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Effects of carotid artery stenosis treatment on blood pressure

Clinical article

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Object. The purpose of this study was to evaluate and compare the long-term effects of carotid endarterectomy (CEA) and carotid artery stenting (CAS) on blood pressure (BP).

Methods. Between January 2003 and December 2009, 134 patients underwent 145 procedures for treatment of carotid artery stenosis. Patients with at least 1 year of clinical and radiographic follow-up after treatment were included in this study. A total of 102 patients met this criterion and were placed in the CEA group (n = 59) or the CAS group (n = 43) according to their treatment. The percentage change in BP decrement and the number of patients with a normotensive BP were evaluated and compared between the groups.

Results. There were no significant differences between the groups with regard to baseline characteristics. Compared with the pretreatment BP, the follow-up BPs were significantly decreased in both groups. At the 1-year follow-up, the percentage change in the BP decrement was greater in the CAS group (percentage change: systolic BP 9.6% and diastolic BP 12.8%) than in the CEA group (percentage change: systolic BP 5.9% [p = 0.035] and diastolic BP = 8.1% [p = 0.049]), and there were more patients with a normotensive BP in the CAS group (46.5%) than in the CEA group (22.0%, p = 0.012).

Conclusions. Both CEA and CAS have BP-lowering effects. