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Abstract Objective. Recently there has been an emphasis on reconstructing diseases native valves as an alternative to prosthetic valve replacement. Whereas; the surgical repair of aortic valve stenosis has been always problematic. This study was performed to estimate the clinical results after aortic valve debridement using ultrasonic energy.

Methods. Between 1990–1994 26 patients underwent ultrasonic aortic valve decalcification. There were 15 females and 11 males, the age was in average 74 years. As a concomitant diagnosis 88% patients (23) had mostly mild, aortic valve insufficiency, 16 (61%) had coronary artery disease and 11 (42%) had mild mitral valve incompetence. All of the patients were operated with cardiopulmonary bypass using moderate hypothermia, cardioplegical arrest and topical cooling for myocardial protection. The calcifications were removed tangentially using Cavitron Ultraasonic Surgical Aspirator (CUSA), CAVITRON, USA. Leaflet perforation and/or unsatisfactory valve closure have been indications for aortic valve replacement.

Results. Two operative death (8%) have occurred and six patients have died in the further course (follow-up mean 17 months ranging from 4 to 61 months). Postoperative Doppler-Echocardiography results taken directly after surgery and then again 17 months later (n = 18) showed a decrease of peak and mean gradients across the aortic valve three and two times respectively (p<0.001). 17 months after debridement we observed a mild rise in both gradients (by peak gradient p<0.05). Directly postoperative, the aortic valve area increased doubly and decreased 17 months later slightly, but it was still statistically significant in comparison with our preoperative data (p<0.001). Follow-up echocardiography demonstrated late onset of moderate aortic valve insufficiency in 6 patients. The classification of New York Heart Association was improved in 13 (72%) survivors after 17 months.

Conclusions. Ultrasonic debridement of aortic valve stenosis allows precise and energy-controlled removal of calcium, increased doubly the valve area and decreased of peak and mean gradients statistically significant. The advantages of preserving the native aortic valve in elderly patients are relative good arguments; although a longer follow-up is necessary to establish this procedure.

Key words Aortic stenosis · Aortic valve debridement · Surgery · Reconstructing · Follow-up
Introduction

Recently there has been an emphasis on reconstructing diseased native valves as an alternative to prosthetic valve replacement. Whereas the surgical techniques have proved to the straightforward in mitral and tricuspid valve reconstruction, the repair of aortic valves has always been problematic [15, 23]. Mechanical debridement of the aortic valve was used as a treatment to relieve aortic stenosis of a different etiology many years before valves substitutes were available. The recurrences of valve stenosis and development of valve regurgitation as well as the availability of valve prostheses led to the abandonment of this technique [12]. Despite the improved results, complications associated with valve replacement have led to more interest in aortic valvuloplasty in selected patients [13, 15].

By applying new technology, it has been demonstrated during the past several years that stenotic aortic valves can be successfully decalcified by means of ultrasonic energy. Aortic valve debridement, using an ultrasonic surgical aspirator, has been reported to be an effective alternative to valve replacement, especially in cases of degenerative, aortic stenosis – Mönckeberg type – mostly in elderly patients [1, 6, 7, 8].

Altogether there are three basic types of aortic valve stenosis: 1) the endocarditis lenta type, 2) rheumatic aortic valve stenosis and 3) degenerative aortic stenosis. In the third type, which can be seen mostly in elderly patients, a tricuspid valve, without commissural fusion, is present (type Mönckeberg, 1904) [10]. The leaflets are generally slightly thickened and the coarse calcific particles and atheromatous deposits fill the aortic side of the belly of the cusps (Fig. 1a). The histological findings show fibrosis and calcification of the valvular annulus and of the proximal parts of the cusps as well. Quite often the calcifications extend to the commissures (Fig. 1b, c). The Mönckeberg aortic stenosis is frequently associated with coronary artery disease. Balloon – valvuloplasty would be unsuccessful, since the valve area could not become larger due to the calcified commissural parts of the cusps. The massive calcifications in the sinus of the cusps would hinder the mobility of the valve as well. Decalcification of this type of stenosis would be favorable because the calcifications are not caused by any inflammatory process. The purpose of the operation is to enlarge the valve area by increasing the mobility of the cusps.

