Surgical treatment of hypertrophic obstructive cardiomyopathy (HOCM) started in Duesseldorf in 1963 with transaortic subvalvular myectomy (Morrow). After gaining more experience (1980), myectomy was enhanced:

- Subvalvular vertical incisions: near the nadir of the right coronary aortic cusp (RCC); septum beneath the commissure of the RCC and LCC (Bigelow incision)
- Septal extension of myectomy after subvalvular connection of both primary incisions in the direction of the apex of the left ventricle (LV) down toward the base insertion of the anterior papillary muscle
- Myectomy of the hypertrophied lateral LV wall after retraction of the anterior part of the posterior mitral valve chordae.

From 1963 to 2003, 553 patients underwent operations (early mortality rate, 4.5%; n/H11005/26). The number of surgical patients has decreased considerably since 1994 after the introduction by cardiologists of transcoronary ablation of septal hypertrophy (TASH).

Present surgical indications are ineffective TASH, HOCM plus additional congenital, coronary, or valvular cardiac lesions.

Introduction

The experience with surgical treatment of hypertrophic obstructive subaortic muscular hypertrophy in Duesseldorf began on January 10, 1963 when B. Loehr and W. H. Bircks performed a transaortic subvalvular myectomy and a resection of a thickened fibrotic subvalvular layer in a 20-year-old woman (archive number 01-63-0023).

This patient had to be reoperated as the 16th patient of our series 8 years later on January 21, 1971 because of the clinically insufficient relief of left ventricular outflow tract obstruction (LVOTO). In addition to transaortic subvalvular re-myectomy, unusual fibrotic connections between the LV wall and the papillary muscles were removed and an intraventricular obstruction was resected. The postoperative course was complicated by an immediate but not permanent total atriovenous block, a left pneumothorax, and development of lung edema with need for re-intubation (third postoperative day), and attacks of ventricular tachycardias (ninth postoperative day). The patient died with signs of myocardial insufficiency on the ninth postoperative day.

In total, 553 patients with HOCM were operated in our unit from 1963 to 2003 (95% of the operations were performed by two surgeons). The early mortality rate was 4.5% (n = 26).

During the last phase (1990-2003) (Table 1), the early mortality rate was reduced to 3.7% but the number of yearly operations also decreased considerably because of the increased use of and improvements in the TASH procedure used by cardiologists since 1995.

The clinical diagnosis was generally confirmed by patient history and by results of echocardiographic, angiocardio- graphic, and heart catheterization studies.

Our pathophysiological evaluation of the myocardium and mitral valve function was influenced primarily by our own cardiologists Loogen and Kuhn and by Wigle and co-workers (Fig 1). The increased emphasis on morphologic and anatomical features and on surgically practical procedures was related to the studies of van der Wall, who demonstrated morphologically different types of intraventricular obstructions (typical subvalvular, atypical midventricular, and apical), and their pathophysiological consequences (Fig 2).

We maintained stringent indications for surgical treatment in our unit; in general, the patients should belong clinically to class III NYHA.

Only those younger patients who had life-threatening tachyarrhythmias and those patients who were successfully resuscitated were accepted for surgery independent of their clinical classification.

At present, our surgical indications are as follows:

- Symptomatic patients who fail the TASH procedure
- HOCM patients who have in addition a congenital heart defect, coronary heart disease, or a valvular lesion with indication for surgery.

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In our experience, another prospective aspect is the considerably higher operative risk in patients with concurrent cardiac diseases that must be corrected at the same time that TSM is performed.

Our surgical management goals are closely aligned with the goals of the cardiologists (Table 2).

Until 1979 we used the classical transaortic subvalvular myectomy procedure described as first-step myotomy by Morrow and Brockenbrough4 and re-described as the classical myectomy procedure by Morrow.5

In only a few cases of a combined subvalvular and apical type of HOCM was the additional apical left ventricular (fish-mouth-like) incision introduced by Kirklin and Ellis6 performed.

Bigelow and co-workers7 (Fig 3) introduced a subcommisural incision between the right and left coronary aortic cusps (RCC and LCC) and additional enlargement of the myotomy by finger pressing, another important and helpful procedure.

During the late 1970s, our clinical and surgical experiences indicated that the subvalvular area should be surgically widened to a greater extent than it was in the original classical procedure to increase the effect of early relief of LVOTO.1,8,9 Therefore, we introduced stepwise the following additional techniques, described later in detail.

