

## Predictors of complications in acute type B aortic dissection

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### Abstract

**Objectives:** Medical treatment is generally advocated for patients with acute type B aortic dissection without complications. The objective of this retrospective analysis was to determine whether there are any initial findings that can help predict the long-term course of the disease. **Methods:** Case records of the 130 patients treated for type B aortic dissection between 1988 and 1997 were reviewed; 41 (31%) were operated on in the acute phase (<14 days), 31 (24%) were operated on in the chronic phase and 58 (45%) were treated medically. **Results:** Overall acute mortality was 10.8%; 22% for patients operated on in the early phase and 5.6% for medically treated patients. Age ( $P = 0.002$ ), persistent pain ( $P = 0.01$ ) and malperfusion ( $P = 0.001$ ) were significant independent predictors of the need for surgery. Paraplegia/paraparesis ( $P = 0.0001$ ), leg ischaemia ( $P = 0.003$ ), pleural effusion ( $P = 0.003$ ), rupture ( $P = 0.0001$ ), shock ( $P = 0.0001$ ), age ( $P = 0.003$ ), cardiac failure ( $P = 0.002$ ) and aortic diameter  $>4.5$  cm ( $P = 0.002$ ) were significant predictors of poor survival. Age and shock also emerged as independent risk factors. Patients without malperfusion ( $P = 0.0001$ ), pleural effusion ( $P = 0.003$ ), rupture ( $P = 0.0001$ ) and shock ( $P = 0.0001$ ) had a significantly better event-free survival (freedom from repeat surgery and death). The actuarial survival rate for high-risk patients (malperfusion, rupture, shock) was 62% at 1 year and 40% at 5 years; the corresponding values for low-risk patients were 94 and 84%, respectively. **Conclusions:** Rupture, shock and malperfusion are significant predictors of poor survival in patients with acute type B aortic dissection. © 2002 Published by Elsevier Science B.V.

**Keywords:** Acute type B aortic dissection; Survival; Mortality; Predictors

### 1. Introduction

Dissection is the most common catastrophic event that affects the thoracic and abdominal aorta. Unfortunately, it also remains one of the most challenging disorders facing the cardiovascular surgeon. There is still some controversy concerning the therapeutic strategies for patients with type B aortic dissection [1]. The gradual improvement in recent years of outcome after surgery for acute dissection of the aorta has resulted in a consensus favouring operative intervention for almost all type A dissections and an increasing number of type B dissections. Although most surgeons now agree that immediate surgery is appropriate for acute type B dissection if there is intractable pain, uncontrollable hypertension or serious organ malperfusion [2], medical treatment with a beta blocker is generally advocated for patients without complications [3]. One important question, however, is whether any predictors of negative survival of a patient with

type B aortic dissection already exist at the time of initial hospitalisation.

### 2. Materials and methods

Between 1988 and 1997, 130 patients were treated for type B aortic dissection at the University Hospital Zurich. Of these, 26 (20%) were females and 104 (80%) were males. The mean ( $\pm$ SD) age at the time of hospitalisation was  $61.0 \pm 11.2$  years (range 23–84 years); 80 patients (61.5%) were more than 60 years old and 31 (23.8%) were more than 70 years old. Symptoms and clinical findings at the time of hospitalisation are listed in Table 1. The patients were also suffering from the following conditions: hypertension (81.5%), coronary artery disease (23.8%), gastrointestinal disease (16.9%), previous aortic surgery (13.8%), renal insufficiency (11.5%), chronic obstructive pulmonary disease (10.8%), heart failure (5.4%), previous neurological event (3.8%), diabetes mellitus (3.1%) or others (26.2%). The diameter of the aorta at the time of diagnosis was  $<3.4$  cm in 16.1%, 3.5–4.4 cm in 33.9%, 4.5–5.4 cm in 27.4%, 5.5–6.4 cm in 11.3% and  $>6.5$  cm in 11.3%.

