Effect of Medical and Surgical Therapy on Aortic Dissection Evaluated by Transesophageal Echocardiography

Implications for Prognosis and Therapy

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Background. Aortic dissection still has a poor prognosis despite progress in therapy. Therefore, this prospective follow-up study was designed to determine whether the degree of communication between true and false lumen in relation to the type of dissection, analyzed by transesophageal echocardiography, influences the risk after initiation of medical or surgical therapy.

Methods and Results. In eight centers, 168 patients (124 men and 44 women) of age range of 23–84 years with proven aortic dissection were examined by transesophageal echocardiography in the acute phase, after start of medical and/or surgical therapy, and during follow-up (0–65 months; mean, 10 months). Analyses were performed prospectively according to a detailed study protocol. Patients were subdivided by transesophageal echocardiography according to a modified DeBakey classification. Type I aortic dissection was found in 35%, type II aortic dissection in 17%, and type III aortic dissection in 48%. Preoperative mortality was 3%, 7%, and 2%, and survival rates were 52%, 69%, and 70%, respectively. Type III aortic dissection could be subdivided into those with communication and antegrade dissection (ca) (50%), with communication and retrograde dissection limited to the descending aorta (cr desc) (10%), with dissection extended to the aortic arch and ascending aorta (cr asc) (27%), and with noncommunicating (nc) aortic dissection (13%). An open false lumen with no thrombus formation was present in types I, II, III ca and III cr asc aortic dissection in 17%, 21%, 39%, and 27% respectively, although it was most pronounced in types III nc and III cr desc (75% and 78%). During follow-up in patients who survived, thrombus was demonstrated in the false lumen in 80% of type I aortic dissection and 81% of types III ca and III cr asc. Open false lumen was seen in type II aortic dissection in 18%. Spontaneous healing was found in 4% with type II and 4% with type III aortic dissection (mainly in patients with type III nc aortic dissection). Patients with fluid extravasation, pleural effusion, pericardial tamponade, and periaortic effusion as well as mediastinal hematoma had a mortality of 52%. Reoperations were necessary in 12–29%, with the highest rate in patients with type III ca aortic dissection. Survival for patients with types III nc and III cr desc aortic dissection was higher than those with types I, II, III ca, and III cr asc.

Conclusions. Preoperative mortality appears to be reduced by transesophageal echocardiography, allowing rapid initiation of treatment. Intraoperative and postoperative mortality in aortic dissection remains high. Risk factors are fluid extravasation and an open false lumen with high communication. Thrombus formation in the false lumen can be regarded as a good prognostic sign. Surgery appears to be only a first step in the treatment of aortic dissection. Second surgery or closure of entry sites based on intraoperative echocardiography may be considered to induce thrombus formation and reduce aortic wall stress. (Circulation 1993;87:1604–1615)

KEY WORDS • aortic dissection • echocardiography, transesophageal • imaging • mortality • surgery

Aortic dissection has a significant in-hospital and follow-up mortality.1-6 Improved hospital and long-term survival rates have been reported,2,6 but they could not be confirmed by others.1,5,7,8 Nevertheless, the 3-year mortality remains 21% for proximal and 29% for distal aortic dissections.6 Reoperations are necessary in 7–20% of patients1,5,6 but appear not to be

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Received August 21, 1992; revision accepted January 22, 1993.

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related to the type of operation, to whether the primary intimal tear was resected or the aortic valve was replaced, to the type and acuity of the dissection, to the extent of aortic involvement, to the presence of Marfan's syndrome, or to histological results. Younger age, site of the intimal tear (arch and descending aorta), and tamponade do appear to increase the reoperation rate. The sensitivity and specificity of transesophageal echocardiography for the diagnosis of aortic dissection are 99% and 88%, respectively, compared with 83% and 100% for computed tomography and 88% and 94% for angiography. Transesophageal echocardiography allows not only visualization of the intimal flaps and intimal tears but also differentiation of true and false lumens and detection of complications. Transesophageal echocardiography appears to be ideally suited for follow-up studies because it makes it possible to detect and locate intimal tears, which seem to be the major determinants of dissection healing and cause of death.

See p 1765

The purpose of this prospective study was to analyze the prognosis of patients with aortic dissection after initiation of medical or surgical therapy based on follow-up studies using transesophageal echocardiography.

Methods

In eight European centers from 1986 to 1991, 168 consecutive patients with proven aortic dissections were prospectively examined by transesophageal echocardiography in the acute phase. Those who survived were reevaluated after the initiation of medical and/or surgical therapy and during follow-up. Each center compiled with a detailed study protocol and provided the necessary data for this prospective analysis.

