Dynamic cardiomyoplasty in patients with end-stage heart failure: anaesthetic considerations

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Dynamic cardiomyoplasty is used increasingly for patients with chronic heart failure, with approximately 500 cases having been performed. The latissimus dorsi muscle is prepared maintaining its vascular supply and the muscle flap is wrapped around the heart and connected to a cardiomyostimulator. The muscle is later stimulated synchronously with ventricular systole to augment the heart. Our experience of 22 patients with chronic heart failure (NYHA III–IV) undergoing dynamic cardiomyoplasty is described from the anaesthetist’s point of view. Two patients are reported as case reports. The challenge is to manage patients with severely impaired left ventricular function, who do not obtain immediate benefit from the operation. Our experience supports the importance of early use of inotropic agents.

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Although heart transplantation is effective and safe for patients with severe heart failure, this procedure is limited by lack of donor organs and the need for immunosuppressive therapy for the rest of the patient’s life. In 1985, Carpentier and Chachques¹ introduced dynamic cardiomyoplasty as an alternative to heart transplantation in patients with chronic heart failure. With this operation the latissimus dorsi muscle is mobilized, drawn into the thorax and wrapped around the heart. The muscle is then connected to a cardiomyostimulator and later stimulated synchronously with ventricular systole. With this technique there is no need for immunosuppression and there is no limitation of donor organs. Worldwide clinical experience is of more than 500 cases.² Our experience with this operation is reported and the principles of anaesthetic management of these patients are presented.

Surgical procedure

The operation consists of two steps. First, the patient is placed on the right side and the left latissimus dorsi muscle is prepared maintaining its vascular supply. The muscle is connected to electrodes and tested electrophysiologically. During this test, absence of neuromuscular block is essential. The muscle is then placed in the left hemithorax through lateral thoracotomy. Second, the patient is placed in the supine position. After median sternotomy and pericardiotomy, the muscle is wrapped around and fixed at the heart. Cardiopulmonary bypass (CPB) is not necessary, but should be immediately available for emergency. After having placed epicardial sensing electrodes, a cardiomyostimulator (Medtronic, Denver, CO, USA) is implanted. The muscle is not stimulated at the end of operation. The first stimulation starts 2 weeks after surgery. Three months after surgery the trained muscle is finally stimulated in a two-to-one mode, so that the muscle is stimulated with every second heart beat.

Anaesthetic management and own experiences

From April 1993 to July 1997, 22 patients (aged 33–67 yr) underwent dynamic cardiomyoplasty. There were 16 men and six women. Two patients had ischaemic cardiomyopathy (ICMP) and the others had a dilated cardiomyopathy (DCMP). Mean left ventricular ejection fraction (LVEF) was 23.6 (SD 5.8) % and mean left ventricular end-diastolic pressure (LVEDP) was 20.7 (11.6) mm Hg (Table 1). Five patients were classified as NYHA IV and 17 as NYHA III. Mean duration of operation was 321 (range 200–465) min.

All patients received oral premedication with flunitrazepam 1–2 mg. Except for digitalis and diuretics, patient medication was continued until the morning of surgery. Before induction of anaesthesia a peripheral vein, the right radial artery and the right internal jugular vein were cann-
 Anaesthesia and cardiomyoplasty

lated under local anaesthesia. A two-lumen central venous catheter and a pulmonary artery catheter were inserted via the right internal jugular vein. In patients with severe pulmonary hypertension or an ejection fraction <30%, we suggest haemodynamic monitoring before induction of anaesthesia.