Patients and methods

From January 1990 to September 1994, 26 (3.1%) patients (15 females and 11 males) of a group undergoing 833 aortic valve procedures were treated with ultrasonic aortic valve decalcification at the Augsburg Medical Center. The age ranged from 59 to 81 (mean 74) years. Diagnosed lesions were aortic stenosis and mild insufficiency in 23 patients and aortic stenosis only in 3 patients. As a concomi-

Fig. 1 Atheromatous deposits on the aortic side of the belly of the valve cusps. Calcifications of the valvular annulus and of the proximal parts of the valve cusps (Gravanis 1987)
Fig. 2 Degenerative, calcified aortic valve. Debridement by ultrasonic technique. Leaflets after completion of debridement process.

Tent diagnosis, 16 (61%) patients had coronary artery disease (1 with single vessel disease and 15 with double vessel disease) and 11 (42%) had mitral valve incompetence (1 severe, the others mild). In nine patients hypercholesterinemia was diagnosed and in 6 diabetes mellitus. Preoperative peak systolic gradients across the aortic valve ranged from 22 to 120 mmHg (mean 76.87±31.88), the mean gradient was 51.37±18.01 mmHg. The aortic valve area ranged from 1.2 to 0.3 cm² (mean 0.74±0.31 cm²). The mean ejection fraction (EF) was 59.04%±16.78% and the mean left ventricular end-diastolic pressure was 18.75±9.25 mmHg.

In 26 patients the cause of the disease, based on anamnesis and visual examinations at the time of operation, was degenerative aortic stenosis. Three patients were excluded from this study since the valve debridement was unsuccessful and aortic valve replacement had to be performed. The time of aortic cross-clamping using Bretschneider cardioplegia was 61 min in the whole group, and 81 min in the patients with additional coronary artery bypass grafting (CABG). In the last 15 patients, the aortic valve debridement was performed within less than 30 min.

Postoperatively, follow-up assessment was obtained by clinical examination and Doppler-echocardiography. Echocardiographic measurements were made with Acuson Mod. 128 XD¹ using colour Doppler imaging (CDI), pulsed-wave (pw) and continuous-wave (cw) mode. Scanning was carried out with a 2.5/3.5 MHz transducer (Acuson V 319 vector scan)¹. The multiple comparisons between mean values were made at different time intervals by the Mann-Whitney test.

All patients were operated on with cardiopulmonary bypass and underwent moderate hypothermia 28–30°C. A pulmonary artery vent was inserted. The myocardial protection was achieved through Bretschneider cardioplegia and topical cooling with 4 °C cold saline solution. After cross-clamping of the aorta, the valve was first carefully inspected to determine the type of the stenosis. If the valve showed typical degenerative changes, a Follies catheter was inserted across the valve into the left ventricle and inflated just behind the valve. On the one hand, the catheter protected the ventricle from possible particles and on the other it was possible to drain the left ventricle and expose the valve. The left coronary ostium was protected using continuous suction. The calcifications were then removed tangentially using a Cavitron Ultrasonic Surgical Aspirator (CUSA)² (Fig. 2). This device works specifically for tissue as it protects collagen and elastic structures. Due to electric energy, an ultrasonic frequency of 23000 cycles/sec is performed, which activates a magnetostructure transducer in the hand piece. The longitudinal contractions of this acoustic vibrator lead, above the instrument, to a fragmentation which is proportional to the water content of the tissue. Adjustment of the vibration amplitude gives more or less deep penetration of the tissue. This procedure is therefore suitable for the resolution of calcifications. Simultaneous irrigation with saline and aspiration of the cell fragments is possible as well.

Our previous experiments have shown that the valves are easily perforated, since the top of CUSA works perpendicularly to the valve area. It is therefore important to use it at an angle. To reach deeper layers, the amplitude was reduced to 10–20%; thus, a better tissue differentiation was possible. The looser calcifications can be removed easily now by using another increase of the amplitude. The irrigation was adjusted to 3 cm³/min. Proper cooling of the system and a fairly good suspension of the fragments was achieved. The suction on top of the CUSA was adjusted to 10–15 mmHg. The cusps of the valve were supported by using a bent teaspoon which was introduced through the commissures of the valve.

After decalcification and careful irrigation of the left ventricle and the aorta, the Follies catheter was removed. Leaflet perforation and/or unsatisfactory valve closure would have been indications for aortic valve replacement. Out of 26 patients in our series, 16 underwent additional procedures at the time of decalcification (16 had CABG (1–5)), 1 patient also had mitral valve decalcification using the CUSA technique, and 1 patient underwent mitral valve replacement.

