Effects of a Lack of Aortic “Windkessel” Properties on the Left Ventricle

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After the long bypass grafting between ascending and abdominal aorta with exclusion of the aortic arch using conventional synthetic vascular graft, a considerably remarkable hemodynamic change and progressive hypertrophy of the left ventricle occurred until stabilized. In the clinical cases and animal experiments, systolic hypertension, diastolic pressure decrease and consequent pulse pressure widening were observed. Furthermore, the phase difference between flow and pressure waves approximated to zero.

Elevation of the afterload due to systolic hypertension and widening of pulse pressure may result in energy loss in vascular pulsation, not maintaining forward flow but increasing the left ventricular external work. Furthermore, as the peak flow approximates the peak pressure and its point situates relatively early in systole, external work and wall stress of the left ventricle are markedly elevated. All those factors mentioned above lead concentric hypertrophy of the left ventricle to normalize the wall stress.

Fall in the diastolic pressure at the aortic root may decrease coronary flow to lead ischemia of the hypertrophied left ventricle. This can occasionally lead to fatal heart failure after a long postoperative period.

It may be concluded that these new findings are produced by a loss of compliance (Windkessel properties) in aortic root which occurred as consequence of using conventional synthetic vascular graft with exclusion of aortic arch.

The natural aorta, especially the aortic root, is a compliant vessel which expands according to the increment of the pressure to absorb energy of the left ventricular rectangular pressure during systole which is released in diastole. This function is known as a “Windkessel” property in the aorta. Conventional prostheses, such as woven Dacron vascular devices do not distend as the arterial pressure rises, although they can be elongated by the existence of crimp. The “Windkessel” properties, therefore, are absent in the conventional vascular prosthesis. The replacement or bypass surgery of the diseased aorta, which is currently performed routinely either anatomically or extra-anatomically using the conventional synthetic prosthesis, may change the properties of the aortic system. In this study, we investigated how much influence the change of the aortic properties might give upon the left ventricular morphology and function. The data were obtained from our clinical and experimental experiences of systolic hypertension and resulting left ventricular hypertrophy after major aortic reconstructive surgery.

MATERIAL AND METHODS

1. Clinical studies

The clinical studies were based on five patients in our surgical department (Fig. 1).

Case 1 was a 51-year-old male with a very large ruptured aneurysm of the aortic arch. We performed an extra-anatomical bypass procedure between the ascending and abdominal

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- Exclusion of aortic arch
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Fig.1. Cases of extra-anatomical bypass using long woven Dacron graft with exclusion of huge aortic aneurysm. Note in case 4: A bypass using a long knitted Dacron graft for decompression of aortic root.

Fig.2. The pull-back manometry of the ascending to abdominal aorta through and across the bypass graft in case 1. Reflected pressure wave and peaking and steepening phenomenon were observed along the graft.

aorta using a long woven Dacron graft 22 mm in diameter. Between the proximal graft and three major branches of aortic arch, we used a trifurcated Y-graft in combination with exclusion of the arch aneurysm.

Case 2 was a 51-year-old male with a large ruptured aneurysm of the thoraco-abdominal aorta. We performed the extra-anatomical bypass procedure between the ascending and abdominal aorta using a long woven Dacron graft 22 mm in diameter in combination with exclusion of the thoracic portion of the aneurysm.

Case 3 was a 60-year-old male with DeBakey II and IIIb dissecting aneurysm of the aorta. He received an extra-anatomical bypass between the ascending aorta and the replaced abdominal aortic Y-graft utilizing a long woven Dacron graft 22 mm in diameter. Another bypass was done to the excluded left subclavian artery in combination with the closure by a permanent clamp for the dissected thoracic aorta between the left common carotid and subclavian arteries by which thromboexclusion of the dissected aneurysm was expected.

Case 4 was a 48-year-old hypertensive female with coarctation of the abdominal aorta due to Takayasu aortitis. She received an extra-anatomical bypass grafting between the ascending and abdominal aorta distal to the stenosis using a long knitted Dacron graft 16 mm in
diameter.

Case 5 was a 49-year-old female with thoraco-abdominal aortic aneurysm. We performed an extra-anatomical bypass grafting from the descending aorta, just distal to the left subclavian artery, to the abdominal aorta using a long woven Dacron graft 20 mm in diameter. The thoracic portion of the aneurysm was excised.

During the postoperative follow-up of these patients, the electrocardiogram (ECG), echocardiogram (Echo) and chest roentgenogram were recorded. Cases 2 and 3 died 26 days and 6 days respectively after operation and were examined at autopsy.