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Table 1  
Symptoms and clinical findings at hospitalisation in relation to acute and in-hospital mortality rate

	Number of patients	Acute mortality N (%)	In-hospital mortality N (%)
Pleural effusion	44	8 (18%)	12 (27%)
Visceral ischaemia	24	0	3 (12.5%)
Aortic wall haematoma	20	2 (10%)	3 (15%)
Leg ischaemia	19	6 (32%)	6 (32%)
Persistent pain	17	0	0
Rupture	15	8 (53%)	9 (60%)
Aortic diameter progression	7	1 (14%)	1 (14%)
Shock	6	4 (67%)	6 (100%)
Resistant blood pressure	3	0	0
Para paresis	3	1 (33%)	1 (33%)
Paraplegia	2	2 (100%)	2 (100%)
Fistula	1	0	1 (100%)

Fifty-two of the 130 patients (40%) were treated surgically at first hospitalisation (Fig. 1), 41 as emergency cases during the acute phase (<14 days after onset of symptoms) and 11 as urgent cases before discharge. The most frequent indications for emergency surgery were malperfusion (34%) (presenting as leg ischaemia in eight patients and visceral ischaemia in six patients, five of them with renal ischaemia and one with ischaemia of the coeliac axis), potential rupture (27%) (presenting as pleural effusion in eight patients and large (>6 cm) aortic diameter in three patients) and aortic rupture (19%). Indications for urgent surgery, i.e. before discharge from the first hospitalisation, were malperfusion (36%), left pleural effusion (18%), increasing aortic diameter (18%), persistent pain (9%) and other indications (19%).

In the absence of rupture or complications of the aortic dissection, the remaining 78 patients (60%) were primarily treated medically. The mean ( $\pm$ SD) age of the medically treated patients was  $64.0 \pm 12.3$  years. As previously recommended [3], the majority of these patients (51/78; 65.4%) received treatment with beta blockers.

The records of all patients who were treated for type B aortic dissection were reviewed. Those patients who were still alive were contacted and asked to complete a question-

naire with the help of their doctor; particular attention was focused on collecting CT scan data. The total follow-up period encompassed 194 years, with a mean observation time of  $4.2 \pm 2.2$  years. The in-hospital mortality rate was 14.6% (19/130) and, of the remaining 111 patients, follow-up was completed in 105 (95%). A total of 37 patients died during follow-up.

In order to determine any predictors affecting survival rate and survival rate free from any event (death and surgery, respectively) and/or influencing the indication for surgery, the following variables were analysed: persistent pain, paraplegia/para paresis, malperfusion, leg ischaemia, visceral ischaemia, persistent hypertension, pleural effusion, rupture, shock, aortic diameter at hospitalisation, poor left ventricular ejection fraction, cardiac failure, coronary artery disease, known hypertension, diabetes mellitus, arteriosclerosis, previous aortic surgery, pre-existing neurological, intestinal, renal or pulmonary disease, localisation of the dissection and the condition of the false lumen.

### 2.1. Statistical analyses

Variables were reported either as a percentage or mean  $\pm$  standard deviation. Statistical analyses were performed using SPSS 6.1 software. Nominal variables were evaluated using the Chi-quadrant test and independent variables using the Mann–Whitney and Kruskal–Wallis test; univariate analyses were performed on continuous variables using the Wilcoxon-signed rank test. Differences between groups were analysed using the log-rank test. Significance was assumed at a *P* level of <0.05.

## 3. Results

The overall mortality rate in the acute phase was 10.8% (14/130); 5.6% (5/89) of patients who had until then only received medical treatment and 22% (9/41) of patients operated on in this phase (i.e. emergency surgery). The in-hospital mortality rate was 9% (7/78) for the medically treated patients, compared with 9.1% (1/11) for patients undergoing

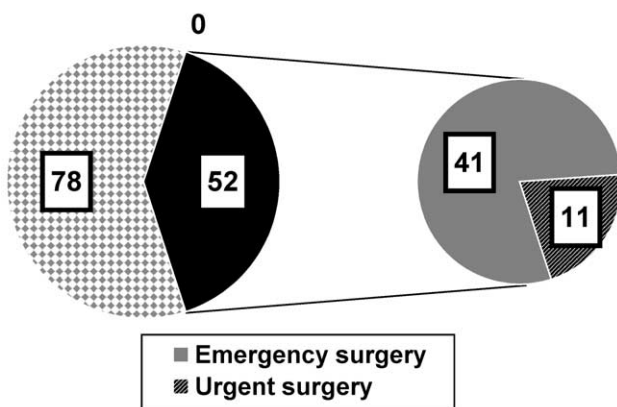


Fig. 1. Therapeutic strategies in the treatment of acute type B aortic dissection.

