

## Maximum Aortic Diameter as a Simple Predictor of Acute Type B Aortic Dissection

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**Objectives:** To identify the most prognostic predictor of Stanford type B aortic dissection at admission.

**Patients and Methods:** Forty-three patients with Stanford type B aortic dissection were divided into two groups: (1) those who developed dissection-related events later (EV group: n = 18), including the need for surgery (n = 12), rupture (n = 1), dissection-related death (n = 5), and aortic enlargement  $\geq 5$  mm in diameter per year (n = 15); (2) those without later events (NoEV group: n = 25). Clinical features, aortic diameters, and blood flow status were compared.

**Results:** The maximum aortic diameter at admission was  $41.5 \pm 1.7$  mm for the EV group, which was significantly greater than the NoEV group ( $34.4 \pm 0.9$  mm,  $p < 0.001$ ). A maximum aortic diameter  $\geq 40$  mm was found in 11 patients (61%) of the EV group, whereas this maximum was found in 4 (16%) of the NoEV group ( $p = 0.004$ ). A patent false lumen at admission was found in all patients of the EV group and in 17 (68%) of the NoEV group ( $p = 0.013$ ). Other factors were not significant. A Cox hazard analysis indicated a maximum aortic diameter  $\geq 40$  mm as a significant predictor for dissection-related events (hazard ratio 3.13,  $p = 0.032$ ). The presence of a patent false lumen did not reach a statistical significance. **Conclusion:** Our results indicated that a maximum aortic diameter  $\geq 40$  mm at admission was the most prognostic factor for developing late dissection-related events, rather than the presence of a patent false lumen. (*Ann Thorac Cardiovasc Surg* 2008; 14: 303–310)

**Key words:** aortic dissection, false lumen, Stanford type B, medical treatment

### Introduction

Conservative medical treatment is generally recommended for patients with Stanford type B aortic

dissection, unless the patients present with dissection-related complications such as rupture, aortic enlargement, visceral ischemia, or lower limb ischemia at the time of onset.<sup>1–3</sup> The preference of conservative medical treatment is based on the favorable prognosis in the acute period in those with type B dissection, many of whom, however, may suffer those complications during the chronic period.<sup>3–5</sup> In fact, the reported 1-year and 5-year survival rates of medically treated patients are almost equivalent to those of surgically treated patients, but the 10-year survival rates of medically treated patients is lower than that of surgically treated patients.<sup>2,3,6</sup> Treatment results will be improved if we can identify those who will develop dissection-related complications and require surgical treatment soon after

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Received June 7, 2007; accepted for publication September 18, 2007

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**Table 1. Clinical characteristics of 43 patients investigated in this study**

Clinical profile	No.	(%)
Male/female	29/14	
Age (mean)	69.2 ± 1.8	
DeBakey's classification		
Type IIIa	12	(27.9)
Type IIIb	31	(72.1)
Preexistent risk factors		
Hypertension	24	(55.8)
Cerebrovascular disorders	7	(16.3)
Angina pectoris	6	(14.0)
Diabetes	3	( 7.0)
Hyperlipidemia	2	( 4.7)
Time from the onset of symptoms to admission	0.7 ± 0.2	(days)
Maximum aortic diameter at admission	37.3 ± 1.0	(mm)
The mean follow-up period	50.9 ± 5.3	(months)

the onset of dissection. The reported predictors of complications associated with aortic dissection include the presence of a patent false lumen, a maximum aortic diameter greater than 40 or 45 mm upon admission, or location of the most dilated aortic segment at the distal arch.<sup>7-10)</sup> The purpose of this study was to examine whether those reported predictors prognosticated the outcome of our cases during the past decade and to identify the most prognostic predictor at admission in patients with type B aortic dissection.