Anaesthesia was induced with sufentanil 2–3 µg kg⁻¹ and flunitrazepam 0.01 mg kg⁻¹ i.v. After the start of continuous neuromuscular monitoring (Relaxograph, Datex, Helsinki, Finland), a short-acting, non-depolarizing neuromuscular blocking drug (atracurium 0.5 mg kg⁻¹) was given to facilitate tracheal intubation. No further neuromuscular blocking drugs should be used because at the end of the preparation of the muscle, its function is tested electrophysiologically. After this test, neuromuscular blocking drugs can be used if necessary. Short-acting, non-depolarizing neuromuscular blocking drugs (e.g. atracurium) should be used because additional electrophysiological tests may be necessary. After intubation, the patient’s lungs were ventilated with a mixture of oxygen in air to maintain $P_{A_{O_2}} > 10.7$ kPa and $P_{A_{CO_2}}$ at approximately 5.3 kPa. Anaesthesia was maintained using continuous infusion of sufentanil 1–2 µg kg⁻¹ h⁻¹ and additional i.v. boluses of flunitrazepam. If necessary, isoflurane was added up to 1 MAC. Routine monitoring consisted of a five-lead ECG, end-tidal carbon dioxide, pulse oximetry, body temperature, urine output, neuromuscular block, respiratory monitoring, arterial pressure, pulmonary artery pressure (PAP), pulmonary capillary wedge pressure (PCWP), continuous cardiac output (CCO, using a thermodilution technique (Vigilance; Baxter, Irvine, CA, USA)) and continuous mixed venous oxygen saturation ($S_{V_{O_2}}$). Arterial and mixed venous blood gases were obtained if necessary.

If cardiac index (CI) was less than 2.5 litre min⁻¹ m⁻² after induction of anaesthesia, we gave dobutamine to increase CI; 19 of 22 patients received continuous infusion of dobutamine. The mean dose was 4.4 (range 2.8–11.3) µg kg⁻¹ min⁻¹ (Table 2). Three patients did not achieve a CI >2.5 litre min⁻¹ m⁻² even when dobutamine was increased to 12 µg kg⁻¹ min⁻¹. As systemic and pulmonary vascular resistances were normal, we gave epinephrine to achieve an adequate CI. Interestingly, these were two patients with ischaemic cardiomyopathy, whereas only one patient suffering from dilatational cardiomyopathy required epinephrine. One of these patients died intraoperatively (see case No. 2) and one patient died on the third day after operation as a result of an irreversible low output syndrome. If an adequate CI of >2.5 litre min⁻¹ m⁻² cannot be achieved with inotropic support, the perioperative risk of patients undergoing cardiomyoplasty appears to be increased.

Fourteen of 22 patients received nitroglycerin at a mean dose of 0.91 (range 0.42–2.8) µg kg⁻¹ min⁻¹ because of an increase in filling pressures (CVP, PCWP). Nineteen patients had sinus rhythm during operation. Three patients had known atrial fibrillation, and one patient with a previous sinus rhythm presented with new atrial fibrillation which was cardioverted into sinus rhythm before surgery. During preparation of the muscle, no arrhythmia was noted, whereas during wrapping of the muscle around the heart, ventricular arrhythmias were common. Arrhythmia consisted mainly of multifocal ventricular premature beats caused by mechanical irritation of the heart by the surgeon. Ventricular tachycardia or ventricular flutter did not occur. Ten patients received lidocaine (lignocaine) 100–200 mg i.v. to treat premature ventricular beats and in four patients continuous infusion of lidocaine was necessary.

There was no deterioration in pulmonary function during placement of the muscle into the thorax or during wrapping of the muscle around the heart.

We gave packed red blood cells if haemoglobin decreased to less than 85 g litre⁻¹ or $S_{V_{O_2}}$ was less than 70%, despite adequate arterial oxygenation and CI >2.5 litre min⁻¹ m⁻². Four patients received 2 u. (600 ml) and one patient received 3 u. (900 ml). No patient received fresh frozen plasma or platelet concentrates.

Warming blankets and low-flow anaesthesia (flow 1.0–1.5 litre min⁻¹) were used in all patients to prevent cooling. Additionally, fluids were warmed to 37.0°C. Keeping the patient warm is essential as the procedure may last up to 5 h. The lowest temperature measured in the bladder during operation was 34.6±0.9°C, and temperature at the end of surgery was 34.9±0.9°C.

