Treatment of Acute Profound Heart Failure by Ventricular Assist Device

HISATERU TAKANO, M.D., TAKEI SHI NAKATANI, M.D., YOSHIYUKI TAE NAKA, M.D.
YOSHITUGU KITOH, M.D., KATSUHIRO HIRAMORI, M.D.
KAZUO HAZE, M.D., AKIRA ITOH, M.D., TSUYOSHI FUJITA, M.D.
AND HISAO MANABE, M.D.

Sixteen patients with acute profound heart failure (HF) have been treated with the left ventricular assist device (LVAD), nine of them were successfully weaned from LVAD, and three of them were discharged and survived longer. Decompression of the left ventricle (LV) at the beginning will prevent overextension of impaired myocardium and accelerate scar formation. Gradual increase of LV work will promote the compensation ability of the residual myocardium. We found that continuous LVAD assistance can give time for the impaired heart to recover while maintaining normal circulation. For patients with profound HF which is beyond the limit of intra-aortic balloon pumping’s (IABP) capability, LVAD is a more powerful and effective means. Although the heart recovered, many patients later died of multiple organ failure (MOF) which was probably caused by prolonged ischemia before LVAD application. For completely successful recovery from profound HF, diagnosis and deciding to use LVAD should not be delayed. It should be applied before major organs including the heart itself suffer irreversible damage. We have established a systematic therapeutic concept of treating acute HF patients using assisted circulation including LVAD. (Jpn Circ J 1992; 56: 100–110)

The use of intra-aortic balloon pumping (IABP) or venoarterial bypass has yielded good results for the treatment of profound heart failure (HF). However, limitations of the technique of IABP in cardiac assistance have also been recognized.1,2 The left ventricular assist device (LVAD) used to replace cardiac pump function is an effective means for treatment of profound HF beyond the limit of IABP’s capability.3–7 Despite this potent treatment, however, the results of treating acute and profound HF patients are still unsatisfactory.8–9 The purpose of this study is to report on our experience with an LVAD, which was originally developed in our institute, to discuss the problems associated with treatment of acute profound HF patients using LVAD to prolong survival, and to establish a systematic therapeutic concept of treating acute HF patients using assisted circulation including LVAD.

MATERIALS AND METHODS

Description of LVAD
The LVAD consists of blood pump and control-drive unit.

1. Ventricular Assist Blood Pump
Two sizes of our original pneumatic and diaphragm-type pumps are now available for clinical use. The effective stroke volume of the adult-sized pump is 70 ml and that...
TABLE I  OUTLINE OF THE PATIENTS TO WHOM LVAD WAS APPLIED FOR THE TREATMENT OF PROFOUND HEART FAILURE