A diagnosis of aortic dissection was confirmed by transesophageal echocardiography and was correlated with surgical and/or autopsy findings. In the case of medical therapy, confirmation of the diagnosis by one additional diagnostic method (computed tomography, magnetic resonance imaging, or angiography) was necessary. Dissection was defined as acute when the diagnosis was made within 14 days of the onset of symptoms.

Echocardiography

M-mode and cross-sectional echocardiograms were recorded in standard planes using parasternal, apical, and suprasternal transducer positions. Transesophageal Echocardiography

For transesophageal echocardiography, 3.5 MHz and biplane 5 MHz (Sonographer SSH 830, SSH 870, Aloka, Japan) and 5 MHz (SSH 160 Toshiba; HP 1000 Hewlett Packard; Vingmed CFM 700 Sonotron, Germany) were used. The echoscopes used ranged in diameter from 9 to 12 mm. In addition to two-dimensional echocardiography, color Doppler flow imaging was used and superimposed and imaged as previously described. Images were stored on video tape and hard copies. Together with patient data, they were collected at the coordinating center in Mainz.

Transesophageal echocardiography was performed in the fasting state (4–5 hours) after excluding the possibility of an esophageal disorder, except in emergency situations when examinations were performed without fasting. Premedication was used, as previously reported. Blood pressure was monitored, and antihypertensive therapy (mainly β-blocking agents and vasodilators) was started for patients with suspected aortic dissection. Physicians were free to decide on additional therapy in individual cases.

The standard examination technique, as previously described, was used in all the centers. The thoracic aorta was visualized in horizontal scan planes, with the exception of the upper part of the ascending aorta and the beginning of the aortic arch because of interposition of the trachea. After evaluation of the left ventricle and the aortic valve, the ascending aorta was visualized in the long axis to detect aortic insufficiency. The descending aorta was imaged usually from several centimeters below the diaphragm to the proximal abdominal aorta. The probe was pulled back stepwise in 3–5-cm increments with cross sections of the aorta labeled by noting the distance from the teeth in centimeters. At the level of the aortic arch, a distance of 18–23 cm from the teeth, the scope was rotated clockwise so that the aortic arch could be imaged in a longitudinal scan plane.

Color Doppler was used in every image plane to evaluate the flow in the true and false lumens and to detect small intimal tears seen as turbulent jets. The same standardized scan planes were recorded during initial and follow-up studies to detect changes in structure and flow patterns as described previously.

Echocardiographic Criteria

The diagnosis of aortic dissection was made if two lumens separated by an intimal flap could be seen within the aorta. Positive criteria were in case of complete obstruction of a false lumen, central displacement of intimal calcification, separation of intimal layers from the thrombus, and shearing of different wall layers. A tear was defined as a disruption in the continuity of the flap, with fluttering of the ruptured initial border. Smaller intimal tears could be detected by color Doppler, when jets traversing the dissection membrane were registered. The number of tears was recorded. The differentiation of the true from the false lumen was based on M-mode, two-dimensional, and Doppler signs. The criteria for identifying the true lumen were systolic expansion and diastolic collapse, the absence or lower density of spontaneous echocardiographic contrast, systolic jets directed away from the lumen, and systolic forward flow, whereas criteria for the false lumen were diastolic increase in the diameter; spontaneous echocardiographic contrast, reversed, delayed, or absent flow; and thrombus formation. Flow signals within the false lumen represented a sign of communication (c), whereas the absence of flow signals in multiple cross sections meant no communication (nc). If a communication was found distally to the beginning of the dissection, a retrograde dissection (cr) was present, which was further differentiated in retrograde dissections reaching or not reaching the ascending aorta and aortic arch (cr asc) or limited to the descending aorta (cr desc).

A thrombus was diagnosed when a mass that could be distinguished from the intimal flap and the aortic wall was imaged in the free space of the false or true lumen, taking into account the pathology criteria of Roberts.
and Roberts. Because thrombus formation is dependent on flow velocity, it was graded to provide an estimate of the degree of communication: 1) no thrombus formation, 2) segmental thrombus formation with partial or complete opacification of the false lumen in one cross-sectional image without further vertical progression, 3) extensive thrombus formation (i.e., thrombus formation in the false lumen in more than one cross-sectional image), and 4) complete thrombosis of the false lumen.