In all patients, the trachea remained intubated and the lungs ventilated after surgery. The trachea was extubated when standard criteria for extubation were reached: the patient must

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**Table 1** Patient and perioperative data (mean (SD) [range] or number). LVEF= left ventricular ejection fraction; LVEDP= left ventricular end-diastolic pressure; DCMP= dilated cardiomyopathy; ICMP= ischaemic cardiomyopathy

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Sex (M/F)</th>
<th>NYHA II/IV</th>
<th>LVEF (%)</th>
<th>LVEDP (mm Hg)</th>
<th>DCMP/ICMP 2/2</th>
</tr>
</thead>
<tbody>
<tr>
<td>55.6 [33–67]</td>
<td>73.6 (11.8) [50–93]</td>
<td>178 (23) [164–189]</td>
<td>16/6</td>
<td>18/4</td>
<td>23.6 (5.8) [15–30]</td>
<td>20.7 (11.6) [8–40]</td>
<td>5</td>
</tr>
</tbody>
</table>

**Table 2** Cardiovascular agents used in patients undergoing dynamic cardiomyoplasty (mean (SD) [range])

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose (µg kg⁻¹ min⁻¹)</th>
<th>n</th>
<th>Dose (µg kg⁻¹ min⁻¹)</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td>4.7 (2.7) [2.8–11.3]</td>
<td>19</td>
<td>0.91 (0.06) [0.42–2.8]</td>
<td>14</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>0.01 mg kg⁻¹ i.v.</td>
<td>3</td>
<td>1.7 (0.2) [1.4–2.5]</td>
<td>10</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>0.9 [0.9–8.10]</td>
<td>14</td>
<td>1.7 (0.2) [1.4–2.5]</td>
<td>10</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>0.91 (0.06) [0.42–2.8]</td>
<td>14</td>
<td>1.7 (0.2) [1.4–2.5]</td>
<td>10</td>
</tr>
</tbody>
</table>

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**Note:** The data in Tables 1 and 2 are rounded to two decimal places.
be awake, without signs of centralization, with a \( P_{\text{aO}_2} \) of >10.7 kPa while breathing spontaneously with a continuous positive airway pressure (CAP) and an \( F_{\text{O}_2} \) of less than 0.5 or \( P_{\text{aCO}_2} \) less than 6.7 kPa using a pressure support (ASB: assisted spontaneous breathing) of 10 cm H2O. In our patients, the trachea was usually extubated within 12–36 h after surgery. Postoperative analgesia was provided by patient-controlled analgesia (PCA) using opioids (piritramide).

### Case reports

**Patient No. 1**

A 54-yr-old male was scheduled for cardiomyoplasty caused by chronic heart failure (NYHA III). He had suffered myocardial infarction in 1989 and 1995. Ventricular angiography showed two-vessel disease with occlusion of the RCA and stenosis of the left anterior descending artery (LAD) which was treated successfully by implantation of a stent. No other revascularization was indicated. LVEF was 28% and LVEDP 17 mm Hg. The patient refused cardiac transplantation. Haemodynamic monitoring was started before induction of anaesthesia, arterial pressure decreased from 100/50 to 75/45 mm Hg just before intubation and PAP and PCWP increased further. Haemodynamic stability was obtained with epinephrine 3 \( \mu \text{g kg}^{-1} \text{min}^{-1} \) and right circumflex artery (RCX) (40%) were confirmed. After starting haemodynamic monitoring in the awake patient, infusion of 200 ml of a colloid solution caused an increase in filling pressures. PAP increased from 15/3 to 43/18 mm Hg, PCWP from 3 to 13 mm Hg and CVP from 0 to 9 mm Hg. Dobutamine 4 \( \mu \text{g kg}^{-1} \text{min}^{-1} \) was started. At induction of anaesthesia, arterial pressure decreased from 100/50 to 75/45 mm Hg just before intubation and PAP and PCWP increased further. Haemodynamic stability was obtained with epinephrine 3 \( \mu \text{g kg}^{-1} \text{min}^{-1} \) while dobutamine was continued. The patient remained stable with a greater dose of dobutamine (maximum dose 6.7 \( \mu \text{g kg}^{-1} \text{min}^{-1} \)). As CI increased, dobutamine was reduced, but the patient did not tolerate stopping dobutamine infusion. Surgery lasted 290 min and the lowest temperature was 34.4°C. The total dose of sufentanil was 23.7 \( \mu \text{g kg}^{-1} \) and total dose of flunitrazepam was 16.1 \( \mu \text{g kg}^{-1} \).