- The primary subvalvular incisions are near the nadir of the right coronary aortic cusp and directly beneath the commissure of the right and left coronary cusps, the original Bigelow incision.7 The third subvalvular incision for myectomy is directed parallel to the left part of the right coronary cusp, connects both primary incisions, and is directed parallel to the ventricular septum toward the apex of the LV under finger control from the surface of the RV.

- In many patients with considerable mitral regurgitation, we observed a bulging hypertrophic muscle at the left lateral wall of the LV under the chordae connected to the posterior mitral leaflet (Fig 5). We envisioned this additional bulging myocardium under systolic contraction

![Table 1 Frequency of HOCM and TSM (1990–2003)]

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<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Surgery with ECC</td>
<td>5859</td>
<td>5411</td>
<td>3604</td>
<td>14874</td>
</tr>
<tr>
<td>HOCM/TSM (n)</td>
<td>187</td>
<td>45</td>
<td>10</td>
<td>242</td>
</tr>
<tr>
<td>(%)</td>
<td>3.2</td>
<td>0.8</td>
<td>0.3</td>
<td>1.6</td>
</tr>
<tr>
<td>Early mortality (n)</td>
<td>6</td>
<td>2</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>(%)</td>
<td>3.2</td>
<td>4.4</td>
<td>10</td>
<td>3.7</td>
</tr>
</tbody>
</table>

![Figure 1] HOCM: Pathophysiological consequences (Wigle and co-workers²).
as a hypomochlion that shortens the chordae and thereby prevents sufficient systolic closure of the mitral valve; this impression was confirmed by TEE sequences. After additional surgical reduction of this part of the hypertrophic muscle, the degree of mitral regurgitation was considerably reduced in nearly all cases.

- The first part of the anterior myectomy is relatively easy to perform, but it is not really possible to view the deeper parts of the residual hypertrophied interventricular septum and to look into the LV cavity (Fig 4).
- Therefore, septal myectomy has to be continued down to the base of the anterior papillary muscle insertion using our specially designed angled instruments (Fig 5). After this procedure, one has a good view into the LV cavity, which allows an exact evaluation of the papillary muscles, the chordae, and their relations to the LV wall and septum.
- In contrast to other authors (Messmer and co-workers\textsuperscript{10,13}), we did not find it necessary to reduce the papillary muscle mass.

![Figure 2](image)

**Figure 2** HOCM: Consequences on morphology and geometry (van der Wall\textsuperscript{3}).

<table>
<thead>
<tr>
<th>Morphology</th>
<th>Geometry</th>
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<tbody>
<tr>
<td>1. Typical HOCM</td>
<td>1. Angulation of LV long axis</td>
</tr>
<tr>
<td>2. Atypical HOCM (midventricular)</td>
<td>2. Torsion of LV–cavity</td>
</tr>
<tr>
<td>3. Atypical HOCM (apical)</td>
<td>3. Spatial rotation</td>
</tr>
<tr>
<td>(E. van der Wall, 1985)</td>
<td>4. Preferential flow towards</td>
</tr>
<tr>
<td></td>
<td>mitral orifice</td>
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**Table 2** HOCM: Surgical Management Goals

<table>
<thead>
<tr>
<th>Effective enlargement of LVOT</th>
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<tr>
<td>Excision of thickened fibrotic</td>
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<tr>
<td>endocardium</td>
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<tr>
<td>Correction of long-axis</td>
</tr>
<tr>
<td>deviation between LV and</td>
</tr>
<tr>
<td>ascending aorta</td>
</tr>
<tr>
<td>Reduction of degree of mitral</td>
</tr>
<tr>
<td>regurgitation</td>
</tr>
<tr>
<td>Avoidance of disease related</td>
</tr>
<tr>
<td>secondary complications: AF,</td>
</tr>
<tr>
<td>VF, VT, syncope, sudden death,</td>
</tr>
<tr>
<td>arterial embolism, myocardial</td>
</tr>
<tr>
<td>failure</td>
</tr>
<tr>
<td>Intra-, post-operative: laceration of mitral chordae, aortic cusps, total av-block, secondary VSD</td>
</tr>
<tr>
<td>Positive influence on early</td>
</tr>
<tr>
<td>outcome and on long-term</td>
</tr>
<tr>
<td>prognosis (?).</td>
</tr>
</tbody>
</table>

- In contrast to other authors (Messmer and co-workers\textsuperscript{10,13}), we did not find it necessary to reduce the papillary muscle mass.
Figure 3  Bigelow—incision and extension of subvalvular myectomy [modified after Morrow4,5 and Bigelow7 from nadir of RCC to commissure of RCC and LCC (From Herz-, Thorax-, Gefaesschir. 2151, 1988)].