Echo-free spaces resulting from the presence of free fluid around the aorta were judged to be a sign of penetration and periaortic hematoma. Additional mediastinal hematoma (mh) was described when the distance from the esophagus to the left atrium or the aorta increased and pleural effusion developed. The probability was enhanced when pleurocentesis revealed hemorrhagic fluid. Pericardial effusion was described as an echo-free space between the epicardium and pericardium.

Aortic regurgitation was diagnosed when diastolic disturbed flow signals in the left ventricular outflow tract were detected. The grading was performed according to Perry et al.

### Table 1. Clinical Data

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>I ca</th>
<th>II ca/nc</th>
<th>III ca</th>
<th>III cr asc</th>
<th>III cr desc</th>
<th>III nc</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>No. a/c</td>
<td>No. a/c</td>
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<td>No. a/c</td>
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<td>Mean age (years)</td>
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<td>59/58/61</td>
<td>58/58/56</td>
<td>62/64/50</td>
<td>58/59/48</td>
<td>66/70/58</td>
</tr>
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<td>44/13</td>
<td>19/5/4</td>
<td>32/23/9</td>
<td>14/11/3</td>
<td>7/6/1</td>
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</tr>
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<td>9/5/4</td>
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<td>1/1/0</td>
<td>2/2/0</td>
</tr>
<tr>
<td>Pre-existing disease</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>2/0/2</td>
<td>-</td>
<td>2/2/0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Abdominal aneurysm</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Coarctation</td>
<td>-</td>
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<td>-</td>
<td>-</td>
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<td>-</td>
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<tr>
<td>Aortic valve prosthesis</td>
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<td>1/0/1</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Marfan syndrome</td>
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<td>-</td>
<td>-</td>
<td>-</td>
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</tr>
<tr>
<td>Aortitis luetica</td>
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<td>-</td>
<td>1/1/0</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>AD type III</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Preoperative mortality</td>
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<td>2/2/0</td>
<td>1/1/0</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Operation</td>
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<td>21/15/6</td>
<td>14/11/3</td>
<td>21/15/3</td>
<td>2/2/0</td>
<td>-</td>
</tr>
<tr>
<td>Reoperation</td>
<td>6/5/1</td>
<td>3/0/3</td>
<td>4/2/2</td>
<td>3/3/0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Medical therapy</td>
<td>7/7/0</td>
<td>7/5/2</td>
<td>27/21/6</td>
<td>1/1/0</td>
<td>6/5/1</td>
<td>11/7/1</td>
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<td>Survivors</td>
<td>30/26/4</td>
<td>18/11/7</td>
<td>26/19/7</td>
<td>12/10/2</td>
<td>7/6/1</td>
<td>9/5/3</td>
</tr>
</tbody>
</table>

a, acute AD; AD, aortic dissection; c, chronic AD; ca, communicating antegrade dissection; cr, communicating retrograde dissection limited to the descending aorta (cr desc); nc, noncommunicating type of dissection.
The DeBakey classification of aortic dissection was modified according to the above-mentioned criteria: type I ca, involvement of the ascending and descending aorta with communication and antegrade dissection; type II ca/cr/nc, ascending aorta only, communicating antegrade, retrograde, and noncommunicating dissection; type III ca, descending aorta, communicating, and antegrade dissection; type III cr asc, communication in the descending aorta and retrograde extension to the ascending aorta; and type III cr desc, communication in the distal part of the descending aorta with retrograde dissection but limited to the descending aorta (Figure 1).

**Surgical Techniques**

The surgical technique has been described previously and was left to the individual surgeon. For proximal dissections, the distal anastomosis was prepared in the aortic arch under deep hypothermia and circulatory arrest by the open technique. Cold cardioplegia was used. The ascending aorta was replaced in dissection involving the ascending aorta with or without reimplantation of the coronary arteries. End-to-end anastomosis to the graft was placed after circumferential transsection of the aorta. The aortic wall was sutured, and fibrin glue was applied to the false lumen. In some patients, the aortic wall was wrapped using Dacron felt sandwiched between the dissected layers. Aortic valve replacement was performed only when resuspension by supporting the commissures was not possible. In dissection involving the descending aorta alone, the diseased segment of the aorta with the primary tear was replaced with a woven Dacron tube. As the contracted inner lumen could not always be sutured to the tube graft, the true and false lumens were both attached after resection of part of the intimal flap. Distal aortic perfusion was performed by arteriofemoral bypass. The site was selected on the basis of sonography or angiography. Aortocoronary bypass surgery was performed when coronary luminal narrowing of more than 50% had been demonstrated at coronary angiography.