## Patients and Methods

This retrospective study was performed on 43 patients with Stanford type B aortic dissection who were indicated for medical treatment at admission from January 1997 to December 2006 in Division of Cardiovascular Surgery, Aishin Memorial Hospital. All patients were Japanese and consisted of 29 males and 14 females with ages ranging from 40 to 91 (mean 69.2 ± 1.8) years old. Clinical characteristics of these patients are shown in Table 1. The diagnosis was established by the findings of enhanced computed tomography (CT) in all cases. Patients suspected of having aortic dissection subsequently also received digital subtraction angiography (DSA). Aortic dissection was confined to the thoracic descending aorta (DeBakey's type IIIa) in 12 patients and the descending thoracic and abdominal aorta (DeBakey's type IIIb) in 31. The determinations as to whether the false lumen was thrombosed or patent were performed based on the findings of CT and DSA. When the false lumen was completely thrombosed and exhibited no enhancement, it was judged to be thrombosed.

When blood flow was seen in the false lumen that communicated with the true lumen, even in the case of a partially thrombosed lumen, the false lumen was judged to be patent. The largest diameter of the dissecting aorta measured on CT images was defined as the maximum aortic diameter (mm). In this study, acute phase describes the period within 14 days of the onset of symptoms suspected of aortic dissection. Chronic phase describes the period at least 14 days after the onset of these symptoms. The intervals between the onset of symptoms and admission ranged from 0 to 7 (mean 0.7 ± 0.2) days, thus all patients belonged to the case of "acute phase" dissection.

All patients were initially treated with antihypertensive medications unless surgical treatment was soon determined according to the following indications: (1) consistent enlargement of the false channels, (2) aortic rupture, (3) uncontrollable pain, (4) visceral ischemia or lower limb ischemia resulting from aortic dissections. Patients presenting with retrograde dissection extended to the proximal aortic arch or to the ascending aorta at admission were not included in this study. No patients with Marfan syndrome were in this study, and the etiologic source of aortic dissection was considered to be atherosclerosis in all patients. Preexistent risk factors found in the 43 patients included hypertension in 24 (56%), ischemic heart disease in 6 (14%), hyperlipidemia in 2 (5%), diabetes in 3 (7%), a history of cerebral infarction in 7 (16%), and renal failure requiring hemodialysis in 2 (5%) cases. The maximum aortic diameter at admission was 37.3 ± 1.0 (mean ± standard error of mean [SEM]) mm.

The 43 patients were divided into the following two

groups: (1) those who developed dissection-related events after admission or during the follow-up period (EV group:  $n = 18$ ). Dissection-related events were defined as the need for aortic surgery, aortic rupture, dissection-related death, and mean aortic enlargement greater than 5 mm in diameter per year. (2) This group comprised those who developed no events later (NoEV group:  $n = 25$ ). Death from unknown causes was included in the dissection-related deaths. The mean aortic enlargement rate (mm per year) was calculated by dividing the difference in maximum aortic diameters by the intervals (years) between any times during the follow-up phase. Clinical features, preexistent risk factors, aortic diameter, and blood flow status in the false lumen were compared between the two groups.

#### Antihypertensive treatment

Patients with acute type B aortic dissection were admitted to a critical care unit, where blood pressure, electrocardiogram, and percutaneous oxygen saturation were closely monitored. Patients received nicardipine hydrochloride by continuous intravenous infusion immediately after admission, and in some cases with additional continuous infusion of nitroglycerin. Blood pressure was controlled with a target systolic pressure of 120 mmHg unless urine production was decreased. Laboratory tests, including serum creatinine and blood urea nitrogen (BUN), were frequently assessed during the period of a week after admission. When patients exhibited a stable condition at least for a week after a target blood pressure was obtained, they were switched to oral administrations. In principle, patients received oral  $\beta$ -blockers concurrently with additional angiotensin-converting enzyme inhibitors as appropriate unless they presented with bradycardia, obstructive pulmonary disease, or severe heart failure. Oral calcium-channel blockers were selected instead of  $\beta$ -blockers in some cases. Surgical treatment was performed when patients developed any indications for its need as described above.

After discharge, patients continued to take oral medications at our hospital. The control of blood pressure during the follow-up period was assessed by blood pressure measurements at outpatient clinics at intervals of 2 to 4 weeks. Patients who were referred to other hospitals or lost to follow-up within 1 year after discharge were not included in this study. Blood pressure at outpatient clinics was controlled with a target systolic pressure of less than 140 mmHg and diastolic pressure of less than 90 mmHg. The control of

blood pressure was judged to be favorable when it was controlled within a target pressure. Otherwise, control was judged to be poor, and the patient exhibiting poor control pressure was asked to visit the outpatient clinic at 1-week intervals. The mean follow-up period after admission was  $50.9 \pm 5.3$  months.