2. Animal experiments

Six mongrel dogs weighing between 19 and 30 kg received 25 mg/kg sodium pentobarbital intravenously. After tracheal intubation and commencement of artificial respiration, the thorax was opened through the left third intercostal space. The pericardium was opened and woven Dacron graft was anastomosed to the ascending aorta similar to the clinical cases. The graft was 30 cm in length and 63–90% of the aortic diameter. The abdominal aorta was, thereafter, dissected extraperitoneally through left lower abdominal incision and was anasto-

mosed to the distal end of the graft with side-to-end fashion.

Cardiac output was measured by an electromagnetic flow probe (Nihon Kohden K.K.) and pressure manometry was performed in the ascending aorta, proximal and distal graft, and abdominal aorta through 18 gauge needles connected to pressure transducers (Toyo Baldwin Co., Ltd., Type DPLU-0.1). These circulatory parameters were measured at the state of clamping the graft (normal pathway) and after clamping of the ascending aorta just distal to the graft anastomosis with declamping the graft through

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**TABLE I BLOOD PRESSURE CHANGES, PRE- AND POST-BYPASS OPERATION**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Preoperation mmHg</th>
<th>Postoperation mmHg</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>165/80</td>
<td>120–180/75–80</td>
</tr>
<tr>
<td>2</td>
<td>140–160/90–110</td>
<td>160–190/60–80</td>
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<tr>
<td>3</td>
<td>132–166/96–104</td>
<td>170–180/80–90</td>
</tr>
<tr>
<td>4</td>
<td>278/70 (150)</td>
<td>200/75</td>
</tr>
<tr>
<td>5</td>
<td>124/70</td>
<td>124/70</td>
</tr>
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</table>
in case 1, but decreased from 20 mm preoperatively to 11–14 mm postoperatively in case 4. Postoperative LV relative wall thickness (R/Th), expressed as the ratio of end-diastolic radius (R) to wall thickness (Th), was 1.85 to 1.05 in case 1 and 2.17 in case 5. This showed marked concentric hypertrophy, especially in case 1. Postoperative LV ejection fraction was as high as 84–89% in all cases.

In case 1 which excluded the aortic arch aneurysm, the pullback pressure manometry of the ascending to abdominal aorta through and across the 25 cm long bypass graft was performed 1 month after operation (Fig. 2). The results were: 132/42 mmHg of aortic root pressure; 24 mmHg of pressure drop into the graft; and the appearance of reflected pressure wave next to the systolic pressure wave in the graft, the former of which was higher and both of which formed a biphasic peak and fused together toward the distal anastomosis. Moreover, an increasingly higher systolic pressure and the peaking and steepening phenomenon, increasing to the distal along the graft, were shown.

In two cases (2 and 3) concentric hypertrophy of the LV was revealed at autopsy, although preoperative values of $R_{V3} + S_{V1}$ on ECG was 4.8 mV and 3.2 mV respectively (Fig. 3). LV wall thickness was 20–25 mm in both cases by direct measurement at autopsy. Pressure manometry revealed systolic hypertension and concomitant decrease in diastolic pressure (Table 1).

2. Animal experiments

After the extra-anatomical bypass with conventional synthetic prosthesis, blood pressure in the aortic root was elevated from 117.5 ± 21.6 (control) to 160.9 ± 39.3 mmHg (after bypass) in systole, decreased from 79.5 ± 12.8 to 67.5 ± 11.3 mmHg in diastole, from 96.7 ± 14.4 to 100.5 ± 14.4 mmHg in mean pressure and pulse pressure was widened from 38 ± 12.8 to 96 ± 34.9 mmHg (p ≤ 0.001) (Fig. 4). Although heart rate did not change (140.0 ± 36.2/min), cardiac output was decreased from 1.82 ± 0.35 to 1.76 ± 0.35 L/min (p ≤ 0.005) and then vascular resistance was slightly increased from 4315 ± 589 to 4697 ± 764 dyne-sec-cm⁻² (p ≤ 0.001).

In two out of six experiments, systolic hypertension, diastolic hypertension and widening of the pulse pressure were observed after bypass, whereas cardiac output and vascular resistance were unchanged.
In a relationship between pressure and flow waves, the peak flow preceded the peak pressure before bypass. On the other hand, the peak flow approached the peak pressure after bypass and, therefore, the phase difference approximated to zero. In the ascending aorta, the peak of blood pressure had come almost at the end of cardiac ejection before bypass, whereas it came almost at the middle of cardiac ejection after bypass of the aortic arch (Fig. 5).