**Statistical Analysis**

The relation between the variables studied and the occurrence of late morbid events and mortality was determined using the univariate Cox-Mantel log-rank test with the corresponding Kaplan-Meier curves. The standard error estimated by the Kaplan-Meier can be obtained by a method described previously.

**Results**

Of the 168 patients (124 men and 44 women) who were included in the study, 58 (35%) had type I aortic dissection, 28 (17%) had a type II dissection, and 82 (48%) had a type III dissection. The patients’ characteristics and type of medical therapy are given in Table 1. The age of the patients ranged from 23 to 84 years, and the mean follow-up time was 10 months (range, 0–65 months).

**True and False Lumen, Intimal Tear, and Communication**

Although all true type I dissections were found to have communications and antegrade extension, the entry tear was in the descending aorta with retrograde dissection into the ascending aorta in 22 of 82 patients (27%) (type III cr asc). These were not included in the group (n=58) with communication in the ascending aorta and antegrade extent (type I ca). Only 52% of type I ca patients survived (Figure 2).

In type II dissections, two of 28 patients (7%) showed no communication (type II nc); one of the two died after surgery (Figure 3). During follow-up, 69% of the patients survived.

In 41 of 82 patients (50%) with type III dissection, a communication was found at the proximal part of the dissection and extended antegrade (III ca) (Figure 4). In 22 of 82 patients (27%), the tear was found in the descending aorta with retrograde dissection into the aortic arch or ascending aorta (III cr asc) (Figure 5). In eight of 82 patients (10%), the communication was not at the level of the subclavian artery but rather was more distally located and dissected retrogradely but remained limited to the descending aorta (III cr desc). Only one of eight patients died during follow-up. In type III dissections, 11 of 82 patients (13%) had no communication (nc), and only two patients died.

**Thrombus Formation in the False Lumen**

In nine of 54 patients (17%) with type I ca dissection, thrombus formation in the false lumen was found. After successful surgery, 24 of 30 survivors (80%) showed localized or more extensive thrombus formation, which was complete in 6.6% (Figure 6). Thus, 93% of the patients had a patent false lumen during follow-up. In type II, dissection surgery eliminated the false lumen in the ascending part in most patients. An open false lumen was seen in two of 18 patients (11%) without thrombus formation (Figure 6). In type III ca dissection, thrombus formation was present in 16 of 41 patients (39%). During medical therapy, it was observed in 22 of
27 patients (81%). After surgery, only a few showed progressive thrombus formation (Figure 7). Thrombus formation was present in six of 22 patients (27%) in type III cr asc dissection and in eight of 12 patients (67%) who survived. Only two of 12 patients (17%) showed complete thrombosis of the false lumen (Figure 7). More progressive thrombus formation occurred in types III cr desc and III nc dissection (Figure 8).

Fluid Extravasation

The echocardiographic data also were analyzed for the presence of fluid extravasation: pericardial tamponade, pleural effusion, periaortic hematoma, mediastinal hematoma, and/or aortic rupture (Table 2). It was shown that affected patients were at a high risk and had a high mortality.

Reoperation

Reoperation was necessary in six of 31 patients (19%) with type I ca dissection, in three of 16 patients (19%) with type II ca/nc dissection, and in seven of 21 patients (33%) with types III ca and III cr asc dissections. Table 3 shows the indication and time of the reintervention. Figures 6–8 show the relation to the subtypes and thrombus formation.

Mortality

Table 4 gives the number and causes of death in the different patient groups. Figure 9 gives the survival curves for patients with types I ca and II ca/nc and all type III dissection, and Figure 10 gives the survival curves of the subtypes of type III aortic dissection.

Discussion

Effect of Medical and Surgical Therapy on Communication Between True and False Lumens and Thrombosis

Thrombosis in the false lumen was observed in type I ca aortic dissections in 17% of the patients and was progressive during follow-up in about 80% of surviving patients. Progressive thrombus formation as a sign of healing was observed by autopsy in only 6% of those with entry tears in the ascending aorta. In our study, complete elimination of the false lumen in the ascending aorta was seen after resection or wall wrapping in type II dissections, and thrombus formation was found in 30–40% of the patients in type III aortic dissections. The latter results compare well with those of autopsy studies reporting complete thrombosis in 45% of type III dissections.