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Operation</th>
<th>Indication</th>
<th>Assist</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>36</td>
<td>F</td>
<td>MSR+TR+Giant LA</td>
<td>MVR, TAP, LA Plication</td>
<td>Postop. LOS</td>
<td>LVAD</td>
</tr>
<tr>
<td>2.</td>
<td>62</td>
<td>F</td>
<td>AMI+VSP</td>
<td>Patch Closure, Resection of LV Infarcted Area</td>
<td>ECC dependent</td>
<td>LVAD</td>
</tr>
<tr>
<td>3.</td>
<td>52</td>
<td>M</td>
<td>ASR+MSR, AMI &amp; VF during CAG</td>
<td>AVR, CABG (1)</td>
<td>ECC dependent</td>
<td>LVAD+RVAD</td>
</tr>
<tr>
<td>4.</td>
<td>3</td>
<td>M</td>
<td>VSD+PH</td>
<td>Patch Closure</td>
<td>ECC dependent</td>
<td>LVAD</td>
</tr>
<tr>
<td>5.</td>
<td>66</td>
<td>F</td>
<td>MS</td>
<td>MVR (LV Rupture)</td>
<td>Intractable repair</td>
<td>LVAD</td>
</tr>
<tr>
<td>6.</td>
<td>66</td>
<td>M</td>
<td>OMI, AMI+VT during CAG</td>
<td>CABG (2)</td>
<td>ECC dependent</td>
<td>LVAD</td>
</tr>
<tr>
<td>7.</td>
<td>71</td>
<td>F</td>
<td>AMI+VSP</td>
<td>Patch closure</td>
<td>Elective use</td>
<td>LVAD</td>
</tr>
<tr>
<td>8.</td>
<td>60</td>
<td>M</td>
<td>AMI+Shock</td>
<td>CABG (1)</td>
<td>Card. Shock</td>
<td>LVAD</td>
</tr>
<tr>
<td>9.</td>
<td>26</td>
<td>M</td>
<td>Supravalvular AS</td>
<td>Aortoplasty (V-A bypass)</td>
<td>ECC dependent</td>
<td>LVAD</td>
</tr>
<tr>
<td>10.</td>
<td>69</td>
<td>M</td>
<td>AMI+Shock</td>
<td>(—)</td>
<td>Card. Shock</td>
<td>LVAD</td>
</tr>
<tr>
<td>11.</td>
<td>73</td>
<td>F</td>
<td>AMI+VSP</td>
<td>Patch Closure</td>
<td>ECC dependent</td>
<td>LVAD</td>
</tr>
<tr>
<td>12.</td>
<td>44</td>
<td>M</td>
<td>OMI+VT+LV Aneurysm</td>
<td>Aneurysmectomy Cryosurgery</td>
<td>Postop. LOS</td>
<td>LVAD</td>
</tr>
<tr>
<td>13.</td>
<td>73</td>
<td>M</td>
<td>AMI+Shock</td>
<td>(—)</td>
<td>Card. Shock</td>
<td>LVAD</td>
</tr>
<tr>
<td>14.</td>
<td>57</td>
<td>M</td>
<td>OMI+AP, AMI during PTC</td>
<td>CABG (3)</td>
<td>Postop. LOS</td>
<td>LVAD</td>
</tr>
<tr>
<td>15.</td>
<td>64</td>
<td>M</td>
<td>AMI+Shock</td>
<td>CABG (2)</td>
<td>Card. Shock</td>
<td>LVAD</td>
</tr>
<tr>
<td>16.</td>
<td>73</td>
<td>M</td>
<td>Ao Aneurysm</td>
<td>Replacement</td>
<td>ECC dependent</td>
<td>LVAD</td>
</tr>
</tbody>
</table>


of the pediatric pump is 20 ml, and the maximum output is 7.0 and 2.4 L/min, respectively. The surface in contact with blood is a newly developed Japanese made medical grade segmented polyether polyurethane.10,11

2. Automatic Control-Drive Unit (CDU)

The main part of our CDU consists of an automatic bypass flow control system based on automatic level control of left atrial pressure (LAP) and total systemic flow (TF). It is desirable to change bypass flow through the pump closely following changes in cardiac function. Since function of the failing heart is reflected in LAP levels, the automatic level control of LAP was adopted as the primary control method for bypass flow. Based on this method, with changes in cardiac function, LAP fluctuates, thus activating the automatic level control system for LAP and consequently changing bypass flow to maintain LAP at the preset level. An automatic level control system for total systemic flow has also been incorporated as the secondary control of bypass flow. Both LAP and total systemic flow of patients are maintained within preset range with this system during heart failure and the bypass flow can be changed closely following changes in cardiac function.12,13

Clinical Cases, Indication, and Weaning Procedure

Since 1982 sixteen patients with acute profound HF, detailed in Table I, have been

treated with the LVAD. Patients' age ranged from 3 to 73 years and the patients consisted of 11 males and 5 females. Ten cases suffered from acute myocardial infarction (AMI). The major indication for use of LVAD was dependence on extracorporeal circulation (8 patients), cardiogenic shock following AMI (3 patients), and postoperative low output syndrome (3 patients). Two patients with cardiogenic shock after AMI were treated without simultaneous coronary artery bypass grafting.

The main criteria of profound HF are cardiac index (CI) below 2.0 L/min/m² in adult and 2.3 L/min/m² in pediatric cases, systolic arterial pressure below 80 mmHg, and LAP or pulmonary wedge pressure above 18 mmHg for left heart failure and for right heart failure, right atrial pressure (RAP) above 18 mmHg but LAP below 5 mmHg. Other criteria include urine output below 0.5 mL/h, mixed venous oxygen saturation below 65%, and arteriovenous oxygen difference above 7.0 Vol%. Clinical impressions are also important. If the hemodynamic condition continued within these limits in spite of medical therapy and the use of IABP, LVAD was applied.¹⁴

All pumps were implanted between the right side of the left atrium and the ascending aorta under extracorporeal circulation. Since our blood pump is developed for the purpose of temporary use, the pump is

---

Fig. 2. Hemodynamic changes of Case 11 supported with LVAD. HR: heart rate, mAoP: mean arterial pressure, mPAP: mean pulmonary arterial pressure, mLAP: mean left atrial pressure, mRAP: mean right atrial pressure, TF: total flow, BF: bypass flow, VSR: ventricular septal rupture, POD: postoperative day.

