

# Follow-up Study after Intracranial Percutaneous Transluminal Cerebral Balloon Angioplasty

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## Summary

To find the angiographic lesions specific characteristics appropriate for intracranial percutaneous transluminal cerebral angioplasty (PTCBA).

Forty-two clinically symptomatic patients with 42 haemodynamically significant intracranial lesions (% diameter stenosis >70) were treated by PTCBA between January 1992 and May 1996. Before the angioplasty treatment, the patients were classified into three groups according to the angiographic lesions' characteristics summarised as follows: type A, a short and concentric stenosis; type B, a tubular lesion, or an extreme eccentric lesion; and type C, a diffuse lesion. They were followed after PTCBA from one month to six years to compare between the three groups. Primary end points were death, stroke, or bypass surgery.

The clinical success rates in type A, B and C groups were 92%, 86% and 33% ( $p=0.0032$ ), respectively. Cumulative risks of fatal or nonfatal ischaemic stroke / ipsilateral bypass surgery in type A, B and C groups were 8%, 26% and 87% ( $p<0.0001$ ), respectively. The cumulative risk of 8% in type A group patients appeared to be smaller than in historical studies.

PTCBA for intracranial simple (type A) lesions produces a favourable clinical outcome for symptomatic patients.

## Introduction

Pharmacological treatments of ischaemic stroke are in progress<sup>1</sup>. However, these treatments are sometimes ineffective in patients suffering from recurrent or crescendo neurological symptoms from a high grade stenosis or occlusion of the intracranial arteries and it has not been proven whether or not medical therapy can effectively prevent future strokes in patients who have experienced a transient neurological deficit or minor stroke arising from an intracranial lesion. This is of a major concern as arterial stenotic lesions seem to occur more frequently, intracranially, in Japanese people<sup>2</sup>.

Revascularisation to the coronary artery by bypass surgery has been shown to be effective in reducing symptoms and improving clinical outcome of patients with stable angina pectoris<sup>3</sup>. In contrast, extracranial-intracranial (EC/IC) arterial bypass surgery has not yet been proven to be effective<sup>4</sup> and a high complication rate can be anticipated particularly for the posterior cerebral circulation<sup>5</sup>. Percutaneous transluminal angioplasty (PTA) has not been until the past few years that percutaneous transluminal cerebral balloon angioplasty (PTCBA) of the intracranial artery has been reported<sup>6-8</sup>.

In general, transluminal dilatation of small and fragile intracranial arteries appears to be more dangerous than that of the extracranial,

coronary or other peripheral arteries. A previous study has reported that a short, concentric or mild eccentric, and not angulated intracranial arterial stenosis may be appropriate to initial or repeated PTCBA<sup>9</sup>. In the present study, therefore, the purpose was to assess the safety and efficacy of initial and elective cerebral angioplasty, to investigate the clinical outcome, and finally to determine the subgroup appropriate to PTCBA amongst three groups classified by angiographic lesions' characteristics prior to PTCBA.

### Subject and Methods

Clinically symptomatic patients underwent PTCBA and their prognosis was followed thereafter from one month to six years. All the patients gave informed written consent for PTCBA and treatments during follow-up. Inclusion criteria for the study were 1) haemodynamically significant stenosis (>70%) of one of the following major intracranial arteries: the distal internal carotid artery (DICA), the middle cerebral artery (MCA), the distal vertebral artery (DVA), the basilar artery (BA) and the posterior cerebral artery (PCA); 2) recurrent or crescendo transient ischaemic attacks (TIAs) unresponsive to maximal medical therapy, or a minor stroke with persistence of symptoms or signs in the distribution of the stenotic artery; and 3) patients within 6 months from the last attack.

Exclusion criteria were as follows: 1) patients with prior intracranial PTCBA; 2) patients in the acute stroke stage; 3) patients with a severe neurological deficit from a major stroke; 4) patients presenting total occlusion older than 6 months; 5) patients with chronic total occlusion that were longer than 10 mm in length, and 6) patients presenting chronic total occlusion without visible distal segment on angiograms.