Thrombus formation appears to depend on the subtype of dissection, which has not been differentiated previously. In noncommunicating and retrograde dissections confined to the descending aorta, thrombosis was more common than in communicating antegrade and retrograde dissections into the aortic arch and ascending aorta. Complete obliteration of the false lumen can occur despite open communication and possibly is related to the degree of communication. Patients who already have thrombus formation during the acute study seem to have a lower mortality, particularly when thrombus formation during follow-up progressed. This
In our study, persistent false lumen was present in more than 90% of the patients with type I dissection, confirming previously reported preliminary data. Using computed tomography, seven of eight patients in whom obliteration was attempted by surgery demonstrated persistence of the false lumen, whereas in type III dissection a patent false lumen was less often found and progressive thrombus was more common (30%). Using angiography in nine of 11 patients with types I, II, and III dissections, all had persistent intimal flaps, and two had residual free communications. Thus, surgical interventions in type I aortic dissections can interrupt the progression of the dissection into the aortic root and pericardium but rarely leads to complete obliteration of the false lumen. This relates to the finding of multiple tears in the ascending and descending aorta in type I dissection, as detected by color Doppler, which remain untreated. It explains that resection of tears only in the ascending aorta cannot be of prognostic significance and that the rate of reoperation seems not to be significantly lower.

Our follow-up study suggests that it is important to detect and resect intimal tears as patients with communication have a higher reoperation and mortality. These complications are more frequent if there are signs of high flow in the false lumen. Mortality reached 37% (type III ca) and 43% (type III cr asc) in these patients. Arch repair for type I aortic dissection with tears in the transverse arch has been recommended. Until now, only a few surgeons have attempted to occlude all tears based on intraoperative epicardial...
Aortic Dissection Type III ca (41)  
FL Thrombosis

unknown  4  
0  21  
I  6  
II  9  
III  1  
Total  41

1st TEE

Aortic Dissection Type III cr asc (22)  
FL Thrombosis

unknown  2  
0  14  
I  3  
II  3  
III  0  
Total  22

FU TEE

FIGURE 7. Thrombus formation in the false lumen (FL) for types III ca and III cr asc aortic dissection. OP, surgery; +a, preoperative, +b, intraoperative, and +c, postoperative mortality. Degree of thrombus formation is indicated by 0, no thrombus visible; 1, localized thrombus formation visible; 2, extended thrombus formation in the false lumen; and 3, complete thrombus formation.

and transesophageal echocardiography, some going so far on to perform additional laparotomy. This has been found to lead to a reduction in the need for reoperation. For type III dissections, a dismal prognosis is still reported, suggesting that further improvement of the surgical technique is needed. But it has

Aortic Dissection Type III cr desc (8)  
FL Thrombosis

unknown  2  
0  2  
I  3  
II  3  
III  0  
Total  8

1st TEE

Aortic Dissection Type III nc (11)  
FL Thrombosis

unknown  2  
0  2  
I  2  
II  3  
III  2  
Total  11

spontaneous healing

FU TEE

FIGURE 8. Thrombus formation in the false lumen (FL) for types III cr desc and III nc aortic dissection. OP, surgery; +a, preoperative, +b, intraoperative, and +c, postoperative mortality. Degree of thrombus formation is indicated by 0, no thrombus visible; 1, localized thrombus formation visible; 2, extended thrombus formation in the false lumen; and 3, complete thrombus formation.
TABLE 2. Fluid Extravasation in Aortic Dissection

<table>
<thead>
<tr>
<th>AD type</th>
<th>No. of patients</th>
<th>Pericardial tamponade (a/c)</th>
<th>Pleural effusion (a/c)</th>
<th>Periaortic hematoma (a/c)</th>
<th>Mediastinal hematoma/hematotherax (a/c)</th>
<th>Aortic rupture (a/c)</th>
<th>Surgery (a/c)</th>
<th>Mortality (a/c)</th>
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<td>I ca</td>
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<td>5/0</td>
<td>10/0</td>
<td>2/0</td>
<td>1/0</td>
<td>16/0</td>
<td>8/16 /—</td>
</tr>
<tr>
<td>II ca/nc</td>
<td>8/28</td>
<td>3/0</td>
<td>0/1</td>
<td>4/0</td>
<td>1/0</td>
<td>1/0</td>
<td>7/0</td>
<td>5/7 /0/1</td>
</tr>
<tr>
<td>III ca</td>
<td>12/41</td>
<td>—</td>
<td>2/1</td>
<td>4/0</td>
<td>4/2</td>
<td>1/0</td>
<td>6/3</td>
<td>4/6 /2/6</td>
</tr>
<tr>
<td>III cr asc</td>
<td>9/22</td>
<td>3/1</td>
<td>1/0</td>
<td>1/0</td>
<td>1/1</td>
<td>1/0</td>
<td>7/2</td>
<td>3/7 /1/2</td>
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<tr>
<td>III cr desc</td>
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<td>1/0</td>
<td>—</td>
<td>1/0</td>
<td>—</td>
<td>1/0</td>
<td>1/1 /0/1</td>
</tr>
<tr>
<td>III nc</td>
<td>3/11</td>
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<td>1/0</td>
<td>—</td>
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<td>1/0</td>
<td>—</td>
<td>1/2 /1/1</td>
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</tbody>
</table>