¹ Acuson, Erlangen, Germany
² Cavitron, Stamford, USA
**Results**

**Early results**

Two operative deaths (8%) occurred. Both patients were operated electively, had additional coronary artery disease, and were in NYHA class II or III preoperatively. Mild aortic valve incompetence and mitral ring calcifications were seen in both angiograms. Aortic valve debridement and additional CABG were performed successfully. The first patient died 1 day after operation due to cardiac failure. The pharmacological and mechanical support (IABP) were unsuccessful. The second patient had respiratory insufficiency and could not be weaned from the ventilator. He died due to *Pseudomonas* sepsis on the 14th postoperative day. Another patient required prolonged ventilatory support for 8 days. Prolonged neurological recovery without stroke symptoms was observed in four patients. Nineteen patients had no early postoperative complications.

**Late results**

There were six deaths during the follow-up period. During the operation, four of the patients underwent CABG addi-

![Graph 3](image)

Fig. 3 Mean values for peak and mean aortic gradients preoperatively, immediately postoperatively and after 17 months (Peak gradients, mean gradients)

![Graph 4](image)

Fig. 4 Mean values for aortic valve area preoperatively, immediately postoperatively and after 17 months (Aortic valve area)

Echocardiographic results

Echocardiography was used to compare valvular parameters directly postoperatively and, in the later course, on an average 17 months postoperatively (n=18) (range 4–61 months). After aortic valve debridement, the peak and mean gradients across the aortic valve were reduced by about two-thirds and half, respectively (P<0.001) (Fig. 3). Comparison of the immediately postoperative values with those 17 months later revealed a rise in both gradients. The mean gradient’s increase was not significant, whereas the peak gradient’s change was slightly significant (P<0.05), which suggests that a mild degree of restenosis may have occurred during this period.

In the early postoperative course, the aortic valve area (Fig. 4) was doubled. Seventeen months later a slight decrease of this value was observed, however it was still statistically significant in comparison with the preoperative data (P<0.001).

Valvular regurgitation

In six patients, aortic valve insufficiency had increased significantly on echocardiography 17 months after aortic valve decalcification. Two patients with only aortic stenosis preoperatively developed, respectively, mild and moderate valve incompetence (AI) while four patients with preoperative mild AI developed moderate AI (Fig. 5). No relief of aortic valve regurgitation has been observed.

Functional classification

Late follow-up information obtained from 18 surviving patients on examination 4–61 months (mean 17 months) after operation, showed that the functional NYHA classification had improved in 13 (72%) survivors. Four patients...
Borkon and associates [3] reported, for patients 70 years of age and older with mechanical failure, valve tissue degeneration, thrombosis, and anticoagulation [18]. This is especially true in the elderly population. Logeais and associates [14] reported a surgical mortality of 12.4% from 632 older patients after aortic valve replacement, 5-year valve-related mortality of 10.7% and 17.6% for biological and mechanical prostheses, respectively.

Later on many surgeons attempted opening the valve with the use of several valvulotomes. They reported favorable results, but operative mortality and late complications were relatively high [2, 11]. With the introduction of cardiopulmonary bypass, several investigators performed mechanical debridement of calcific deposits in the annulus and leaflets of the aortic valve leading to improved mobility of the valve cusps and relief of stenosis. Although the early results were optimistic, late follow-up showed high rates of restenosis and valvular insufficiency [3].

The last 35 years of experience with prosthetic aortic valve replacement as a standard operative treatment for aortic stenosis have shown persistent morbidity associated with mechanical failure, valve tissue degeneration, thromboembolism events, and complications due to infections and anticoagulation [18]. This is especially true in the elderly population. Logeais and associates [14] reported a surgical mortality of 12.4% from 632 older patients after aortic valve replacement using bioprostheses. Borkon and associates [3] reported, for patients 70 years of age and older after aortic valve replacement, 5-year valve-related morbidity of 10.7% and 17.6% for biological and mechanical prostheses, respectively.

In some patients with aortic stenosis, percutaneous balloon valvuloplasty can be used to open the commissures. By the degenerative, aortic stenosis in all our patients this method would be unsuccessful, because the commissures were open and only the cusps and valve annuli were calcified. A renewed interest in preservation of the native valves was stimulated by Carpentier and associates [5] in mitral valve operations and, from 1972, by Brown and Davies [4] who introduced the ultrasonic decalcification of cardiac valves.

There are practical advantages of repair compared with valve replacement. Because the native valve is preserved, no foreign material is implanted and the risk of infection is reduced. In addition, these patients do not need long-term anticoagulation and the absence of a prosthetic ring means that there is no obstruction to the aortic outflow tract, especially in small aortic roots [13, 16]. Ultrasonic debridement of the aortic valve, in comparison to mechanical debridement, allows more precise and energy-controlled removal of the calcium. Leaflet perforations still occur, mostly due to incorrect use of the instrument, i.e. in inaccurate direction of application and too high amplitudes.