A total of 61 consecutive PTCBAs for the intracranial artery were performed in 61 patients between January 1992 and May 1996 in the authors' institute. PTCBA was not attempted in patients with exclusion criteria of 3 to 6. Twelve patients with prior intracranial PTCBA and 7 patients with PTCBA in the acute stroke stage were excluded from further analysis. Forty-two patients (33 men and 9 women) aged 4 to 76 years were therefore analysed. The mean age of the patients was 59 years ( $\pm 13$ ). Lesions in-

involved the DICA in 8 patients, the MCA in 21 patients, the DVA in 6 patients, the BA in 5 patients and the PCA in 2 patients. Twelve patients (7MCAs, 1DVA and 4BAs) exhibited recurrent or crescendo transient ischaemic attacks (TIAs) and the other 30 patients had a nondisabling stroke with persistence of symptoms or signs.

Before PTCBA, the patients received a complete neurological examination, routine laboratory tests, head computed tomography (CT) scanning or magnetic resonance (MR) imaging to examine the cerebral lesions. Although two patients, a 4-year-old boy and a 26-year-old man, had an unknown aetiology of a stenosis<sup>10</sup>, the other 40 patients, older than 40 years, had atherosclerotic stenosis. Among 29 patients with lesions in the anterior cerebral circulation, 22 patients without crescendo TIAs underwent brain single photon emission CT (SPECT) scanning before and after acetazolamide challenge<sup>11,14</sup> which demonstrated low perfusion or impaired vasodilatory capacity in the affected territory prior to PTCBA. Because emergency SPECT scanning has not been performed in the authors' institute, patients suffering from impending stroke did not undergo SPECT scanning due to time limitations.

Traditional arteriosclerotic risk factors were assessed on admission and treated with medications when considered clinically appropriate. All patients, except the boy, received 81~162 mg of acetylsalicylic acid (aspirin: ASA) per day approximately one week before and after PTCBA.

Stenosis rate was calculated on angiograms before and immediately after PTCBA, and at the follow-up using the North American Symptomatic Carotid Endarterectomy Trial (NASCET) 15 criteria which compares the diameter at the site of greatest narrowing to the diameter of a normal artery distal to the lesion. Angiographic success of PTCBA was defined as lesions reduced to less than 50% diameter stenosis immediately after dilatation and the clinical success was defined as angiographic success with no major complications such as in-hospital mortality, stroke, or emergency EC/IC bypass surgery. Neurological symptoms during balloon inflation were not regarded as complications.

Anticipation of the likelihood of a successful procedure is required before performing PTCBA. Cerebral angiographic classification de-

scribed elsewhere<sup>9,16</sup> summarised the length and geometry of lesions as follows: *type A*, a short (5 mm or less in length) concentric or moderately eccentric lesions less than totally occlusive; *type B*, a tubular lesion (5-10 mm in length), an extreme eccentric lesion or total occlusion <3 months old; and *type C*, a diffuse lesion (more than 10 mm in length), an extremely angulated (>90°) lesion, a lesion with excessive tortuosity of proximal segment or total occlusion 3 months or older.

Every patient except a 4-year-old boy underwent PTCBA under local anaesthesia via a femoral arterial route. Low molecular weight dextran (LMWD) (1,000-2,000 per day) was started one hour before PTCBA and continued until the next day. Heparin (10,000 units) was intravenously injected to avoid thrombus formation during the procedure and isosorbide dinitrate (2.5 to 5.0 mg) was injected into a target vessel through a guiding catheter to prevent balloon catheter-induced vasospasm. A guide wire was navigated across the stenosis and advanced into the distal segment. The balloon catheter was guided over the wire and then inflated twice to the nominal pressure for 60 seconds (s).