#### Statistical analysis

Clinical features such as age and gender, site of entry, preexistent risk factors, aortic diameters, and blood flow status in the false lumen were compared between the two groups. Continuous variables are presented as the mean  $\pm$  standard error (SE) and compared using the unpaired Student's  $t$ -test. A Chi-square test was employed for comparisons of nominal variables. The event-free rate was analyzed by the Kaplan-Meier method, and comparisons between the two groups were performed by the log-rank test and generalized Wilcoxon's test. A Cox proportional hazard model was applied for all patients to determine the effects of variables on dissection-related events.

Differences were statistically considered significant at  $p < 0.05$ . SPSS (Ver.12.0J) software (SPSS Inc., Chicago, IL) was used for all statistical analyses.

#### Results

Of 43 patients with Stanford type B aortic dissection who were admitted to our hospital, 3 underwent acute phase surgery performed within 14 days of the onset. Of the 40 remaining, 9 required chronic phase surgery and 31 continued to receive medical treatment without surgery during the chronic phase. Thus 12 of 43 patients (27.9%) required acute or chronic phase surgery.

The indication for acute phase surgery was lower limb ischemia resulting from the advancement of dissection in all 3 patients. They underwent fenestration of the aorta on days 3, 4, and 8 after the onset, respectively. None died during his or her first hospitalization, but 2 died later, 1 of pneumonia (11 months after onset) and the other of hepatic cholangioma (23 months after onset).

Of the 9 patients receiving chronic phase surgery, indication for surgery was an increased aortic diameter in 5, development of the retrograde aortic dissection extending to the ascending aorta in 2, recurrence of dissection in 1, and visceral and lower limb ischemia in 1. There were no operative deaths in the 5 patients within 30 days after their surgery, but 3 died later. Two

**Table 2. Comparative studies between the EV group and the NoEV group**

Group	EV group: (n = 18)	NoEV group (n = 25)	P value
Age	71.2 ± 2.6	67.7 ± 2.4	0.325
Male/Female	15/3	14/11	0.099
DeBakey's type of dissection			
DeBakey's IIIa/IIIb	5/13	7/18	0.999
Maximum aortic diameter	41.5 ± 1.7 mm	34.4 ± 0.9 mm	0.0003
Maximum aortic diameter greater than 40 mm			
yes/no	11/7	4/21	0.0035
Blood flow status in the false lumen (thrombosed/patent)	0/18	8/17	0.0132
The site of entry (distal arch/descending aorta)	14/4	17/8	0.732
The site of reentry (descending aorta/abdominal aorta)	8/10	9/16	0.753
Atherosclerotic risk factors			
Cerebrovascular disorders	3 (16.7%)	4 (16.0%)	0.999
Angina pectoris	1 ( 5.6%)	5 (20.0%)	0.375
Hypertension	9 (50.0%)	15 (60.0%)	0.550
Diabetes	2 (11.1%)	1 ( 4.0%)	0.562
Hyperlipidemia	1 ( 5.6%)	1 ( 4.0%)	0.999

EV group, patients who developed dissection-related events during and after the acute or chronic phase; NoEV group, patients who did not develop dissection-related events throughout the study period.

were patients who underwent graft replacement of the descending aorta: 1 of pneumonia (67 months after surgery) and the other of sudden death (77 months after surgery). The third patient, who underwent endovascular stent graft surgery, died of leakage from the stent graft 25 months after the first surgery.

Among the 31 patients who continued to receive medical treatment throughout the acute and chronic phases, 25 (80.6%) survived and 6 died during the chronic phase. Of those who died, 2 experienced dissection-related deaths, including rupture in 1 and sudden death in another. Of the 4 others, 2 died of pneumonia, 1 of malignancy, and 1 of severe burns.