DISCUSSION

In three (cases 1, 2 and 3) of the four cases with long aortic bypass with conventional prosthesis between the ascending and abdominal aorta, rapidly and markedly developed LV hypertrophy was observed by means of ECG, Echo and autopsy. The hypertrophy was of the concentric type. The systolic hypertension and concomitant decrease in diastolic pressure were also revealed postoperatively. In case 4 in which aortic root was decompressed, however, the LV hypertrophy was gradually decreased as the blood pressure dropped. The LV was only slightly increased in case 5 bypassed from the descending to abdominal aorta with neither exclusion nor blind pouch formation of the aortic arch. The blood pressure was not elevated postoperatively. From these results it is clear that the LV concentric hypertrophy and concomitant systolic hypertension developed more markedly in the patients whose aortic arch had been excised or formed a blind pouch.

In the animal experiments, systolic hypertension, diastolic hypotension and consequent pulse pressure widening at the aortic root were also revealed after long aortic bypass grafting with cross clamping just distal to the ascending aorta. Vascular resistance was slightly elevated by 8.9% in consequence of 3.9% elevation of mean aortic pressure and 4.4% drop of cardiac output. The elevation of mean pressure was far smaller when compared with 153% widening of the pulse pressure. Furthermore, it was observed in two animal experiments that cardiac output, mean aortic pressure and systemic resistance were unchanged in spite of the marked widening of pulse pressure at the aortic root.

In general, increment of resistance elevates systolic, diastolic and mean blood pressure, but makes the pulse pressure smaller. On the other hand, a decrease in aortic compliance elevates systolic pressure, decreases diastolic pressure and then enlarges pulse pressure, but does not change mean pressure. It is possible that the circulatory changes at the aortic root after long aortic bypass grafting may be due to a decrease in compliance.
of the aorta caused by the aortic bypass grafting rather than increase in resistance. The ascending and arch aorta has a distinguished "Windkessel" function. However, synthetic vascular graft conventionally used is noncompliant and has no "Windkessel" property. It is of concern that compliance of the aortic root diminishes impedance to flow as a consequence of two components in piles. Those phenomena observed here have a strong resemblance to circulation in arterial degenerative disease like sclerosis of the aorta in which the blood pressure is increased during systole and is reduced in the ascending aorta during diastole. The elevation of systolic pressure increases the afterload of the LV; the fall of diastolic pressure, which is a major factor in determining coronary blood flow,^6^ may lead to relative myocardial ischemia.

The energy to maintain a forward blood flow in the aorta is given by mean pressure generated by the LV. From the concept that the pulsatile component of blood pressure loses energy in vascular pulsation,^6^ widened pulse pressure without change in mean pressure becomes a cause of large energy loss for maintaining the forward blood flow. The LV must, therefore, generate excessive energy to pump the same amount of blood through an unchanged peripheral resistance. This results in increase in LV work.

The phase difference between pressure and flow waves approximated to zero after the long aortic bypass. As the instantaneous cardiac work is obtained by the formula, flow x pressure, the cardiac work increases markedly when the peak flow approaches to the peak pressure. By the time the aortic root pressure reaches peak point, however, the LV can not eject enough because the LV can not contract enough and its chamber radius remains large. The wall of the LV is not sufficiently thickened, so ejects the blood into the aorta with very high pressure. As the LV wall stress (cm) is directly proportional to ventricular pressure (P) and chamber radius (R) and is inversely proportional to wall thickness (Th) from Laplace's equation, cm = P x R/Th, the LV wall stress elevates markedly in such a circumstance. Since stress peaks early in systole, wall thickness due to hypertrophy plays a greater role in minimizing peak stress than thickening due to contraction. Furthermore, it is believed that the most important factor controlling cardiac hypertrophy is the systolic force or tension generated by the myocardial fibers. Then, the LV may be hypertrophied to normalize the wall stress.

As reported, the exercise training-induced adaptive changes in LV dimensions occur rapidly. In the clinical cases we observed this kind of concentric hypertrophy of the LV can also rapidly develop. It was observed in case 3 that this change may have been within one week since the LV posterior wall thickness became 23 mm at the 6th postoperative day at autopsy, although the preoperative value of Rv3 + S1 on ECG was only 3.2 mV.

The patients' cardiac function, such as ejection fraction, was unchanged by speedy myocardial adaptation to the circulatory changes which led to markedly elevated cardiac work. However, it is important to note that LV hypertrophy accompanied by decrease of the effective coronary perfusion pressure may infrequently but positively lead to fatal heart failure after a long postoperative period.

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