Fig. 3. Hemodynamic changes of Case 6 supported with LVAD. HR: heart rate, mAoP: mean arterial pressure, mPAP: mean pulmonary arterial pressure, mRAP: mean right atrial pressure, mLAP: mean left atrial pressure, AMI: acute myocardial infarction, CAG: coronary angiography, VT: ventricular tachycardia, PD: peritoneal dialysis, Vf: ventricular fibrillation.

placed extracorporeally on the chest wall.

When the recovery of the function of the natural heart is observed hemodynamically and by ultrasonic cardiography, the weaning from the LVAD is initiated by increasing the value of the LAP of the preset range of the automatic level control system. This adjustment decreases bypass flow. LAVD will be discontinued when cardiac output exceeds 2.5 L/min/m², with LAP higher than normal, but less than 18 mmHg⁹,¹⁴

RESULTS

Duration of LVAD assistance was between 1 hour and 41 days. Nine patients were successfully weaned from LVAD. Three patients survived longer and were discharged. Five patients could not be weaned from LVAD.

Hemodynamic Changes Before, During and After LVAD Application

Figure 1 shows hemodynamic changes before, during, and after LVAD application in 9 patients who were weaned from LVAD. Most cases were associated with right HF, but an LVAD alone could maintain the normal circulation when RAP was kept at a higher level of 15 mmHg and by the use of catecholamines. Right HF recovered within 3 days. But in some cases, which suffered biventricular failure, RAP remained higher than normal range.

At the beginning, LAP was intentionally kept below 5 mmHg in order to reduce left ventricular (LV) work sufficiently and to maintain total systemic flow at a somewhat higher level of 3.0 L/min/m². During the recovery period, the preset level of LAP was gradually increased. Thus, automatic level control of LAP worked as expected and bypass flow decreased. Heart rate decreased when LVAD was applied, and increased as the preload of LV increased.

The patients' entire circulation was well maintained during the period of 3—15 days. The failed heart gradually recovered and LVAD was successfully removed when the CI exceeded 2.5 L/min/m². After removal of LVAD, heart rate, RAP, and LAP were still higher than normal, but tended to decrease gradually with time.

Representative Hemodynamic Changes

1. A case weaned from LVAD and discharged

Figure 2 shows an AMI case (patient 11) with ventricular septal perforation. In the initial stage of LVAD assistance the patient

---

TABLE II RESULTS, COMPLICATION, AND REMARKS OF THE PATIENTS WHO WERE WEANED FORM LVAD

<table>
<thead>
<tr>
<th>No.</th>
<th>Lung</th>
<th>Kidney</th>
<th>Liver</th>
<th>DIC</th>
<th>Brain</th>
<th>Infection</th>
<th>Thrombus</th>
<th>Results</th>
<th>Cause of Death</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td></td>
<td>Mitral</td>
<td>Death</td>
<td>MOF, Infection</td>
</tr>
<tr>
<td>2</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>Hospital</td>
<td>Death</td>
<td>Cerebral bleeding</td>
<td>Delayed application (LOS: 24 h)</td>
</tr>
<tr>
<td>4</td>
<td>⬤*</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>Lung</td>
<td>Death</td>
<td>Respiratory failure</td>
<td>Delayed application (Prolonged ECC: 7 h) (*Pulmonary infarction)</td>
</tr>
<tr>
<td>6</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td></td>
<td>Death</td>
<td>MOF</td>
<td>Delayed application (Cardiac massage: 1 h)</td>
</tr>
<tr>
<td>7</td>
<td></td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td></td>
<td>Alive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td></td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td></td>
<td>LV Death</td>
<td>MOF, Infection</td>
<td>Delayed application (LOS: 14 h)</td>
</tr>
<tr>
<td>11</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td></td>
<td>Alive</td>
<td>Pneumonia</td>
<td>(*Cerebral infarction)</td>
</tr>
<tr>
<td>13</td>
<td></td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td></td>
<td>Late Death</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td></td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td>⬤</td>
<td></td>
<td>Alive</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


---

TABLE III CAUSES OF DEATH AND REMARKS OF THE PATIENTS WHO WERE NOT WEANED FROM LVAD

<table>
<thead>
<tr>
<th>No.</th>
<th>Assist</th>
<th>Cause of Death</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>4 hrs</td>
<td>Peripheral insufficiency</td>
<td>Delayed application (Cardiac massage: 1.5 hr)</td>
</tr>
<tr>
<td>5</td>
<td>22 hrs</td>
<td>Bleeding</td>
<td>Inadequate application (Incomplete operation)</td>
</tr>
<tr>
<td>9</td>
<td>3 hrs</td>
<td>Resp. failure, Brain bleeding</td>
<td>Inadequate application (Post V-A bypass)</td>
</tr>
<tr>
<td>10</td>
<td>12 days</td>
<td>Respiratory failure</td>
<td>Impossible recovery (Intractable arrhythmia)</td>
</tr>
<tr>
<td>12</td>
<td>6 days</td>
<td>Multiple organ failure</td>
<td>Delayed application (LOS: 20 hrs)</td>
</tr>
<tr>
<td>15</td>
<td>41 days</td>
<td>Multiple organ failure</td>
<td>Impossible recovery (Broad infarction)</td>
</tr>
<tr>
<td>16</td>
<td>1 hr</td>
<td>Respiratory failure</td>
<td>Delayed application (Prolonged ECC)</td>
</tr>
</tbody>
</table>


Fig. 4. The pathological appearance of the left ventricle of Case 1 stained with hematoxylin-cosin (left) and Masson-trichrome (right).

had biventricular failure, but LVAD alone maintained the normal circulation by keeping RAP at a higher level, and with the use of catecholamines. At the beginning, bypass flow stayed high to reduce LV work sufficiently and to maintain total systemic flow at a somewhat higher level of around 3.0 L/min/m². The total circulation was maintained by LVAD alone in the first two days. On the 3rd day weaning from LVAD was attempted. The preset level of LAP was gradually raised. Automatic level control of LAP was successful and subsequently bypass flow decreased. The failed heart gradually recovered and when the CI exceeded 2.5 L/min/m², LVAD was removed. Heart rate was high, above 140 beats/min, after removal of LVAD, but gradually decreased. The patient was discharged from the hospital and is doing well.

2. A case weaned from LVAD but died after removal

Case 6 had one and a half hours of cardiac massage following AMI with shock, and then was put on extracorporeal circulation (Fig. 3). The heart recovered smoothly and LVAD was successfully discontinued. However, the patient needed peritoneal dialysis and plasma exchange, and died of multiple organ failure (MOF).

Complications and Remarks on Cases Who were Weaned

Table II shows complications and remarks on the patients who were weaned from LVAD. All circles indicate complications and closed circles indicate some intensive treatments were required. In spite of a high weaning rate from LVAD, eventually only three patients could leave the hospital. Other patients died of renal, hepatic, respiratory and/or other organ failure which probably originated from the prolonged period of low cardiac output before LVAD application. A close correlation was seen between the severity of complications and delayed application. Cerebral infarction occurred in two patients. Three patients who were treated with the LVAD immediately after their circulatory condition deteriorated were discharged from the hospital in a satisfactory condition.

Cause of Death and Episode in Non-weaned Cases

Table III shows the causes of death and remarks on the patients who were not weaned from the LVAD. There was con-
considerable delay before the application of LVAD in non-weaned cases. One of the major causes of death in patients unable to be weaned from the LVAD was intracable HF.

The biochemical findings in non-weaned cases were worse, due to HF, respiratory failure, multiple organ failure (MOF), and infection.

Fig. 6. Ring slice of the heart which did not recover from profound heart failure shows extensive hemorrhagic necrosis (Case 15).

Pathological Findings

1. Pathological Findings of Main Organs

Autopsies were performed in all cases, except the survivors. Microscopic findings of major organs in a case who was weaned from LVAD but needed dialysis and plasma exchange revealed patchy fibrosis was scattered amongst normal tissues.

2. Pathological Examination of the Heart

Pathological examination of the heart, which was exposed to low cardiac output postoperatively, revealed that the remaining surviving areas of myocardium were surrounded by granulation and connective tissue which replaced the necrotic degenerated myocardium. Newly developed connective tissue was found strongly bound with each areas of myocardium (Fig. 4).

In AMI cases, infarcted myocardium changed into granulation tissue in the patient who died shortly after using LVAD, or was replaced with fibrous tissue and became solid scar in the patient who died over one month after AMI as shown in Fig. 5 (right lower picture). On the other hand, residual myocardium became hypertrophic as compared with a normal myocardium as shown in Fig. 5 (right upper picture). Left picture of Fig. 5 shows a ring slice of case 2 with

ventricular septal perforation after AMI. The reduced LV capacity after infarctectomy returned to almost the normal size.

Emergency coronary artery bypass graftings were performed in 4 cases. However, in some cases in which grafting was performed 3 h after onset, the heart showed hemorrhagic necrosis. One case, who underwent 2 grafts 4 h after onset of AMI, did not recover from HF, and the heart showed extensive hemorrhagic necrosis (Fig. 6).

**Thrombus Formation Inside the Left Ventricle**
Growing thrombus echo was observed in LV cavity with poor LV function in 2 cases who suffered from AMI and in a case who underwent mitral valve replacement with a pericardial xenograft.

**DISCUSSION**

IABP is a pressure support system and from our previous study15 its capacity to increase the CI is 0.44±0.22 L/min/m². The LVAD used to replace cardiac pump function is an effective means for treatment of profound HF beyond the limit of IABP's capability3–7 Objectives for temporary use of LVAD are to maintain the normal circulation irrespective of the severity of HF and to restore the failing heart by gradually decreasing the bypass flow as the heart recovers. However, the results of treating acute and profound HF patients are still unsatisfactory.

From our experience with an LVAD, the problems associated with treatment of acute profound HF patients for improvement of survival rate were discussed and a systematic therapeutic concept of treating acute HF patients using assisted circulation including LVAD was established.

**Recovery from profound HF**
The severely impaired heart can not maintain the normal circulation without LVAD assistance. Especially after inadequate myocardial protection during anoxic arrest, the heart can not contract strongly enough due to the lack of high energy phosphate, as well as the presence of dead myocardium16,17. However, after recovery from profound HF by LVAD the heart can maintain almost normal circulation. Pathological examination of the heart shows that the necrotic myocardium has been replaced with connective tissue and resident myocardium becomes
hypertrophic. As we previously reported in animal experiments, removal of overload and decompression of LV will prevent overextension of impaired myocardium and simultaneously accelerate the scar formation, which will help prevent dyskinetic movement and can effectively transmit myocardial contraction. Gradual increase of LV work will promote compensation by the residual myocardium. Continuous LVAD pumping can gain time for the impaired heart to recover while maintaining normal circulation. Appropriate, safe, and automatic circulation control during HF and subsequent recovery from profound HF can be achieved by our automatic LVAD.

Cause of Death During and After LVAD Assist

1. Failure to be weaned from LVAD
   One of the major causes of failure to be weaned from the LVAD was unrecoverable HF or combined profound right HF when LVAD alone was used. Since most cases were associated with right HF, if LVAD alone could not maintain the normal circulation with RAP above 15 mmHg and catecholamines, a right ventricular assist device (RVAD) should be applied without delay. For unrecoverable HF, as shown in Fig. 6, heart transplantation should be carried out but at present in Japan this is not common practice.

2. Multiple Organ Failure as a Complication of LVAD Use
   In our clinical experience, 56% of patients could be weaned from LVAD. This means that the damaged heart which cannot maintain systemic circulation in the early stages of acute HF might recover if the patient is promptly treated by LVAD.

   However, some of the patients who recovered from acute profound HF needed dialysis or plasma exchange and could not survive longer. Biochemical profiles and microscopic studies of major organs indicate that major organs were exposed to prolonged ischemia before LVAD assistance. It is assured that ineffective cardiac massage or low cardiac output continued for many hours prior to LVAD application in our cases. If the use of LVAD was delayed, damage to major organs would follow even though the heart might recover from profound HF. It should be kept in mind that the decision to apply the use of LVAD should not be delayed. It should be applied before major organs including the heart itself are irreversibly damaged. Establishment of the criteria for contraindication is also important.

Emergency CABG
   The life-saving effect of emergency coronary artery bypass grafting (CABG) still remains uncertain. The most appropriate time for CABG is unclear and delaying CABG may not only be ineffective as regards recovery of heart function but may even result in hemorrhagic necrosis of the ischemic myocardium. The patient in shock is occasionally intolerant to major surgery. This suggests that if an ischemic attack does not occur and the time for CABG to be performed exceeds 3h after the onset of AMI, LVAD alone may be enough to support the patient in cardiogenic shock, and CABG can be considered after recovery from the shock.

Thrombus Formation Inside the Left Ventricle
   Hypokinetic or akinetic heart is one of the risk factors for thrombus formation. Little attention has been paid to the heart as an origin of thrombus formation under LVAD pumping except when artificial prostheses such as valves were used. Prevention of LV thrombus is important to reduce the chance of systemic thromboembolism. For prophylaxis and treatment of thromboembolism, systemic anticoagulation should be considered. However, blood in the LV is not affected if the LV can not contract effectively, but systemic bleeding tendency is increased. For such patients intra-LV cavity anticoagulation should be applied. Even if the LV dysfunction is severe, anticoagulant agents enter the LV cavity directly. Required doses are adequate to reduce the possibility of thrombus formation, and do not increase the systemic bleeding tendency.

Therapeutic Strategies of Acute Profound HF
   It is important to establish a systematic therapeutic concept of treating acute profound HF patients with cardiogenic shock using assisted circulation including LVAD. Our concept is shown in Fig. 7.

Initially, medical therapy including volume
control is applied to these patients. If the hemodynamic condition is at the borderline of or within the criteria of profound HF in spite of vigorous medical therapy, we will go ahead and apply IABP. Despite IABP assistance, if hemodynamic condition is still within the criteria, we will then apply LVAD.

1) **Timing of LVAD Application**

When low cardiac output continues for a long time, other major organ dysfunctions become prominent and compromise the patient’s condition even if the failing heart itself recovers. Timing of the LVAD application is one of the important factors in patient survival.

Our criteria of LVAD application following IABP are; 1) Hemodynamics still deteriorate in spite of use of IABP, 2) Hemodynamics are unchanged 1h after use of IABP, 3) Hemodynamics stay in criteria of profound HF 3h after use of IABP.

2) **Prophylactic Use of IABP in Special Cases**

In high-risk patients undergoing cardiac surgery, we apply IABP prophylactically. In myocardial ischemia intractable to drug therapy or infarction such as unstable angina, impending infarction, or intractable ventricular arrhythmia, we also apply IABP for prevention of worsening myocardial ischemia or infarction.

3) **LVAD Application Without IABP**

If the patient’s condition is beyond the limits of IABP’s capability from the beginning, LVAD will be used without a trial of IABP. We apply LVAD directly when CI is less than 2.0 L/min/m² or the flow of extracorporeal circulation is greater than 1.0 L/min/m².

4) **LVAD Application in Cardiogenic Shock After AMI**

AMI patients with shock are to be treated bearing in mind the possible use of LVAD.

The following patients group are considered as candidates for LVAD application, because the survival rates of these patients are poor even after the use of potent medical therapy and IABP; 1) Killip 4th and Forrestet subset 4th stage of cardiogenic shock, 2) Killip 4th and old myocardial infarction, 3) Killip 3rd and ventricular septal perforation, 4) Free LV wall rupture.

For these patients IABP is immediately introduced percutaneously, but we apply LVAD to maintain the circulation and to prevent the overload of impaired myocardium without delay. Surgical treatments are also performed in ventricular septal perforation or free wall rupture. In these cases, LVAD is used after surgery when low cardiac output persists or suture deficiencies are suspected.

5) **Criteria for RVAD application**

Usually LVAD is effective when pulmonary vascular resistance is low but RVAD should be applied when the CI is less than 2.0 L/min/m² in spite of high RAP of 18 mmHg, even after administration of adequate pharmacologic therapy. RVAD application should be considered when right HF is persistent even if LVAD can maintain the total circulation.

**Step-by-Step Weaning**

The best weaning procedure from the LVAD in profound HF is a step-by-step weaning according to the recovery of the heart. At the beginning of LVAD application, IABP is stopped and catecholamine therapy should be tapered down as soon as possible. In the initial stage, bypass flow was kept high to keep LAP at the preset level (0-5 mmHg) and to maintain total systemic flow (3.0 L/min/m²). During the recovery stage of HF, the preset level of LAP was gradually increased. When the natural heart output exceeds 2.5 L/min/m² in adult and 2.7 in pediatric cases, and LAP below 18 mmHg, LVAD can be withdrawn. Appropriate, safe, and automatic circulation control during HF and subsequent recovery from profound HF can be achieved by our automatic LVAD.

**REFERENCES**

4. WOLNER E, DEUTSCH M, LOSERT U, et al: Clinical application of the ellipsoid left heart assist