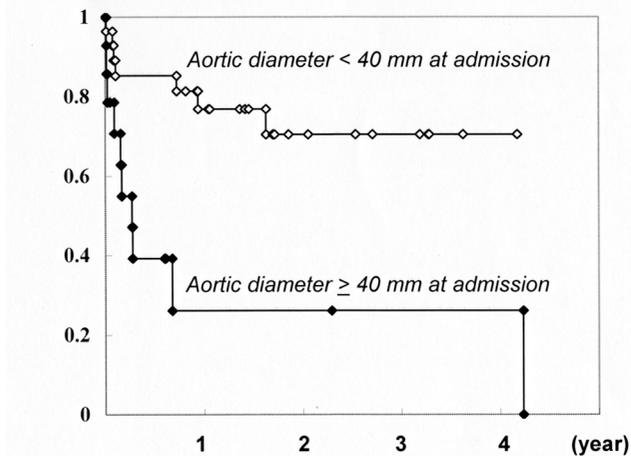
With regard to dissection-related events among 43 patients investigated in this study, a total of 18 patients (41.9%) developed following 33 events: the need for aortic surgery in 12, aortic rupture in 1, dissection-related deaths in 5, and mean aortic diameter greater than 5 mm per year in 15 patients. Of 31 patients who did not undergo surgery but continued to receive medical treatment, 6 patients (19.4%) experienced events. Judgment of blood pressure control during the outpatient clinic became favorable within 3 months of discharge in all except 2 patients with orthostatic hypotension (1 in the EV group and 1 in the NoEV group).

#### Univariate analysis of predictors: EV group vs. NoEV group

Univariate analysis was applied for a comparison of following variables between the EV group and the NoEV group: age, gender, DeBakey's type of aortic dissection, maximum aortic diameter at admission, blood flow status in the false lumen (thrombosed or patent), the site of entry, and atherosclerotic risk factors (Table 2). Among these items, significant differences between the two groups were found with regard to the maximum aortic diameter at admission and blood flow status in the false lumen. The maximum aortic diameter at admission was 41.5 ± 1.7 mm for the EV group, which was significantly greater than the NoEV group (34.4 ± 0.9 mm,  $p = 0.0003$ ). When a cutoff value of the maximum aortic diameter at admission was set at 40 mm according to previous reports,<sup>4,9,10</sup> the EV group included a significantly larger number of patients with a maximum aortic diameter greater than 40 mm (11 of 18 patients, 61.1%) compared with the number of those patients in the NoEV group (4 of 25, 16.0%) ( $p = 0.004$ ). A patent false lumen at admission was found in all patients (100%) of the EV group and 17 of 25 (68.0%) of the NoEV group ( $p = 0.013$ ). Four patients presenting with a maximum aortic diameter greater than 40 mm in the NoEV group

**Table 3. Results of Cox proportional hazard analysis**

Investigated factors	Hazard ratio (95% confidence intervals)	P value
Aortic diameter $\geq 40$ mm	3.13 (1.10–8.88)	0.032
Patent false lumen	3.59 (0.46–27.84)	0.22
Male patient	2.07 (0.57–7.54)	0.27

**Event Free Rate****Fig. 1.** Kaplan-Meier curves indicating freedom from dissection-related events in the group of patients having a maximum aortic diameter at admission of  $\geq 40$  mm and those who did not.

included those without a patent false lumen ( $n = 2$ ) and those who died of pneumonia and malignancy ( $n = 2$ ). Other factors such as age ( $p = 0.099$ ), site of entry ( $p = 0.732$ ), and atherosclerotic risk factors ( $p = 0.375$  to  $0.999$ ) were not significant between the two groups. Among patients of the EV group, dissection-related events occurred as surgery ( $n = 12$ ), rupture ( $n = 1$ ), dissection-related death ( $n = 5$ ), and enlargement of the aortic diameter ( $n = 15$ ), as described above.

**Analysis of Cox proportional hazard model and event-free curves**

Of the predictors examined in the univariate analysis, those with a  $p$  value of less than 0.1 were assessed using the Cox proportional hazard model to identify correlations with event occurrences. The items assessed included a maximum aortic diameter greater than 40 mm, the presence of a patent false lumen, and a male patient as a gender risk. The results are shown in Table 3, which indicates that the event occurrence rate was significantly correlated with a maximum aortic diameter greater than 40 mm at admission (hazard ratio 3.13,  $p = 0.032$ ). The presence of a patent false lumen and male patient as a gender risk did not reach a statistical signifi-

icance ( $p = 0.22, 0.27$ , respectively).

Figure 1 shows a comparison of Kaplan-Meier curves indicating the rate of freedom from events between patients having a maximum aortic diameter greater than 40 mm at admission and those who did not. The end point of each patient for calculating these curves is the day of dissection-related events occurring in the EV group and the last follow-up day in the NoEV group. The event-free rate of patients with a maximum aortic diameter greater than 40 mm at admission was significantly lower than that of other patients, both by log rank test ( $p = 0.006$ ) and generalized Wilcoxon's test ( $p = 0.006$ ). These results also show that patients with a maximum aortic diameter greater than 40 mm at admission had significantly more chance to develop dissection-related events than those with a maximum aortic diameter of less than 40 mm.

**Discussion**

Thoracic aortic dissection is an extremely serious vascular accident that requires immediate treatment. The treatment for Stanford type B aortic dissection at onset has been a conservative antihypertensive therapy unless

the patient presents with symptoms indicative of surgery.<sup>3-5)</sup> Although treatment results of these patients during the acute period are acceptable, the long-term prognosis has been reported to be unsatisfactory because of the development of an enlargement of the aorta, rupture, and recurrence of dissection, especially in those with a patent false lumen.<sup>2,3,6)</sup> In previous studies on the prognosis of medically treated type B dissection, the persistence of a patent false lumen has been shown to be a strong predictor for the development of dissection-related events or enlargement of the aortic diameter in the later chronic period.<sup>8,9,11,12)</sup> However, Juvonen et al. reported that the presence of a persistently patent false lumen did not increase the risk of rupture.<sup>5)</sup> Some patients with a thrombosed false lumen may develop an ulcerlike projection that predisposes to subsequent expansion of the aorta. Thus the presence of a patent false lumen is not a single predictor of the outcome of Stanford type B aortic dissection.

Our results indicated that a maximum aortic diameter greater than 40 mm at admission was the most prognostic factor for developing late dissection-related events, rather than a presence of a patent false lumen. The existence of a maximum aortic diameter greater than 40 mm has been reported to be a significant predictor as well as a patent false lumen in previous studies.<sup>9-11,13)</sup> Likewise, a cutoff value of the maximum aortic diameter at admission was set at 40 mm in this study, based on the mean aortic diameter in this series and based on those reports.<sup>9,10)</sup> Only 5 patients had a maximum aortic diameter greater than 45 mm, thus a cutoff value of 45 mm was supposed to be less beneficial than that of 40 mm because of the lower sensitivity as a predictor. In the treatment of thoracic aortic disease, the size of aneurysm was reported to naturally have a profound impact on rupture and death.<sup>14)</sup> In previous studies on the prognosis of patients with thrombosed false lumen, older age and appearance of an ulcerlike projection have been reported to be predictive for the progression of the disease.<sup>15)</sup> Furthermore, the disappearance of the thrombosed lumen and a maximal aortic diameter of less than 45 mm were also reported to be predictive for a good prognosis.<sup>7)</sup> It seems quite likely that a maximum aortic diameter at onset can be a common predictor of patients with thrombosed or patent false lumen.

Although a maximum aortic diameter greater than 40 mm at admission was the most prognostic factor assessed by our Cox proportional hazard model; the

presence of a patent false lumen was also found more often in the EV group in univariate analysis. Further study with a larger number of patients is required to determine whether a maximum aortic diameter is really more predictive of prognosis than the presence of a patent false lumen. Determinations as to whether the false lumen was thrombosed or patent were conducted based on our criteria, described previously, but the number of patients with a thrombosed false lumen was relatively smaller than in previous studies. Moreover, this study mainly examined the findings of CT and DSA at admission on the outcome of patients. If the study subjects were limited to those who survived the acute phase without surgery, a patent false lumen could be more predictive of dissection-related events as well as previous studies. In another study the location of the most dilated aortic segment at the distal arch was also reported to be a significant predictor of the prognosis.<sup>8)</sup> This factor was not identified as such in our study.

What is an optimal therapeutic strategy for asymptomatic type B dissection during a chronic period? Elefteriades recommend intervention for the descending aorta at 6.5 cm in the case of atherosclerotic aortic aneurysm and at 6.0 cm for Marfan's disease, based on their risk/benefit analysis of the accumulated data.<sup>16)</sup> The dissected aortic wall is mechanically weaker than the wall of ordinary aneurysms; thus earlier intervention would be necessary for patients with chronic aortic dissection to prevent rupture and recurrence of dissection. Marui et al. suggested that patients with type B acute aortic dissection exhibiting a maximum aortic diameter greater than 40 mm and a patent false lumen should undergo surgery earlier during the chronic phase.<sup>11)</sup> They also advise continuing antihypertensive therapy for patients with a maximum aortic diameter of less than 40 mm and a closed false lumen. Onitsuka et al. also described that it was important to pay careful attention to the predictors of a patent false lumen and a maximum aortic diameter greater than 40 mm at onset to improve the long-term outcome.<sup>9)</sup> Thus it is an acceptable strategy that surgical treatment should be considered relatively early during the chronic phase in patients with both a maximum aortic diameter greater than 40 mm and a patent false lumen. If patients have either of the two predictors, we should probably pay more attention to those with a maximum aortic diameter greater than 40 mm based on our results. Currently, surgical indication for chronic type B aortic dissection is considered for those with a

maximum aortic diameter greater than 50 mm.<sup>1,16)</sup> When the maximum aortic diameter exceeds 40 mm during the follow-up period, a CT examination should be taken more frequently than before to find a good chance to perform surgical treatment before rupture, regardless of the presence of a patent false lumen.

Therapeutic strategy for type B aortic dissection will be remarkably changed by forthcoming progress in treatments with endovascular stent graft. This is a promising and less invasive treatment for selected patients with acute aortic dissection.<sup>17-19)</sup> However, most of the reported stent grafts were handmade instruments and have not been available as a commercially produced, common endovascular stent graft. Furthermore, devices for inserting these stent grafts are not supported by national medical insurance in Japan. The indication of stent graft procedure may be an alternative to surgery in some institutes, but limited to patients who cannot be treated surgically because of worse preoperative conditions in other institutes.<sup>19,20)</sup> Moreover, long-term results of endovascular treatment have not been reported until recently; thus its value in the treatment of aortic dissection has not been well established.<sup>20,21)</sup> Therapeutic strategy for each case of Stanford type B aortic dissection should be determined based on the maximum aortic diameter, blood flow status in the false lumen, or other predictors reported previously until the stent graft procedure becomes an established standard method in the treatment of Stanford type B aortic dissection.

There are some limitations in this study. In its design, patients with Stanford type B aortic dissection were retrospectively divided into the two groups based on the patient outcome as to whether they later had dissection-related events or not as a single center experience. Ideally, a randomized prospective study is required to determine the value of predictor with a larger number of patients. Moreover, the follow-up period is relatively short because patients investigated in this study are those treated during the past 10 years. The short follow-up period may have an influence on the incidence of dissection-related events. As is previously described, the few patients with a thrombosed false lumen may be another problem in the statistical analysis in this study. Although there are many limitations, our study results are not so inconsistent with previous reports and may help to prognosticate the outcome at least in part of the patients with Stanford type B aortic dissection.

In conclusion, our study on the outcome of Stanford type B aortic dissection indicated that a maximum aortic diameter greater than 40 mm at admission was the most prognostic factor for developing late dissection-related events. Although some limitations are present in this study, these results may be beneficial in the assessment of patients with Stanford type B aortic dissection soon after the onset.

## References

1. Crawford ES, Svensson LG, Coselli JS, Safi HJ, Hess KR. Aortic dissection and dissecting aortic aneurysms. *Ann Surg* 1988; **208**: 254-73.
2. Glower DD, Fann JI, Speier RH, Morrison L, White WD, et al. Comparison of medical and surgical therapy for uncomplicated descending aortic dissection. *Circulation* 1990; **82** (5 Suppl): IV39-46.
3. Masuda Y, Yamada Z, Morooka N, Watanabe S, Inagaki Y. Prognosis of patients with medically treated aortic dissections. *Circulation* 1991; **84** (5 Suppl): III7-13.
4. Iguchi A, Tabayashi K. Outcome of medically treated Stanford type B aortic dissection. *Jpn Circ J* 1998; **62**: 102-5.
5. Juvonen T, Ergin MA, Galla JD, Lansman SL, McCullough JN, et al. Risk factors for rupture of chronic type B dissections. *J Thorac Cardiovasc Surg* 1999; **117**: 776-86.
6. Schor JS, Yerlioglu ME, Galla JD, Lansman SL, Ergin MA, et al. Selective management of acute type B aortic dissection: long-term follow-up. *Ann Thorac Surg* 1996; **61**: 1339-41.
7. Nishigami K, Tsuchiya T, Shono H, Horibata Y, Honda T. Disappearance of aortic intramural hematoma and its significance to the prognosis. *Circulation* 2000; **102** (19 Suppl 3): III243-7.
8. Akutsu K, Nejima J, Kiuchi K, Sasaki K, Ochi M, et al. Effects of the patent false lumen on the long-term outcome of type B acute aortic dissection. *Eur J Cardiothorac Surg* 2004; **26**: 359-66.
9. Onitsuka S, Akashi H, Tayama K, Okazaki T, Ishihara K, et al. Long-term outcome and prognostic predictors of medically treated acute type B aortic dissections. *Ann Thorac Surg* 2004; **78**: 1268-73.
10. Kozai Y, Watanabe S, Yonezawa M, Itani Y, Inoue T, et al. Long-term prognosis of acute aortic dissection with medical treatment: a survey of 263 unoperated patients. *Jpn Circ J* 2001; **65**: 359-63.
11. Marui A, Mochizuki T, Mitsui N, Koyama T, Kimura F, et al. Toward the best treatment for uncomplicated patients with type B acute aortic dissection: A consideration for sound surgical indication. *Circulation* 1999; **100** (19 Suppl): II275-80.
12. Sueyoshi E, Matsuoka Y, Imada T, Okimoto T, Sakamoto I, et al. New development of an ulcerlike projection in aortic intramural hematoma: CT evaluation. *Radi-*

- ology 2002; **224**: 536–41.
13. Sueyoshi E, Imada T, Sakamoto I, Matsuoka Y, Hayashi K. Analysis of predictive factors for progression of type B aortic intramural hematoma with computed tomography. *J Vasc Surg* 2002; **35**: 1179–83.
  14. Davies RR, Goldstein LJ, Coady MA, Tittle SL, Rizzo JA, et al. Yearly rupture or dissection rates for thoracic aortic aneurysms: simple prediction based on size. *Ann Thorac Surg* 2002; **73**: 17–27.
  15. Kaji S, Akasaka T, Katayama M, Yamamuro A, Yamabe K, et al. Long-term prognosis of patients with type B aortic intramural hematoma. *Circulation* 2003; **108** (Suppl 1): II307–11.
  16. Elefteriades JA. Natural history of thoracic aortic aneurysms: indications for surgery, and surgical versus nonsurgical risks. *Ann Thorac Surg* 2002; **74**: S1877–80.
  17. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med* 1999; **340**: 1546–52.
  18. Kusagawa H, Shimono T, Ishida M, Suzuki T, Yasuda F, et al. Changes in false lumen after transluminal stent-graft placement in aortic dissections: six years' experience. *Circulation* 2005; **111**: 2951–7.
  19. Dialetto G, Covino FE, Scognamiglio G, Manduca S, Della Corte A, et al. Treatment of type B aortic dissection: endoluminal repair or conventional medical therapy? *Eur J Cardiothorac Surg* 2005; **27**: 826–30.
  20. Leurs LJ, Bell R, Degrieck Y, Thomas S, Hobo R, et al. Endovascular treatment of thoracic aortic diseases: combined experience from the EUROSTAR and United Kingdom Thoracic Endograft registries. *J Vasc Surg* 2004; **40**: 670–9.
  21. Nienaber CA, Zannetti S, Barbieri B, Kische S, Schareck W, et al. INvestigation of STent grafts in patients with type B Aortic Dissection: design of the INSTEAD trial—a prospective, multicenter, European randomized trial. *Am Heart J* 2005; **149**: 592–9.