Figure 4  Schematic survey of intraventricular extension of subvalvular, midventricular, and lateral LV wall hypertrophy (modified from AE Becker, RH Anderson: Cardiac Pathol 198312). (Color version of figure is available online at http://www.us.elsevierhealth.com/optechstcvs.)
Surgical Procedure

A step-by-step overview of our detailed surgical procedure follows:

- For the surgeon a headlight is an obligation; magnifying glasses are also helpful.
- Median longitudinal sternotomy and longitudinal pericardiotomy are performed as usual.
- We prefer direct ascending aorta arterial and bivaval venous cannulation via the right atrial appendage and the lower part of the RA.
- After cannulation and completion of ECC connections, intraoperative pressure measurements are made with a normally beating heart and after provocation (ventricular extrasystolic beat). Intraoperative pressure gradients between the LV and the ascending aorta (Fig 6) and between LV and LA are recorded. An assessment is made of mitral valve function (degree of mitral regurgitation).
- For special requirements and questions, intraoperative TEE can be introduced for evaluation of degree, localization, and extension of the stenotic areas, confirmation of systolic anterior movement of the anterior mitral leaflet (SAM), and determination of the individual degree of mitral regurgitation.
- ECC is initiated with perfusion hypothermia (5 minute with blood temperature of 30-32°C).
- In all HOCM patients, we always insert a LV drainage tube via the right upper lung vein, left atrium (LA), and through the mitral valve orifice for control or measurement of the LV pressure at different intraventricular positions and for myocardial relief during early reperfusion.
- After cross-clamping the ascending aorta, aortic root infusion of 2 to 4 liters of 4 to 6°C cold Bretschneider cardioplegic solution is started under volume and pressure control for 8 minutes to cool down the complete hypertrophied myocardium efficiently.
- Then an oblique incision of the ascending aorta deep into the middle of the noncoronary sinus is performed that allows an excellent view of the aortic valve and into the LVOT (Fig 7 A,B).
- The operating table is turned slightly to the left side, and the ventricular septum is cautiously pushed in the direction of the LVOT by an assistant.
- The first steps of the myectomy procedure (Fig 8 A–D) include the safe retraction of the right coronary aortic cusp, the aortic wall, and the mitral chordae using specially designed angled instruments and a thin flat metallic flexible spatula (see Figs 5 and 8C); the goal is to improve the visual shaft into the LVOT, to protect the mitral chordae, and to localize correctly the anatomy for the necessary primary three incisions into the hypertrophied subvalvular septum.
- After the angled instruments are introduced, the myotomies near the nadir of the RCC and immediately beneath the commissure between the RCC and LCC (see Figs 8 A,B) are precisely performed in the direction of the apex of the LV. With the connecting incision parallel and about 1.5 mm below the RCC, an extended myectomy (see Figs 8C, D) is started and continued by additionally fixing the excised part of the hypertrophied septum with a broad-headed forceps. After this first step, the angled instruments have to be newly positioned to exponate the middle and lower parts of the septum with continuing myocardial excision at least down to the insertion of the anterior papillary muscle (see Fig 4).
- After effective myectomy and insertion of the special instru-
ments, the LV cavity can be opened sufficiently for closer inspection of the papillary muscles, chordae, and mitral leaflets. After retraction of the chordae, surgical reduction of submitral hypertrophic muscle is performed that allows better systolic occlusion of the mitral valve (see Fig 4).

- Some specimens exhibit the subvalvular hypertrophic myocardium and the thickened fibrotic endocardial layer, whereas this fibrotic endocardium is not detectable on additionally resected hypertrophied intraventricular myocardium.

- After effective myectomy, the LV cavity can be opened widely enough for closer inspection of the papillary muscles and of the aortic part of the mitral valve and the adjacent chordae by using the angled instruments and finally resulting in a fairly enlarged subvalvular LVOT (Fig 9).

- Rewarming of the patient begins as these steps are performed.