Before PTCBA, patients were neurologically assessed and after PTCBA monitored every month at an outpatient clinic and neurological status examined by the neurosurgeon (KM). Patients with clinically successful dilatation underwent arteriographic follow-up 3 and 12 months later<sup>9,17,18</sup>. The 3-month follow-up studies were defined as those performed between 8 and 16 weeks after PTCBA and the one-year follow-up studies as those performed between 42 and 62 weeks. Angiographic restenosis was defined as 50% or greater diameter stenosis at the follow-up. When symptoms due to restenosis or new lesions occurred or SPECT scans demonstrated impairment of vascular reserve, EC/IC bypass surgery or PTCBA was performed. When patients with restenosis underwent repeat PTCBA, follow-up angiography was carried out after 3 months and one year.

The primary end point was determined by the occurrence of either death, ipsilateral stroke, or ipsilateral bypass surgery. Because repeatability is one of benefits of PTCBA, repeated PTCBA was not included as one of the primary end points. Death during the follow-up was defined to include death from any cause.

Stroke was defined as any neurological deficit including transient symptoms resolving completely within 24 hours. Ipsilateral bypass surgery was defined as EC/IC arterial bypass for the lesion previously treated by PTCBA. Repeated PTCBA was defined as a repeated percutaneous intervention involving the previously treated lesion after the time of the initial procedure. The primary angiographic end point was the minimal luminal diameter at follow-up. Secondary end points included 1) the angiographic success rate; 2) the clinical success rate; and 3) the rate of restenosis.

The main clinical analysis consisted of a comparison between the three groups classified by the angiographic lesions' characteristics with respect to the primary clinical end point. Continuous variables are expressed as mean plus minus 1 SD and were compared by analysis of variance (ANOVA). Categorical data were compared by Fisher's exact test and ordinal scales by Kruskal-Wallis H test. Survival (hazard) probability was calculated by the life table method of Kaplan and Meier and survival curves were compared using the log rank test. Following ANOVA between group comparisons were made using Bonferroni / Dunn method. A probability (p) of less than 0.05 was considered to indicate statistical significance. The SPSS advanced statistics 6.1 program was used for survival (hazard) probability and the log rank test, whilst StatView 4.5 statistics software was used for ANOVA and non-parametric tests.

## Results

According to the angiographic lesions' characteristics prior to PTCBA, patients were classified into three groups before the angioplasty treatment: type A group (n = 12); type B group (n = 21); and type C group (n = 9). There were no differences in base-line characteristics except blood pressure between the three groups. There were many total occlusions in type B and C groups as a matter of course (p=0.017).

The clinical success rates in type A, B and C lesions were 92% (11/12), 86% (18/21) and 33% (3/9), respectively (p=0.0032, K-W H test) and there were significant differences between type A and C (p=0.0021, Bonferroni / Dunn) and between type B and C (p=0.0023, Bonferroni / Dunn). Therefore, successful procedures

are related to the angiographic characteristics of lesions prior to PTCBA. Overall angiographic and clinical success rates were 79 (33/42) and 76 (32/42) percent, respectively. Ten patients were not clinically treated successfully because of two major complications (1 type A and 1 type C), three lesions (2 type Bs and 1 type C) that were difficult to access, failure to open chronic total occlusion (4 type Cs)<sup>16</sup> and a lesion (type B) that was resistant to dilatation. Two strokes as major complications occurred due to dissection by a guide wire in one patient with a petrous carotid stenosis (type A) and abrupt closure of the MCA stenosis (type C) in another patient. The baseline angiogram showed a pre-PTCBA stenosis of  $81 \pm 8\%$  in 42 patients. In 32 patients with clinically successful dilatation, a pre-PTCBA stenosis of  $82 \pm 10\%$  was reduced to  $30 \pm 10\%$  seen on the immediate post-PTCBA angiogram. Consequently, 32 lesions in 32 patients were eligible for the angiographic follow-up.

Among the 12 patients with recurrent or crescendo TIAs prior to PTCBA (4, 5 and 3 patients in type A, B and C groups, respectively), 10 had a clinically successful dilatation and their symptoms disappeared immediately and completely. However, the other 2 patients underwent superficial temporal artery-middle cerebral artery (STA-MCA) bypass surgery following unsuccessful PTCBA. Of the 22 patients who underwent brain SPECT scanning prior to PTCBA, 6 patients were not clinically successfully treated by PTCBA, whilst the other 16 successfully treated patients underwent the examination within 7 days after PTCBA, which demonstrated remarkable improvement of vascular reserve. PTCBA therefore had a good efficacy in improving perfusion to the affected territory.