Table 2  
Factors significantly affecting necessity for surgery<sup>a</sup>

Factor	P value
Age	0.002
Persistent pain	0.01
Malperfusion	0.008
Leg ischaemia	0.0001
Rupture	<0.0001

<sup>a</sup> Note: age, pain and malperfusion are also independent factors.

urgent surgery and 27% (11/41) for those undergoing emergency surgery. A total of 111/130 patients were discharged alive.

The symptoms and clinical findings that resulted in hospitalisation are shown in Table 1. Pain is the most important symptom of a type B aortic dissection, although it persisted in only 13% of the patients at hospitalisation. For acute mortality, the single symptoms/clinical findings associated with the highest mortality rates were paraplegia (100% mortality rate), shock (67%), rupture (53%) and leg ischaemia (33%). In-hospital mortality was highest for patients with paraplegia, shock, rupture, para paresis and leg ischaemia.

Twenty patients had surgery after discharge from their first hospitalisation. The main reason for surgical intervention was an increase of the diameter of the aorta (14/20), followed by visceral ischaemia (2/20), persistent pain (2/20), leg ischaemia (1/20) and possible rupture (1/20). The initial findings in this group of patients were large aortic diameter (8/20), haematoma of the aorta (3/20), leg ischaemia (3/20), persistent pain (2/20), visceral ischaemia (2/20), resistant blood pressure (1/20) and pleural effusion (1/20). Sixteen patients required a repeat of their surgery during the follow-up period; initial findings in this group included pleural effusion >300 ml on the left side (6), visceral ischaemia (5), persistent pain (4), leg ischaemia (2), resistant blood pressure (1) and haematoma of the aortic wall (1).

As shown in Table 2, age ( $P = 0.002$ ), persistent pain ( $P = 0.01$ ), malperfusion ( $P = 0.008$ ), leg ischaemia ( $P = 0.0001$ ) and rupture ( $P < 0.0001$ ) significantly influenced the necessity for initial surgery. Age, persistent pain and malperfusion were also independent factors. Pre-operative

Table 3  
Factors with a significant negative effect on survival rate<sup>a</sup>

Factor	P value
Paraplegia/para paresis	0.0001
Leg ischaemia	0.003
Pleural effusion	0.003
Rupture	0.0001
Shock	0.00001
Age	0.003
Cardiac failure	0.002
Aortic diameter >4.5 cm	0.002

<sup>a</sup> Note: age and shock are also independent factors.

paraplegia/para paresis ( $P = 0.0001$ ), leg ischaemia ( $P = 0.003$ ), pleural effusion ( $P = 0.003$ ), rupture ( $P = 0.0001$ ), shock ( $P = 0.00001$ ), age ( $P = 0.003$ ), cardiac failure ( $P = 0.002$ ) and aortic diameter >4.5 cm ( $P = 0.002$ ) had a significant negative effect on survival rate (Table 3). Age and shock were also independent factors. For those patients who were treated primarily with surgery, rupture ( $P = 0.006$ ) and pleural effusion ( $P = 0.02$ ) are the only factors that had a significant negative effect on survival rate. Rupture was the single independent factor in these patients. Amongst patients who were treated medically, age ( $P = 0.006$ ), gender ( $P = 0.009$ ), malperfusion ( $P = 0.008$ ), leg ischaemia ( $P = 0.0003$ ), pleural effusion ( $P = 0.0007$ ), rupture ( $P < 0.0001$ ), shock ( $P < 0.0001$ ), cardiac failure ( $P = 0.002$ ) and aortic diameter >4.5 cm ( $P = 0.002$ ) were all factors that had a significant negative effect on survival rate. There were no independent factors in this group of patients. The actuarial survival rate for high-risk patients (malperfusion, rupture, shock) was 62% at 1 year and 40% at 5 years. The corresponding values for low-risk patients were 94 and 84%, respectively. Predisposing disease does not influence actuarial survival.