**Patient No. 2**

A 67-yr-old man with chronic heart failure caused by dilated cardiomyopathy (NYHA III). At left ventricular angiography, LVEDP was 28 mm Hg, LVEF was 17%, and moderate stenosis of the right coronary artery (RCA) (30%) and right circumflex artery (RCX) (40%) were confirmed. After starting haemodynamic monitoring in the awake patient, infusion of 200 ml of a colloid solution caused an increase in filling pressures. PAP increased from 15/3 to 43/18 mm Hg, PCWP from 3 to 13 mm Hg and CVP from 0 to 9 mm Hg. Dobutamine 4 \( \mu \text{g kg}^{-1} \text{min}^{-1} \) was started. At induction of anaesthesia, arterial pressure decreased from 100/50 to 75/45 mm Hg just before intubation and PAP and PCWP increased further. Haemodynamic stability was obtained with epinephrine 3 \( \mu \text{g kg}^{-1} \text{min}^{-1} \) while dobutamine was continued. The patient remained stable with a greater dose of dobutamine (maximum dose 6.7 \( \mu \text{g kg}^{-1} \text{min}^{-1} \)). As CI increased, dobutamine was reduced, but the patient did not tolerate stopping dobutamine infusion. Surgery lasted 290 min and the lowest temperature was 34.4°C. The total dose of sufentanil was 23.7 \( \mu \text{g kg}^{-1} \) and total dose of flunitrazepam was 16.1 \( \mu \text{g kg}^{-1} \).

### Discussion

Cardiomyoplasty is of increasing interest for two reasons: (1) lack of donor organs severely restricts cardiac transplantation in patients with chronic heart failure; (2) it is now recognized that skeletal muscle can be made fatigue-resistant by graded low frequency stimulation over several weeks to allow chronic work like the heart. Fatigue resistance is essential for skeletal muscle to provide support of the circulation. The use of cardiomyoplasty as an alternative to heart transplantation is not clear-cut. Lange and colleagues reserve the treatment for patients with contraindications to heart transplantation. Other authors reported that cardiomyoplasty may be an attractive alternative to heart transplantation and that it does not preclude future orthotopic heart transplantation.

For the first 2 weeks after operation the muscle is not stimulated but allowed to rest. This permits better adherence of the muscle flap to the heart. Preparation of the muscle may cause limited ischaemia, although the muscle retains its vascular supply. Two weeks after surgery, blood flow to the muscle has recovered fully. The muscle is then gradually trained and after 3 months it is finally stimulated with burst impulses in a two-to-one mode, so that the muscle is stimulated with every second heart beat. Schreuder and colleagues tried to improve the synchronization interval, stimulus strength and stimulus duration by beat-to-beat analysis of the left ventricular pressure–volume relation and stroke volume. The improvement in patients with cardiomyoplasty is evident by the upgrading in the NYHA classification, although there are only moderate changes in objective haemodynamic indices. According to Lange and Hagl, cardiomyoplasty improves ventricular performance, limits cardiac dilatation, reduces ventricular wall stress and reverses remodelling of the left ventricular geometry. There is only little systolic improvement of the left ventricle by the latissimus dorsi muscle, but cardiomyoplasty prevents dilatation of the left ventricle and there is a reverse remodelling of the left ventricle so that diastolic function is improved.

The anaesthetic challenge is to manage patients with severely impaired left ventricular function who will not obtain immediate benefit from the operation. Some patients even show worsening of cardiac function after wrapping of the latissimus dorsi muscle around the heart. Early use of inotropic agents is stressed by most authors reporting on cardiomyoplasty. Dobutamine, a beta1 agonist with a low incidence of arrhythmia, is our first choice. Yanagi, Takeuchi and Takeda demonstrated that dobutamine...
improved coronary blood flow and myocardial contractility. Routine use of dobutamine\(^{11}\) and even prophylactic implantation of an intra-aortic balloon pump\(^{10,13}\) are potential techniques for the anaesthetic management of cardiomyoplasty patients. We start dobutamine when CI is less than 2.5 litre min\(^{-1}\) m\(^{-2}\).

Although the population studied was small (\(n=22\)), we identified two groups of patients with a different intraoperative risk during cardiomyoplasty. Twenty of our patients had dilated cardiomyopathy, 17 of these patients needed inotropic support with dobutamine (2.8–11.3 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)) while three patients did not need inotropic support, and CI and \(S\delta\) remained at acceptable levels. Only one patient with a dilated cardiomyopathy died, on the third day after operation as a result of irreversible cardiac failure. Two of our patients had ischaemic cardiomyopathy; in both patients adequate inotropic support could not be achieved with dobutamine and therefore epinephrine was used to achieve CI >2.5 litre min\(^{-1}\) m\(^{-2}\). In one patient, all efforts failed to wean him from CPB because of low output syndrome and ventricular flutter. Although our clinical experience involved only two patients, prophylactic inotropic support with epinephrine or the use of intra-aortic balloon pump should be considered in this subgroup.

Arrhythmia is common during wrapping of the muscle. Different methods are proposed to prevent arrhythmia. Domenegati and colleagues\(^{10}\) spread lidocaine directly onto the pericardium, whereas Haussmann and colleagues\(^{11}\) routinely gave continuous infusion of lidocaine. A total of 55% of our patients did not need anti-arrhythmic therapy and only four patients required continuous infusion of lidocaine. The negative inotropic effects of any anti-arrhythmic drug should be considered in those patients which might further impair left ventricular function.

Double-lumen intubation and single lung ventilation are suggested by Auler and colleagues\(^{14}\) to facilitate thoracic surgery. Single-lumen intubation and ventilation of both lungs appear better because adequate oxygenation is guaranteed during all steps of the operation.\(^{10,11,15}\) Single lung ventilation with a subsequent increase in pulmonary vascular resistance may even be hazardous in these patients.\(^{11}\) Single-lumen intubation caused only a small increase in peak airway pressure after placing the muscle into the left thorax, and adequate ventilation of the patient during wrapping of the muscle around the heart was always achieved. Surgery was not hindered by ventilation of the left lung.

The muscle flap is electrophysiologically tested at the end of its preparation. There must be no neuromuscular block during this test. Some authors reverse the action of a non-depolarizing drug,\(^{16}\) others discontinue continuous infusion and some use only a single dose for intubation.\(^{10}\) We gave a single dose for intubation and a second dose if necessary to change the patient’s position. Neuromuscular monitoring is essential in this situation.\(^{15}\)

In summary, cardiomyoplasty is an encouraging procedure for patients with chronic end-stage heart failure. Whether or not it becomes an alternative to cardiac transplantation\(^{2,5–7,17}\) needs further clinical assessment. Anaesthetic management based on the knowledge of pathophysiology and good cooperation with the surgeon results in low intra- and postoperative mortality. Further clinical studies are necessary to distinguish patients at low risk (e.g. dilated cardiomyopathy) from patients at high risk (e.g. ischaemic cardiomyopathy) of intraoperative complications.

References