Clinical follow-up data was available for 39 of the 42 patients, because 2 patients in type B group and one in type C group were censored immediately after unsuccessful PTCBA was followed by medication at the patients' request. Among the 39 patients, 13 patients reached a primary end point within one year. In addition, contralateral ischaemic stroke occurred in one patient and contralateral small putaminal haemorrhage in another patient with a history of hypertension. In type A group ( $n = 12$ ), ipsilateral ischaemic stroke as a major complication occurred in one patient but otherwise

there were no bypass surgeries and no in-hospital mortality during the initial hospitalisation.

In addition, no ischaemic strokes occurred, no bypass surgery was performed and no patient died during the follow-up period. In type B group ( $n = 21$ ), there were no in-hospital mortalities or ischaemic strokes but one patient underwent STA-MCA bypass surgery following unsuccessful PTCBA during the initial hospitalisation, and two patients were removed from follow-up after unsuccessful dilatation. During follow-up, no major stroke but a TIA due to restenosis occurred, two patients underwent STA-MCA bypass surgery and two patients died: one patient died of subarachnoid haemorrhage from a third PTCBA's complication and the other by a mudslide 4 years after successful PTCBA. Bypass surgery during follow-up was performed either for revascularisation of restenosis or for a new ipsilateral lesion in each patient. In type C group ( $n = 9$ ), an ipsilateral ischaemic stroke as major complication occurred and 4 patients underwent STA-MCA bypass surgery following unsuccessful revascularisation but there was no in-hospital mortality during the initial hospitalisation. After censoring one patient, a TIA referable to restenosis occurred and two patients underwent STA-MCA bypass surgery for restenosis but no patient died during follow-up period.

Angiographic restenosis rates in type A, B and C groups were 0% (0/11), 33% (6/18) and 100% (3/3) at one year ( $p=0.0027$ , K-W H test) and there was a significant difference between type A and C ( $p=0.0008$ , Bonferroni / Dunn). Angiographic restenosis rate in patients suffering from crescendo TIAs before PTCBA was only 10% (1/10) at one year, and the rate in patients with a minor stroke prior to PTCBA 36% (8/22) at one year ( $p=0.21$ , Fisher's test). Overall angiographic restenosis rate was 28% (9/32) at one year. Among 9 patients with restenosis at 3 months in type B and C groups, one patient who experienced transient hemiparesis due to restenosis underwent repeated PTCBA at once, whilst the other 8 patients had repeated PTCBA up to 6 months later as SPECT scans showed restenosis to impair vascular reserve despite no symptomatic recurrence. Consequently, seven patients received two PTCBA, one patient in type B group received three PTCBA and one belonging to type C group received four PTCBA. One patient in

type C group, who had undergone a second PTCBA for asymptomatic restenosis at 3 months, experienced a TIA 3 months later and then the patient underwent a third PTCBA.

Cumulative risks of fatal or nonfatal ipsilateral ischaemic stroke in type A, B and C groups were 8%, 12% and 56% at one and two years ( $p=0.1647$ , log rank test) (figure 3). Fatal or nonfatal ipsilateral stroke / ipsilateral bypass surgery risks in type A, B and C groups were 8%, 26% and 87% at one and two years ( $p<0.0001$ , log rank test) (figure 4) and there were significant differences between type A and C ( $p=0.0005$ ) and between type B and C ( $p=0.0001$ ). Fatal or nonfatal ipsilateral stroke / ipsilateral bypass surgery / repeated PTCBA risks in type A, B and C groups were 8%, 42% and 100% at one and two years ( $p<0.0001$ , log rank test) (figure 5) and there were significant differences between type A and C ( $p=0.0001$ ) and between type B and C ( $p<0.0001$ ), although the difference between type A and B was not significant ( $p=0.0657$ ).

Ipsilateral strokes during follow-up included only two TIAs due to restenosis and one SAH as a third PTCBA's complication, which was included as ischaemic stroke in one patient belonging to type B group. No patients exhibited a major ischaemic stroke following the successful PTCBA treatment. Overall cumulative risks of fatal or nonfatal ipsilateral stroke and fatal or nonfatal ipsilateral ischaemic stroke / ipsilateral bypass surgery were 14 and 33 percent at one and two years, respectively.

Among consecutive 54 elective PTCBAs during the study period, a total of 3 major complications occurred, and overall morbidity and mortality rate was 5.6 percent (3/54).

## Discussion

Although a previous study<sup>19</sup> reported that the survival rate of patients with arterial stenosis of 25 percent or more is significantly lower than the expected survival rate in a general population, there are few reports concerning the natural history of patients with a high-grade stenosis (>70%) of the intracranial artery documented by cerebral arteriography. Some previous studies<sup>20-29</sup>, which includes various grades of stenosis (low-grade to high-grade), report the clinical outcome of medically treated patients with intracranial arterial

stenosis, in contrast to the clinical outcome for high-grade stenosis (>70%) only in the present study. Whilst regarding the great difference in the angiographic characteristics between historical controls and the present study, the clinical outcome must be compared.

We defined stroke as any neurological deficit including a TIA and recalculated cumulative stroke risk by Kaplan-Meier method based on data described in some previous studies<sup>21,22,27</sup>. In the historical controls, cumulative stroke risk ranged from 8 to 100% at 2 years and many studies reported a 13% to 22% ischaemic stroke risk at two years. For instance, ipsilateral stroke risks at one, two and three years were approximately 10, 14 and 18 % respectively in medical patients in the EC/IC study<sup>4</sup> where grade of stenosis was not defined. However, Caplan et al<sup>29</sup> reported that 100% of patients suffering from symptoms referable to bilateral intracranial vertebral arterial occlusion died during hospitalisation.

Cumulative risk of fatal or nonfatal ipsilateral stroke and ipsilateral stroke / bypass surgery was only 8 percent at two years in type A group, where 4 patients exhibiting crescendo TIAs were included, and this seemed to be smaller than in the historical controls. Although there is no statistical difference in fatal or nonfatal ipsilateral stroke risk between the three groups (type A-C), it can be a type II statistical error due to the small population in the present study.

There were significant differences in cumulative risks of ipsilateral stroke / bypass surgery and ipsilateral stroke / bypass surgery / repeated PTCBA between the 3 groups and the risk of cerebral events was the smallest in type A group, although the difference between type A and B is not significant and could be a type II error. Type A lesions were found to be related to a high success rate and a low cerebral event risk and therefore are the most successfully responsive to cerebral balloon angioplasty and thus PTCBA in type A lesions seems to be an appropriate treatment. In contrast, the high incidence of fatal or nonfatal ipsilateral stroke / bypass surgery found in type C lesions following PTCBA suggests that PTCBA should not be attempted on type C lesions.

The outcome of PTCBA for type B lesions remains controversial. However, if patients suffer from crescendo TIAs referable to intracranial

type B lesions in the posterior cerebral circulation where a high complication rate of bypass surgery can be anticipated, then PTCBA seems to be a relatively successful alternative to vascular reconstruction. When maximal medical therapy had no efficacy in improving crescendo TIAs or impending stroke due to an intracranial arterial stenosis or occlusion, there is no alternative but bypass surgery. Indeed, Caplan et al<sup>23</sup> reported that 2 out of 20 patients with severe MCA occlusive disease were treated by STA-MCA bypass surgery because of progression despite adequate anticoagulation therapy, but unfortunately it is not certain whether bypass surgery is really effective in the treatment of patients suffering from such recurrent TIAs. In the present study, 10 out of 12 patients presenting crescendo TIAs prior to PTCBA were successfully treated and their symptoms actually ceased completely after successful dilatation, although two patients in type C group underwent bypass surgery after unsuccessful PTCBA. This suggests that antegrade revascularisation by PTCBA has a great efficacy in improving perfusion to the affected territory and thus the authors expect that PTCBA can be effective in treating patients who suffer from crescendo TIAs or impending stroke.

Restenosis rate of 30% to 50% after standard coronary angioplasty has been reported<sup>18,30</sup> and overall the angiographic restenosis rate of 28% following PTCBA appears to be less than after standard coronary angioplasty. In the type A group, moreover, angiographic restenosis rate following PTCBA was 0% at one year. On consideration that coronary angioplasty is common world-wide despite a restenosis rate ranging from 30 to 50%, the overall restenosis rate of 28% following PTCBA seems to be acceptable.

The success rate and clinical outcome for the type A group is encouraging and warrants fur-

ther investigation and a randomised trial of PTCBA for intracranial atherosclerotic type A lesions. However, the present study has several important limitations. First of all, the study was conducted in a single neurosurgical department, the number of patients was very small and in addition technical skill has been acquired due to experience. Not only stenosis but also chronic total occlusion was treated by PTCBA<sup>31</sup> in the present study. In fact, five of six total occlusions in type B group and one of five total occlusions in type C group were successfully opened in the chronic stage. The present study included two kinds of patients groups, whose symptoms were crescendo TIAs or minor stroke prior to PTCBA and their restenosis rates appeared to be slightly different from one another, although this difference was not statistically significant. The two neurosurgeons who participated in the PTCBA procedures calculated the angiographic stenosis rate pre-and post-PTCBA and the assessor of the patients' clinical status pre- and post-PTCBA was not blind to angiographic results as only successful PTCBA treatments were included in the follow-up, although the assessor was not involved in PTCBA. Moreover, this study does not include a comparison group of patients who received medication only. Like carotid artery vertebral artery transluminal angioplasty study (Cavatas<sup>32</sup>), a randomised study is needed between medical and PTCBA techniques to assess the safety and efficacy of PTCBA therapy more accurately. Although repeatability is one of benefits of PTCBA, whether or not PTCBA should be repeated for restenosis prior to symptomatic recurrence is controversial, even though SPECT scans show lesions to be haemodynamically significant.

In conclusion, our results suggest that PTCBA for intracranial simple (type A) lesions produces a favourable clinical outcome for symptomatic patients.

## References

- 1 Diener HC, Hacke W et Al: the Lubeluzole International Study Group: Lubeluzole in acute ischaemic stroke. A double-blind, placebo-controlled phase II trial. *Stroke* 27: 76-81, 1996.
- 2 Nishimaru K, McHenry LC jr et Al: Cerebral angiographic and clinical differences in carotid system transient ischemic attacks between American Caucasian and Japanese patients. *Stroke* 15: 56-59, 1984.
- 3 Mathur VS, Guinn GA et Al: Surgical treatment for stable angina pectoris. Prospective randomized study. *N Engl J Med* 292: 709-713, 1975.
- 4 The EC/IC bypass study group: Failure of extracranial-intracranial arterial bypass to reduce the risk of ischaemic stroke. *N Engl J Med* 313: 1191-1200, 1985.
- 5 Hopkins LN, Budny JL: Complications of intracranial bypass for vertebrobasilar insufficiency. *J Neurosurg* 70: 207-211, 1989.
- 6 Higashida RT, Tsai FY et Al: Percutaneous transluminal angioplasty of extra- and intracranial cerebral vascular disease. *Neuroradiology* 37 (suppl): 449-450, 1995.
- 7 Clark WM, Barnwell SL et Al: Safety and efficacy of percutaneous transluminal angioplasty for intracranial atherosclerotic stenosis. *Stroke* 26: 1200-1204, 1995.
- 8 Mori T, Fukuoka M, Mori K: Percutaneous transluminal angioplasty for arteriosclerotic lesions of extra- and intracranial arteries. *Neuroradiology* 37: (suppl), 451-452, 1995.
- 9 Mori T, Mori K et Al: Serial angiographic follow-up after percutaneous transluminal cerebral angioplasty. *Neuroradiology* 39: 111-116, 1997.
- 10 Neto JIS, Santos AC et Al: Cerebral infarction in patients aged 15 to 40 years. *Stroke* 27: 2016-2019, 1996.
- 11 Vorstrup S, Brun B, Lassen NA: Evaluation of the cerebral vasodilatory capacity by the acetazolamide test before EC-IC bypass surgery in patients with occlusion of the internal carotid artery. *Stroke* 17: 1291-1298, 1986.
- 12 Touho H: Percutaneous transluminal angioplasty in the treatment of atherosclerotic disease of the anterior cerebral circulation and haemodynamic evaluation. *J Neurosurg* 82: 953-960, 1995.
- 13 Gibbs JM, Wise RJS et Al: Evaluation of cerebral perfusion reserve in patients with carotid-artery occlusion. *Lancet* 1: 310-314, 1984.
- 14 Gibbs JM, Wise RJS et Al: Cerebral haemodynamic changes after extracranial-intracranial bypass surgery. *J Neurol Neurosurg Psychiatry* 50: 140-150, 1987.
- 15 North America Symptomatic Carotid Endarterectomy Trial Collaborators: Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med* 325: 445-453, 1991.
- 16 Ryan TJ, Bauman WB, Kennedy JW: Revised guidelines for percutaneous transluminal coronary angioplasty: a report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee on Percutaneous Transluminal Angioplasty). *Circulation* 88: 2987-3007, 1993.
- 17 Serruyus PW, Luiten HE et Al: Incidence of restenosis after successful coronary angioplasty: a time-related phenomenon. A quantitative angiographic study in 342 consecutive patients at 1,2,3 and 4 months. *Circulation* 77: 361-371, 1988.
- 18 Nobuyoshi M, Kimura T et Al: Restenosis after successful percutaneous transluminal coronary angioplasty: Serial Angiographic Follow-up of 229 patients. *J Am Coll Cardiol* 12: 616-623, 1988.
- 19 Nishimaru K, Takeya Y et Al: Long-term prognosis of cerebral infarction-Influence of arterial stenotic lesion for survival- No Shinkei Geka 31: 1111-1116, 1979.
- 20 Chimowitz MI, Kokkinos J et Al: The warfarin-aspirin symptomatic intracranial disease study. *Neurology* 45: 1488-1493, 1995.
- 21 Hinton RC, Mohr JP et Al: Symptomatic middle cerebral artery stenosis. *Ann Neurol* 5: 152-157, 1979.
- 22 Corston RN, Kendall BE, Marshall J: Prognosis in middle cerebral artery stenosis. *Stroke* 15: 237-241, 1984.
- 23 Caplan L, Babikian V et Al: Occlusive disease of the middle cerebral artery. *Neurology* 35: 975-982, 1985.
- 24 Wechsler LR, Kistler JP et Al: The prognosis of carotid siphon stenosis. *Stroke* 17: 714-718, 1986.
- 25 Marzewski DJ, Furlan AJ et Al: Intracranial internal carotid artery stenosis: Longterm prognosis. *Stroke* 13: 821-824, 1982.
- 26 Craig DR, Meguro K et Al: Intracranial internal carotid artery stenosis. *Stroke* 13: 825-828, 1982.
- 27 Pessin MS, Gorelick PB et Al: Basilar artery stenosis: Middle and distal segments. *Neurology* 37: 1742-1746, 1987.
- 28 Moufarrij NA, Little JR et Al: Basilar and distal vertebral artery stenosis: Long-term follow-up. *Stroke* 17: 938-942, 1986.
- 29 Caplan LR: Bilateral distal vertebral artery occlusion. *Neurology* 33: 552-558, 1983.
- 30 Gruentzig AR, King SB III et Al: Long-term follow-up after percutaneous transluminal coronary angioplasty: the early Zurich experience. *N Engl J Med* 316: 1127-1132, 1987.
- 31 Mori T, Mori K et Al: Percutaneous transluminal cerebral angioplasty for total occlusion of middle cerebral arteries. *Neuroradiology* 39: 71-74, 1997.
- 32 Brown MM: Carotid and vertebral artery transluminal angioplasty study (Cavatas): Progress report. *Cerebrovasc Dis* 6 (S2): 34, 1996.

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