- Before rinsing the LV cavity with Ringer’s solution twice to remove all possible mobile tissue pieces, the cutting rims are corrected and a complete resection of the residual fibrotic intimal layer of the LVOT is performed.

- Final finger control of the created subvalvular septal channel allows a preliminary estimation of surgical effectiveness.

- One must also inspect the aortic cusps and their occlusiveness (Fig 10). The aortic incision is closed from both ends with continuing sutures. Before final knotting, an efficient deairing of the aortic root is necessary after reduction of the perfusion pressure, before and after release of the aortic clamp, and without any suction on the LV vent.

- Additional deairing of the LV cavity and LA is performed after lifting the apex, slightly compressing the heart, inflating the lungs, and performing needle puncture through the apex.

- If necessary, rewarming reperfusion defibrillation is initiated after 3 minutes. Reperfusion is continued until a blood temperature of at least 34°C is reached.

- In all patients, bipolar right atrial and right ventricular pacemaker leads are applied to the myocardium.

- The patient is weaned off bypass under the control of continuous LA pressure monitoring.

- Postoperative intracavitary pressures (LA-LV, ascending aorta-LV) in a normally beating heart are measured after circulatory normalization and after provocation (postextrasystolic beat) (see Fig 6).

- Decannulation of the RA and ascending aorta, insertion of an intrapericardial and retrosternal drainage tube, incomplete closure of the pericardium, and stepwise closure of the chest conclude the surgical procedure.

- The morphologic, pathophysiologic, and hemodynamic consequences of extended effective myectomy are summarized in Fig 11.

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**Figure 6** Preoperative pressure tracings with a normal beating heart (left) and after a ventricular extrasystolic beat (right) demonstrating pathological high gradients (upper graphs a1, a2) postoperatively after myectomy with considerably reduced gradients (lower graphs b1, b2). (From: Progress in Cardiology 2/2, 192, 1989.)
Figure 7  View into the LVOT. (A) Narrowed LVOT with bulging subvalvular hypertrophied septum. (B) Bulging of the mitral chordae crossing a hypertrophied part of the left LV wall. (Color version of figure is available online at http://www.us.elsevierhealth.com/optechstcvs.)
Figure 8  Transaortic subvalvular incisions preparing myectomy. (A) Schematic extension of subvalvular first incision near the nadir of RCC in direction to LV apex. (B) Second incision below the commissure between RCC and LCC parallel to the LCC in direction to the LVOT (modified combined Morrow-Bigelow incisions). (C) Intraoperative view: First incision and second incision (dotted line). (D) Performed first and second incisions, the flexible spatulum retains safely the chordae of the anterior mitral leaflet in the lower LVOT. (E) Third incision parallel to the left part of the RCC leaving a rim of about 1 to 1.5 mm of subvalvular myocardium. The partly excised myocardium is fixed with a broad-headed forceps to ameliorate the myectomy in direction to the apex and the residual LV cavity. (F) Extent of the primary part of TSM which has to be further continued downwards into the LV. (Color version of figure is available online at http://www.us elsevierhealth.com/optechstcvs.)
Figure 9  View into the effectively enlarged LVOT after retraction of the aortic cusps. (Color version of figure is available online at http://www.us ELSEVIERHEALTH.COM/OPTECHSTCVS.)
Figure 10 Control of the aortic cusps after the instrumental and surgical manipulations before closure of the oblique aortic incision. (Color version of figure is available online at http://www.us.elsevierhealth.com/optechstcvs.)

Figure 11 HOCM: Pathophysiological consequences after considerable surgical relief of the LVOTO and of mitral regurgitation.
Conclusion

Over a 40-year period, we gained considerable experience with the medical and surgical treatment and postoperative long-term management of more than 500 patients with HOCM. We learned that surgical relief can be obtained using the classical technique of transaortic subvalvular septal myectomy of Morrow but that the technique should be supplemented. Therefore, we further enhanced this technique over the years to extend it as far as anatomically feasible. The morphologic, pathophysiologic, and hemodynamic consequences of effective myectomy result in improvement in the immediate postoperative and long-term behavior of the patients.

The early and long-term results of extended myectomy demonstrate that a well-prepared, excellently organized surgical procedure, which, although it has a positive effect on symptoms does not heal the original underlying congenital myocardial disease, can enhance the daily life experiences and life expectancy of the individual patient.

The risk of morbidity and mortality remains, especially in patients with additional cardiac lesions to be corrected simultaneously.

References