Cosgrove and associates [6] compared the pathological findings of resected aortic valves in patients who required aortic valve replacement for aortic insufficiency after ultrasonic debridement. In those valves, the elastic fibers were replaced by fibrous tissue and the fibrosa and ventricular layers were not present. These observations suggested that those changes may be explained by the use of high ultrasonic power and the aggressiveness of the debridement. According to our technique, to reach deeper layers of calcium the amplitude was reduced to 10–20%, whereby better tissue differentiation was achieved. In cases of leaflet perforation, valve replacement was performed in three patients, who were not included in this series. Other surgeons repaired the perforations directly using mattress sutures or with pericardial patch. McBride and associates [15] reported that only one of the five patients who had a leaflet perforation is still alive and free from valvular insufficiency at the 6th month. Baeza and associates [1] had only one patient with cusp perforation repaired with a pericardial patch who had no valvular incompetence within 3 years after surgery. Because the natural course of leaflet perforation is unknown, we suggest valve replacement to be the safer procedure in these cases. The early operative mortality in our group was 8% (in other series after ultrasonic valve decalcification, the mortality rates ranged from 6 to 13% [1, 13, 15], but they included aortic stenosis of different etiologies). The percentage of degenerative, aortic stenosis ranged from 10 to 45% in the other series. King and associates [13] reported that of 8 patients with degenerative, aortic stenosis decalcification 100% were alive 1 year, and 50% 5 years, after operation. In our group, after 17 months (range 4–61 months) 69% (18) debrided patients are still alive.

Continuous-wave Doppler echocardiography is commonly used to assess valvular flow characteristics. Recent studies have demonstrated an accuracy in determining valvular areas and pressure gradients. Our echocardiographic analysis showed that the valvular debridement with CUSA was very effective directly after surgery due to the increase in the valve area,
of valve area and decrease of peak and mean gradients ($P<0.001$). Most authors had similar early results after ultrasonic decalcification in their series [9, 15, 18, 19]. A further follow-up of 17 months showed increases of the peak gradient across the aortic valve ($P<0.05$). However, the mean gradient was practically unchanged at the same time. The valve area was slightly decreased but there was statistically no difference. No patients in our series had evidence of significant restenosis requiring reoperation. Similar results were published by McBride and associates [15], who assessed 22 patients with a mean follow-up of 6 months after decalcification. Baeza and associates [1] observed no significant stenosis or insufficiency in the 31 patients studied 3 years after debridement. Recent studies with longer follow-up have revealed that aortic insufficiency developed after aortic valve debridement due to fibrosis and retraction of the valve’s center with loss of coaptation [6, 17]. Craver and associates [7] observed an increase of valve regurgitation requiring reoperation in three of eight patients 1 year after aortic valve decalcification.

In our series, six patients had increases in aortic regurgitation, five of them developed moderate, and one mild, aortic insufficiency (AI) at an average of 17 months after decalcification. When evaluating the late results in detail, we have to include four patients who died due to cardiac failure and who have not been autopsied. In some of these the cause of death could be related to progression of the aortic valve disease. In our study, we observed functional improvement in the further follow-up (17 months). In 72% ($n = 13$), the NYHA classification improved in comparison to the preoperative classification. Only in one patient was a deterioration of his functional status from NYHA II to NYHA III observed. McBridge and associates [15] did not observe significant improvement in NYHA classification in their series. Baeza and Associates [1] observed significant improvement in 22 of 31 patients operated on at 12 and 24 months after aortic valve debridement.

Weinstein and associates [22] outlined the indications for aortic valve decalcification: patients with degenerative calcification (100% in our series), those more than 75 years old, those with calcification of the aorta or the annulus, those with small aortic annuli, those with severe coronary artery disease, those with poor left ventricular function (ejection fraction <25%), and those with contraindications for anticoagulant therapy. Like most the authors, we agree with these indications. We have also accepted, in our patients, preoperative mild valve incompetence which can be caused through calcium particles on the ventricular side, or on the border, of the leaflets.