a, acute AD; AD, aortic dissection; c, chronic AD; ca, communicating antegrade dissection; cr, communicating retrograde dissection limited to the descending (desc) or expanding to the aortic arch and ascending (asc) aorta; nc, noncommunicating dissection.

to be taken into account that closure and thrombosis of the false lumen can be deleterious when organs are perfused via the false lumen. Side branches can be separated from the inner lamina of the aorta and perfused via the false lumen. Improved imaging capabilities, possibly including intraoperative ultrasound, will help to clarify these questions in the future.

Antegrade and Retrograde Aortic Dissection

For further differentiation, we considered the extent of the dissection in addition to the position of the tear as the disease can progress not only antegrade but also retrograde. Patients with tears distal to the subclavian artery and retrograde dissection up to this point (type III cr desc) are at a lower risk than those with more proximal tears extending into the aortic arch and ascending aorta (type III cr asc) and those with antegrade dissection (III ca). Progressive and complete thrombus formation despite communication was found more often in the first than in the latter groups. We have found that patients with tears in the descending aorta and retrograde dissection up to the ascending aorta have a poor prognosis. Present surgical techniques do not result in progression of thrombus formation as replacement of the ascending aorta cannot eliminate flow in the false lumen and support thrombus formation.

Noncommunicating Aortic Dissection

In accordance with the reported pooled data of pathoanatomic studies, the incidence of noncommunicating aortic dissection in this study was 12%, based on the absence of Doppler flow signals, thrombus formation, absence of intimal flap movement, and absence of tears on cross-sectional and color Doppler echocardiograms. Others have reported an incidence between 3% and 5%. In our series, patients with noncommunicating aortic dissection had a better prognosis than those with a communication. Dinsmore et al, using angiography, also reported a better survival (90%) for these patients. Furthermore, the extent of thrombus formation also

TABLE 3. Reoperation After Surgery for Aortic Dissection

<table>
<thead>
<tr>
<th>Type</th>
<th>No. of patients</th>
<th>a/c</th>
<th>Time</th>
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</thead>
<tbody>
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<td>a</td>
<td>1 d</td>
<td>Bleeding, hematotherax</td>
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<tr>
<td></td>
<td></td>
<td>a</td>
<td>3 M</td>
<td>Progressive dilatation</td>
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<td></td>
<td></td>
<td>a</td>
<td>5 M</td>
<td>Progressive dilatation</td>
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<tr>
<td></td>
<td></td>
<td>a</td>
<td>18 M</td>
<td>Progressive distal AD rupture</td>
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<td></td>
<td></td>
<td>c</td>
<td>24 M</td>
<td>Traumatic new progressive AD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a</td>
<td>30 M</td>
<td>Progressive distal AD</td>
</tr>
<tr>
<td>II ca/nc</td>
<td>3</td>
<td>c</td>
<td>1 d</td>
<td>Progressive distal AD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>c</td>
<td>3 d</td>
<td>Tamponade</td>
</tr>
<tr>
<td></td>
<td></td>
<td>c</td>
<td>5 M</td>
<td>Dilatation of FL</td>
</tr>
<tr>
<td>III ca</td>
<td>4</td>
<td>c</td>
<td>1 d</td>
<td>Hematotherax</td>
</tr>
<tr>
<td></td>
<td></td>
<td>c</td>
<td>14 d</td>
<td>Bleeding, hematotherax</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a</td>
<td>25 d</td>
<td>Progressive distal AD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>a</td>
<td>1 M</td>
<td>Retrograde dissection</td>
</tr>
<tr>
<td>III cr</td>
<td>3</td>
<td>a</td>
<td>1 d</td>
<td>Bleeding</td>
</tr>
<tr>
<td>asc</td>
<td>2 d</td>
<td>Retrograde AD</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 d</td>
<td>Bleeding</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

III cr desc—no reoperation
III nc—no operation/reoperation

AD, aortic dissection; d, days; FL, false lumen; M, months; a, acute AD; c, chronic AD; ca, communicating antegrade dissection; cr, communicating retrograde extending to the ascending aorta (asc), limited to the descending aorta (desc); nc, noncommunicating type of dissection.
Survival curves for patients with type I (n=58), II (n=28), and III (n=82) aortic dissection.