Malperfusion ( $P = 0.0002$ ), leg ischaemia ( $P = 0.0001$ ), pleural effusion ( $P = 0.003$ ), rupture ( $P < 0.0001$ ), shock ( $P < 0.0001$ ), hypertension ( $P = 0.004$ ) and aortic diameter >4.5 cm ( $P = 0.008$ ) had a significant negative effect on survival rate free from any event. Shock was the only independent factor.

Fig. 2 shows survival curve reflecting freedom from any event for high- and low-risk patients surviving the first 30 days.

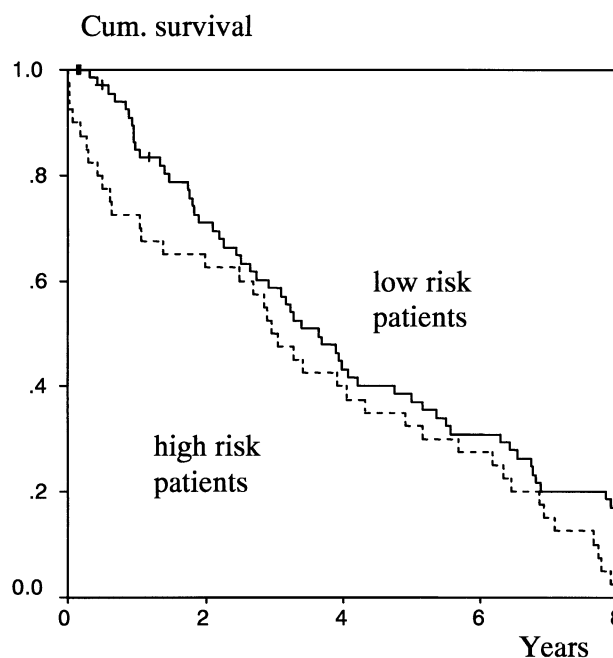


Fig. 2. Actuarial survival curve reflecting freedom from any event for high-risk and low-risk patients.

#### 4. Discussion

Specific initial findings that are indications for emergency surgery (rupture, shock, organ ischaemia) negatively affect the course of type B aortic dissection. It has been generally advocated that patients who have type B acute aortic dissection without complications, such as rupture, potential rupture or organ ischaemia, should be treated with hypotensive drugs during the acute phase and that surgical treatment be carried out if the aortic diameter becomes enlarged during the chronic phase. However, there is some controversy concerning patients with type B aortic dissection. Higher early mortality with surgical treatment than with medical treatment, higher operative mortality in the acute phase than in the chronic phase and higher late mortality in patients treated only medically than in those treated surgically have all been recognised in many institutions [1,4,5].

In our experience, uncomplicated dissections are not an indication for surgery. The survival rate of patients treated medically in the acute phase can be improved significantly by long-term beta-blocker treatment [3]. The higher mortality rate in the acute phase of type B aortic dissection, when surgical treatment is inevitable due to the previously discussed clinical findings, as well as the in-hospital mortality of type B acute aortic dissection, underline the diversity of the two patient groups regarding their risk profile. In contrast to the medically treated patients, surgical patients are, in general, those who have the factors that negatively affect survival rate. In fact, in our patient series, indications for emergency surgery were malperfusion in 34%, potential rupture in 27% and rupture in 19%. In addition to these factors, age also has a statistically significant effect on survival rate. The higher mortality rate in the acute phase was due to patients with rupture (mortality rate 53.3%) and malperfusion (mortality rate 19%). Together with shock and paraplegia, these two clinical findings have the highest mortality rate in the acute phase. Therefore, compared with medically treated patients, surgical patients are at high risk and it would be an error to compare the survival of these two patient groups.