This procedure seems to be ideal for the repair of asymptomatic aortic valve disease in patients undergoing cardiac surgery for other indications [21]. We think that a better understanding of the effects of ultrasonics on the tissues, the use of CUSA at low amplitudes for the removal of deep calcium deposits in and the direction of the hand piece during debridement are important factors in decreasing the development of significant valve incompetence and improving late results. The advantages of preserving the native aortic valve in elderly patients are relatively good arguments for ultrasonic debridement in patients with degenerative, aortic valve stenosis; although a longer follow-up is necessary to evaluate this procedure.

References


Forthcoming meetings and events

1996

June 18–20, 1996 – Cernobbio (CO), Italy
The VII International Meeting 1996 of the Society for Minimally Invasive Therapy – SMIT, Villa Erba, Cernobbio (Como), Italy Information: Emilia Viaggi, The Organizing Secretariat, Congress Meeting Incentive, Piazza Malpighi 3/A, I-40121 Bologna, Italy, Tel. 00 39 51 23 58 92, Telefax 00 39 51 22 12 28

October 3–4, 1996 – Bristol, UK
International Workshop: Recent Developments in Cardiac Surgery (video assisted demonstrations on left ventricular reduction and minimally invasive coronary surgery), Hilton National Hotel, Bristol, UK Information: Mrs. N. J. Merrell, Bristol Heart Institute, University of Bristol, Bristol BS2 8HW, UK, Tel. +44 11 79 28 31 45, Fax. No.: +44 11 79 29 97 37

October 14–15, 1996 – London, UK
Surgical Workshops using Anatomical Prosection: Aortic Root Surgery – Homograft and Autograft replacement. Information: Rachel Kerr, The Royal College of Surgeons of England, Tel. 00 44 17 19 73 21 07, Email: rkerr@rcseng.ac.uk

October 14–16, 1996 – London, UK
European School of Oncology Advanced Course on Chest Tumours, Royal Brompton Hospital, London, UK Information: C Ratcliffe, Thoracic Surgery, Royal Brompton Hospital, Sydney St., London SW3 6NP, UK, Tel. +44 17 13 51 85 67, Fax. No.: +44 17 13 51 85 55

October 20–25, 1996 – Tel Aviv, Israel
9th Annual Meeting of the Mediterranean Association of Cardiology & Cardiac Surgery P.O. Box 50006, Tel Aviv 61500, Israel Information: Prof. B. A. Vidner, President, Local Organizing Committee, Tel Aviv, Israel, Tel. +9 72 35 14 00 14, Fax. No.: +9 72 35 17 56 74/5 14 00 77

October 25, 1996 – Zeist, The Netherlands
One-day Symposium on "Surgery of the Proximal Thoracic Aorta and the Carotid Arteries. Current Concepts and Controversies", Golden Tulip Hotel FIGI, Zeist, The Netherlands Information: Congress Office Tonne verdonck, P.O. Box 113, NL-5660 AC Gelredonc, The Netherlands, Tel. +31 41 02 85 22 12, Fax. No.: +31 41 02 85 19 66

International Workshop on Infrathoracic Staging, Royal Brompton Hospital, London, UK Information: C. Ratcliffe, Thoracic Surgery, Royal Brompton Hospital, Sydney St., London SW3 6NP, Tel. +44 17 13 51 85 67, Fax. No.: +44 17 13 51 85 55

1997

February 13–15, 1997 – San Diego, California, USA
Pathophysiology & techniques of Cardiopulmonary bypass: The 17th Annual San Diego Cardiothoracic Surgery Symposium, San Diego, CA, USA Information: C. R. E. F., P.O. Box 3220, San Diego, CA 92193, USA, Fax. No.: 001 61 95 41 14 47, Email: 742241, 1523@compuserve.com

October 12–17, 1997 – Sydney, Australia
13th Biennial Asian Congress on Thoracic and Cardiovascular Surgery, Sydney Convention and Exhibition Centre, Sydney, Australia Information: ACTCV Congress Secretariat, Conference Action Pry Ltd, P.O. Box 1231, North Sydney NSW 2059, Australia, Tel. +6 12 99 56 83 33, Fax. No.: +6 12 99 56 51 54

1998

June 30–July 4, 1998 – Frankfurt/Main, Germany
10. European Congress of Anaesthesiology Organizer: Deutsche Gesellschaft für Anaesthesiologie und Intensivmedizin Information: Prof. Dr. G. Hempelmann, President, Department of Anaesthesiology and Intensive Care Medicine, Justus-Liebig-University, Klinikstrasse 29, D-35385 Giessen, Germany, Tel. ++49 (0) 6 41/7 02 35 05, Fax: ++49 (0) 6 41/7 02 32 66