Survival seems to be important. Only patients in whom no thrombus formation was present at the first examination died; this may relate to the fact that these patients are already in a more chronic state because of the difficulty of diagnosis in this subgroup. Patients with noncommunicating aortic dissection were nearly 10 years older than those with communication, which may point to a different pathogenesis in this group.

Noncommunicating aortic dissection should be differentiated from intramural hematoma as the latter are localized in circumscribed parts of the aortic wall and can lead to both communicating and noncommunicating dissections.38,39

Spontaneous Healing

Spontaneous healing of aortic dissections can occur. The false lumen disappears, and as a residual sign, wall thickening can be observed.40-42 In our study, this thickening was observed only in 4% of patients with types II and III aortic dissections, in a small number of patients by magnetic resonance tomography in 7%,43 and by computed tomography in 31% of type III dissection.25 Continuous flow via large entry tears seems to prevent spontaneous healing. Moreover, we have demonstrated that dissection occurring in patients undergoing aortic balloon angioplasty also heal spontaneously,40 as has been described by others.25,42 It is different from the healing described as a result of complete thrombosis of the false lumen.15,42,43

Reoperation

Surgical reintervention in our study—14-29% depending on the type of dissection—was comparable to

Survival curves for patients with aortic dissection type III subdivided into those with communication and antegrade dissection (type III ca, n=41), communication and retrograde dissection extending to the aortic arch and ascending aorta (type III cr asc, n=22), communication and retrograde dissection limited to the descending aorta (type III cr desc, n=8), and noncommunicating aortic dissection (type III nc, n=11).
**TABLE 4. Death During Follow-up of Aortic Dissection**

<table>
<thead>
<tr>
<th>Type</th>
<th>No. of patients</th>
<th>Cause</th>
<th>Patients a/c</th>
</tr>
</thead>
<tbody>
<tr>
<td>I ca</td>
<td>28/58 (48%)</td>
<td>Aortic rupture</td>
<td>3/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Low cardiac output</td>
<td>9/7/2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary failure</td>
<td>2/2/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Renal failure</td>
<td>5/5/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Infection</td>
<td>3/2/1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cerebral damage</td>
<td>3/3/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Unknown</td>
<td>3/3/0</td>
</tr>
<tr>
<td>II ca/nc</td>
<td>10/28 (36%)</td>
<td>Aortic rupture</td>
<td>2/2/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Low cardiac output</td>
<td>3/3/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sudden death</td>
<td>1/1/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cerebral damage</td>
<td>2/2/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Infection</td>
<td>2/1/1</td>
</tr>
<tr>
<td>III ca</td>
<td>15/41 (37%)</td>
<td>Aortic rupture</td>
<td>4/4/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Low cardiac output</td>
<td>2/2/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bleeding intraoperative</td>
<td>1/1/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Myocardial infarction</td>
<td>2/1/1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary failure</td>
<td>1/1/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Renal infarction</td>
<td>2/2/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Infection</td>
<td>1/0/1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Unknown</td>
<td>2/2/0</td>
</tr>
<tr>
<td>III cr asc</td>
<td>10/22 (45%)</td>
<td>Aortic rupture</td>
<td>2/2/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Myocardial infarction</td>
<td>1/1/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Renal failure</td>
<td>1/1/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bleeding intraoperative</td>
<td>1/1/0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Low cardiac output</td>
<td>5/4/1</td>
</tr>
<tr>
<td>III cr desc</td>
<td>1/8 (12.5%)</td>
<td>Aortic rupture</td>
<td>1/1/0</td>
</tr>
<tr>
<td>III nc</td>
<td>2/11 (18%)</td>
<td>Aortic rupture</td>
<td>2/1/1</td>
</tr>
<tr>
<td>Not dissection related</td>
<td>5</td>
<td>Myocardial infarction</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Death after CABG</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carcinoma</td>
<td>1</td>
</tr>
</tbody>
</table>

a, acute AD; AD, aortic dissection; c, chronic AD; ca, communicating antegrade dissection; cr, communicating retrograde extending to the aortic arch and ascending aorta (asc), limited to the descending aorta (desc); nc, noncommunicating type of dissection.

the experience reported by others (10—40%).\(^{9,22,44,45}\) Interestingly, in most patients the causes of reoperation were bleeding with hematothorax, dilatation of the aorta, and progressively spreading dissection.\(^{44-46}\) This may be related to the finding of residual open communications after surgery, resulting in persistent high pressure in the false lumen and high wall stress. In these patients, only localized or no thrombus formation was found.

**Preoperative Mortality**

This study demonstrates that despite early diagnosis (by using modern imaging techniques), aortic dissection still has a high mortality. Some patients die before surgery can be performed (2.9%). This is related to the type of dissection. The overall mortality is lower than the preoperative mortality rate of 6.8% reported by Glower et al.,\(^{44}\) which may be due to the introduction of transesophageal echocardiography, allowing rapid bedside diagnosis.\(^{10,11}\)

**Intraoperative Mortality**

Improved surgical techniques have significantly decreased intraoperative mortality over the past years.\(^{1,2,19,44,45}\) In our study, intraoperative mortality for type I dissections was 12%; for type II dissections, 5%; and for type III dissections, 23%. Others have reported mortalities of 11%, 14%, and 25%, respectively.\(^{44}\) Previously, the mortality was 24—89% in acute and 14—67% in chronic aortic dissections.\(^{1,3,5,6,31,46-48}\)

For type III dissections, intraoperative mortality still has not been reduced, reaching 25% in recent series compared with 16—37% in previous reports.\(^{1-3,19,46-48}\) Type III chronic dissections seem to present a lower intraoperative risk than acute dissections.\(^{3,46,48}\)

**Hospital and Follow-up Mortality**

During follow-up, we found the highest mortality occurred during hospitalization. Others recently have also reported a high hospital mortality, reaching 26% for type I, 14% for type II, and 62% for type III dissections.\(^{44}\) The causes of death in our series are similar to those reported by others.\(^{3,5,19}\) In a review of 715 patients, mortality of 24% was reported for patients who had undergone surgery and of 24% for medically treated patients with a rupture in the vicinity of the entrance tear.\(^{44}\) However, such a literature review may be misleading because most type III dissections are treated medically, which in turn leads to a mismatch of medically and surgically treated patients. The high mortality (62%) reported for surgical patients with type III dissections and aortic ruptures corresponds to other reports.\(^{31,44}\) A sign of aortic rupture is extravasation of fluid, which can be visualized with high sensitivity by transesophageal echocardiography. Pleural effusion is a sign of hematothorax, which has to be confirmed by needle puncture.\(^{13}\) We were able to demonstrate that patients with mediastinal hematoma are at an extremely high risk independent of whether they then have a proximal or distal dissection. These patients die rapidly during a second event and sometimes during diagnostic procedures. The mortality of patients with fluid extravasation was 51% overall, reaching 75% in acute type ca/nc dissections. Although it is common practice not to recommend surgical intervention in acute type III dissections,\(^{6,19,44}\) our study suggests that urgent surgery be considered for patients with signs of fluid extravasation because of the high mortality when medically treated. Only 50% of the patients survive 36 months. Current reported survival rates suggest a more aggressive approach.\(^{9}\) Early surgery seems to be indicated in patients with type III ca dissections and type III cr asc retrograde dissections into the ascending aorta.

**Study Limitations**

This cooperative study is based on transesophageal echocardiography, including color Doppler echocardiography. Thus, only the thoracic part of the aorta was examined, not the abdominal part, limiting the information obtained about the extent of types I and III dissections.

Transesophageal echocardiography is able to visualize the aortic root and the ascending aorta up to the cranial part of the right pulmonary artery. Due to the interposition of the trachea, the bifurcation, and the left
bronchus, the distal part of the ascending part of the aortic arch cannot be visualized.10 This means that the distal extent in type II dissections and the proximal part of type III dissections, starting in the aortic arch, sometimes are not seen, even by biplane echocardiography, when they are located in the ascending part of the aortic arch. Computed tomography and magnetic resonance imaging can be used to obtain this additional information.43,49 Intravascular ultrasound also appears to be a promising new technique that may overcome current limitations.50 The information not obtained for these reasons would not have influenced the conclusion of this study insofar as the other parts of the aorta are very well imaged, allowing the detection of entry tears, thrombus formation, intimal flaps, and flow in the true and false lumens.

Acknowledgments

The authors would like to thank all cooperative colleagues, technicians, and nurses, as well as Mrs. Herbrik for her secretarial, Mrs. Gräwe for her photographic, and Mrs. Neuser for her graphical work. We thank also Dr. P. Kearney and Dr. J. Zamorano for their comments.

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Circulation. 1993;87:1604-1615
doi: 10.1161/01.CIR.87.5.1604

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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