PATCH and CP.

Fig.8. Representative left ventricular function curves, demonstrating the-effect of aortic counterpulsation (CP) assist (●---●), D-PATCH assist (△---△) and combined assist of CP and D-PATCH (○---○) in group 3.

Left ventricular function curves were obtained in each assisted condition by progressively increasing flow through the main pulmonary arterial cannula. Arterial pressure was maintained at 50 mmHg by appropriately decreasing flow through the aortic perfusion line, LAP was measured at 500 ml increments of flow between 500 and 4000 ml or until LAP exceeded 30 mmHg. Curves were then inscribed by plotting LAP against left ventricular stroke work (LVSW). Statistical analysis was performed by paired and unpaired t-test. Variables were expressed as mean $\pm$ SD.

RESULTS

1) Group 1

Whole heart Model: In mild heart failure (MAP < 90 mmHg, MLAP < 15 mmHg) MAP was increased 20% ($p < 0.01$), MLAP was decreased 16% ($p < 0.01$), and cardiac output (CO) was increased 17% ($p < 0.01$) by D-PATCH assist. In severe heart failure, typical hemodynamic effects were shown in Fig. 4, MAP was increased 36% ($p < 0.01$), MLAP was decreased 16% ($p < 0.01$), and C.O was increased 50% ($p < 0.01$).

2) Group 2

Hemodynamic indexes: Under strict control of preload volume (80 cc/kg/min in condition A, 40 cc/kg/min in condition B) and afterload (80 mmHg), LAP was decreased significantly with device activation ($41.3 \pm 10.6\%$ in condition A, $37.7 \pm 10.2\%$ in condition B, $p < 0.01$), and in

Fig.9. Representative hemodynamic effect of D-PATCH in severe heart failure in group 3. Note the remarkable effect of D-PATCH on LAP.

Fig.10. Effect of D-PATCH, CP, and combined assist of D-PATCH and CP on LAP, peak LVP, and TTI in group 3.

parallel with the LAP changes left ventricular stroke work was increased significantly (10.3 ± 3.0 in condition A, $11.0 \pm 3.6\%$ in condition B, $p < 0.01$). Although peak developed left ventricular pressure (peak LVP) was increased ($8.0 \pm 6.5\%$ in condition A, $12.7 \pm 9.2\%$ in condition B, $p < 0.05$), tension time index (TTI) was decreased significantly ($21.2 \pm 11.0\%$ in condition A, 25.7 ± 9.4 in condition B, $p < 0.01$) because of significant shortening of left ventricular systolic period (LVSP) ($16.4 \pm 6.4\%$ in condition A, $19.2 \pm 5.1\%$ in condition B, $p < 0.01$) (Fig. 5, 6, 7).

Coronary Blood Flow, MVO_2 : Directly measured coronary sinus blood flow was significantly decreased ($16.8 \pm 2.9\%$ in condition A, $18.0 \pm 6.6\%$ in condition B, $p < 0.001$) and coronary flow AV difference of oxygen content was also decreased, therefore, myocardial oxygen consumption (MVO_2) was decreased significantly ($24.4 \pm 6.5\%$ in condition A, $29 \pm 8.5\%$ in condition B, $p < 0.001$).

3) Group 3

Left Ventricular Function: Typical effect of devices on the left ventricular function curve is shown in Fig. 8. Mean LVSW was increased 70% ($p < 0.05$) by CP assist and 199% ($p < 0.02$) by D-PATCH assist. When heart is assisted by both CP and D-PATCH mean LVSW was increased

Fig.11. Representative effects of devices activation on the segmental shortening in the residual left ventricular anterior wall.

more remarkably (396%, $p < 0.002$). Comparing to CP assist the improvement in the left ventricular function curve by D-PATCH assist is more effective for the profound failure heart.

Hemodynamic Indexes: Typical effect of devices on hemodynamics is shown in Fig. 9. Under strict control of preload volume (40 ml/kg BW), afterload pressure (50 mmHg), and heart rate (120/min), mean LAP was decreased significantly with CP assist (53%, $p < 0.02$), D-PATCH assist (75%, $p < 0.006$), and concomitant assist of CP and D-PATCH (87%, $p < 0.006$). Peak LVP was significantly decreased by the systolic unloading effect of CP assist (20%, $p < 0.002$). Although peak LVP was increased 5.7% by single D-PATCH assist ($p < 0.006$), peak LVP was also significantly decreased (18.4%, $p < 0.002$) when heart is assisted concomitantly by D-PATCH and CP. Consequently TTI was decreased 14% ($p < 0.02$) by CP assist, 15% ($p < 0.02$) by D-PATCH assist, and 23% ($p < 0.01$) by concomitant assist of D-PATCH and CP (Fig. 10).