Although a diagnosis of rupture is clinically clear in the majority of cases, diagnoses of malperfusion of the truncus coelicaeus, mesenterial and renal arteries in patients with type B aortic dissection is extremely difficult despite the fact that malperfusion occurs in up to 30% of patients with an aortic dissection [6–8]. In agreement with Webb and Williams [6], we hypothesise that re-entry, either spontaneous or surgical, is essential to prevent malperfusion in type B aortic dissection. In some cases, atypical abdominal pain may be the only sign of a malperfusion. A suspected diagnosis of malperfusion is therefore frequently only given when minimal clinical signs, such as abdominal tension, increasing metabolic acidosis, progressive elevation of liver enzymes and uncontrollable hypertension as a sign of decreased renal perfusion, persist [9]. The difficult and late diagnosis of visceral malperfusion is one of the reasons why a large proportion of our patients are not operated on in

the acute phase (2 weeks from diagnosis) and why the mortality rate is also increased after this time. When malperfusion is suspected, aggressive clinical, laboratory and radiological assessment is recommended.

In agreement with Carrel et al. [10], rupture and malperfusion do not appear to be the only factors that negatively affect the survival rate at the time of diagnosis of type B aortic dissection. These other factors include pre-existing cardiac failure or pre-operative paraplegia, pleural effusion and the aortic diameter. However, in contrast, Juvonen et al. [11] reported that aneurysm size, as defined by a variety of dimensional variables including maximal diameter in the descending thoracic aorta, is apparently not a significant predisposing factor for rupture. Nevertheless, both pleural effusion and enlarged aortic diameter are clinical findings that are associated with potential rupture.

Enlarged aortic diameter was also found to be a predictor of worse course in the whole group of patients and in the group who were primarily medically treated; pleural effusion was also a predictor in surgically treated patients. The risk of rupture, which is usually fatal, must, however, be balanced against the not inconsiderable morbidity and mortality associated with elective surgery [12]. Calculation of rupture risk for a patient with chronic type B dissection according to the formula developed for patients with non-dissecting aneurysms would be likely to somewhat underestimate the risk for rupture, although it might nevertheless be helpful in trying to determine which individual patients are most vulnerable [11]. For these patients with pleural effusion and/or enlarged aortic diameter, the risk of elective surgery for chronic type B dissection is warranted because rupture is imminent. It is important not to forget, however, the favourable effect of beta blockers in preventing enlargement of the diseased aorta and in increasing survival of chronic type B aortic dissection [3]. Nevertheless, the therapeutic strategies for type B aortic dissection have changed in the previous years, particularly for malperfusion and the locally enlarged aorta, where new interventional methods with fenestration and endoaortic prosthesis show promising results [13,14].

#### References

- [1] Neya K, Omoto R, Kyo S, Kimura S, Yokoto Y, Takamoto S, Adachi H. Outcome of Stanford type B acute aortic dissection. *Circulation* 1992;86(Suppl II):1–7.
- [2] Eleftheriades JA, Hartleroad J, Gusberg RJ, Salazar AM, Black HR, Kopf GS, Baldwin JC, Hammond GL. Long term experience with descending aortic dissection: the complication-specific approach. *Ann Thorac Surg* 1992;53:11–21.
- [3] Genoni M, Paul M, Jenni R, Graves K, Seifert B, Turina M. Chronic beta-blocker therapy improves outcome and reduces treatment costs in chronic type B aortic dissection. *Eur J Cardiothorac Surg* 2001;19:606–610.
- [4] Appelbaum A, Karp RB, Kirklin JW. Ascending versus descending aortic dissection. *Ann Surg* 1976;183:296–300.
- [5] Wheat MW. Acute dissecting aneurysms of the aorta: diagnosis and treatment. *Am Heart J* 1980;99:372–387.

- [6] Webb T, Williams GM. Abdominal aortic tailoring for renal, visceral, and lower extremity malperfusion resulting from acute aortic dissection. *J Vasc Surg* 1997;26:474–480 (see also Discussion p. 480–1).
- [7] Fann J, Smith J, Miller D, Mitchell R, Moore K, Grunkemeir G, Stinson E, Oyer P, Reitz B, Shumway N. Surgical management of aortic dissection during a 30-year period. *Circulation* 1995;92(Suppl 9):113–121.
- [8] Slonim S, Nymann U, Semba C, Miller D, Mitchell R, Dake M. Aortic dissection: percutaneous management of ischaemic complications with endovascular stents and balloon fenestration. *J Vasc Surg* 1996;23:241–253.
- [9] Gysi J, Schaffner T, Mohasci P, Aeschbacher B, Althaus U, Carrel T. Early and late outcome of operated and non-operated acute dissection of the descending aorta. *Eur J Cardiothorac Surg* 1997;11:1163–1170.
- [10] Carrel T, Nguyen T, Gysi J, Kipfer B, Sigurdsson G, Schafner T, Schüpbach P, Althaus U. Akute Aortendissektion Typ B: prognose nach initial konservativer Behandlung und prädiktive Faktoren für einen komplizierten Verlauf. *Schweiz Med Wochenschr* 1997;127:1467–1473.
- [11] Juvonen T, Ergin MA, Galla JD, Lansman SL, Nguyen KH, McCullough JN, Levy D, de Asla RA, Bodian CA, Griep RB. Prospective study of the natural history of thoracic aneurysm. *Ann Thorac Surg* 1997;60:1533–1545.
- [12] Juvonen T, Ergin MA, Galla JD, Lansman SL, McCullough JN, Nguyen K, Bodian CA, Ehrlich MP, Spielvogel D, Klein JJ, Griep RB. Risk factors for rupture of chronic type B dissections. *J Thorac Cardiovasc Surg* 1999;117:776–786.
- [13] Tiesenhausen K, Amann W, Koch G, Hausegger KA, Oberwalder P, Rigler B. Endovascular stent-graft repair of acute thoracic aortic dissection—early clinical experiences. *Thorac Cardiovasc Surg* 2001;49:16–20.
- [14] Bortone AS, Schema S, Mannatrizio G, Paradiso V, Ferlan G, Dialetto G, Cotrufo M, de Luca Tupputi Schinosa L. Endovascular stent-graft treatment for sieases of the descending thoracic aorta. *Eur J Cardiothorac Surg* 2001;20:514–519.

## Appendix A. Conference discussion

**Dr D. Dougenis (Patras, Greece):** I would like you to elaborate a bit on the role of the increased diameter of 4.5 in Type B dissection. Was that an independent parameter in your multivariate analysis? Would you operate on

Type B dissection based only on the fact that the descending thoracic aorta is more than 4.5 cm in diameter?

**Dr Genoni:** I think our problem is that the patients with acute Type B aortic dissection are not in a surgical ICU in their initial hospitalisation, they are in a medical intensive care unit, and so we must ask our colleagues to call us for a decision of the therapeutic strategies. When the diameter is enlarged and the patient is stable, we do not operate on the patient in the very acute phase, but I think we have to treat him in the initial hospitalisation. We saw in our follow-up that the patients who need surgery in the follow-up time for a large aortic diameter are patients who have an enlarged aortic diameter at hospitalisation.

**Dr E. Baudet (Bordeaux, France):** Do you think that the introduction of a stent graft could lead to reconsider the management of this Type B dissection for an early aggressive approach?

**Dr Genoni:** It depends on the symptoms of the patients. If the patients have a malperfusion, I think the surgical treatment is not necessary. Then we have the interventional treatment with stents, with fenestration. But in patients with an enlarged aortic diameter, then the results in our hands of stents are not very good. So we do make surgery in these cases.

**Dr Baudet:** Even if this minimal management is performed very early at the time when the aorta is not too much enlarged?

**Dr Genoni:** It is very difficult because we have two problems. The first is we have not only one entry, and we cannot see where the entry is, and the second problem we have is the peripheral malperfusion after this technique.

**Dr A. Haverich (Hannover, Germany):** The primary referral unit is probably cardiology or internal medicine. Do they refuse patients from being admitted if there is a call from the outside hospital and there is a clear diagnosis of acute Type B dissection? This is important because of the epidemiology of the disease. In our unit, we would not accept patients without enlargement, without complications. We would let them be treated outside their own unit.

**Dr Genoni:** No. They accept the patients, make all initial diagnoses, and then we want to discuss together the therapeutic strategy. After that, the patients go back in the referring hospital.

**Dr Haverich:** A short question regarding your statistics. There were 65% treated medically, 32% emergency operation and 9% were urgent. That makes more than 100. Was there a crossover between the medical and the surgical?

**Dr Genoni:** Yes. In the follow-up time we have not a crossover, but in the initial are only patients in the first phase, and when one patient has been operated, then he is in the surgical group.