Lef Ventricular Regional Segmental Function: Typical effect of device activation on the segmental wall motion in the left ventricular anterior wall is demonstrated in the Fig. 11. Note that the segmental shortening of the ventricular wall was remarkably increased by D-PATCH assist. Although EDCL was not changed, ESCL was decreased 0.8% (NS) by CP assist, significantly decreased 4.0% ($p < 0.05$) by D-PATCH, and 5.0% ($p < 0.05$) by concomitant assist of D-PATCH and CP. Percent systolic segmental shortening was increased 9.8% (NS) by CP assist, 143% ($p < 0.02$) by D-PATCH assist, and 146.5% ($p < 0.02$) by concomitant assist of D-PATCH and CP.

DISCUSSION

Results of medical treatment of hospitalized patients with profound cardiogenic shock secondary to acute myocardial infarction have been extremely poor (hospital mortality: 85–100%).¹¹

In patients who undergo emergency coronary revascularization by percutaneous coronary thrombolysis or surgical aorto-coronary bypass, if patients have a large dyskinetic area in the left ventricular wall, it may be impossible to recover these patients from severe cardiogenic shock even under maximal medical support and IABP.^{1,12}

In these cases, it is beneficial and reason-

able to resect the dyskinetic area combined to emergency coronary revascularization.^{1,4}

In animal experiments, animals in which excision of more than 30% of the free left ventricular wall has been performed with direct suture closure technique did not survive longer than a few hours postoperatively.¹³ Bailey and Gilman³ concluded that the normal mammalian heart is unable to compensate adequately for the luminal reduction which is produced by resection of a large portion of left ventricular wall. While using nondynamic synthetic patch replacement of left ventricular myocardial infarcted area, Collins and Collins¹⁴ reported that extensive resection and nondynamic patch replacement of LV wall exerts a fairly profound detrimental effect on left ventricular function. Therefore, in the clinical setting it also seems reasonable to resect the large dyskinetic myocardium and replace it with a dynamic patch artificial myocardium which assists the left ventricular function with co-pulsation mechanism.

The recovery of the myocardial tissue from ischemic damage is mostly dependent on the balance of myocardial oxygen demand and supply.¹⁵ In the normally beating heart at a basal level, the determination of oxygen consumption depends 15–20% on the volume work, and 60–65% on the pressure work.¹⁶

In group 2 (right heart bypass model), under strict control of stroke volume and afterload during myocardial failure we demonstrated an increase in the peak developed left ventricular pressure ($8.0 \pm 6.5\%$ in condition A, $12.7 \pm 9.2\%$ in condition B) and stroke work ($10.3 \pm 3.0\%$ in condition A, $11.0 \pm 3.6\%$ in condition B). These increases were not produced by the natural myocardium but by D-PATCH, so that the myocardium did not need to expend additional energy in order to meet its increased peak developed tension and stroke work during heart failure. On the other hand, our results showed a definite decrease of the tension-time index ($21.2 \pm 11.0\%$ in condition A, $25.7 \pm 9.1\%$ in condition B) which is mostly due to the decrease of left ventricular systolic period ($16.4 \pm 6.4\%$ in condition A, $19.2 \pm 5.1\%$ in condition B). The effects produced by the decrease of global myocardial oxygen demand resulted in a parallel decrement of coronary blood flow and myocardial oxygen consumption. We do not believe that the observed coronary flow decrease is the result of an increase of coronary resistance mechanically produced by the device, otherwise MVO_2 would

not be decreased.

In group 1 (whole heart model), D-PATCH produces a significant increase of cardiac output (17.9% to 56.7%) and aortic pressure (30.4% to 33.6%). Therefore, we cannot directly extrapolate the results of advantageous myocardial energetics in group 2 to the whole heart model.

To obtain such a advantage in myocardial energetics in the whole heart, we have to avoid the extreme augmentation of peak left ventricular pressure while D-PATCH support. It seems to be reasonable to combine intra-aortic balloon pumping (IABP) to D-PATCH because the systolic unloading effect of aortic counterpulsation by IABP can compensate the excess peak LVP increment and the additional LV pressure work which produced by D-PATCH support as we have demonstrated in the results of group 3. We also consider that the concomitant use of D-PATCH and IABP is natural and reasonable in the clinical setting.

Marrin and his coauthor¹⁷ pointed out that IABP itself depends upon some degree of residual left ventricular function, if the residual heart function is too much deteriorated, single IABP support will not recover the heart function.

Therefore, intra-ventricular copulsation support by D-PATCH and intra-aortic counterpulsation support by IABP are considered to be one of the most ideal mechanical support combination for profound cardiogenic shock patient with a massive myocardial infarction.

In group 3, we also demonstrated by sonomicrometry measurements that the residual myocardium can work more vigorously under D-PATCH support, namely, the systolic segmental dimension of the LV wall was significantly decreased and segmental shortening was effectively increased during D-PATCH assist. We consider that the advantageous myocardial energetics under D-PATCH support may relate to the regional functional improvement in the residual LV wall when the heart is assisted by D-PATCH.

The results of this study suggest that the concomitant use of D-PATCH and CP can compensate their weak point each other and provides effective cardiac support while decreasing LAP, peak LVP, and TTI, thus, is particularly appropriate for the treatment of refractory cardiogenic shock due to massive acute myocardial infarction.

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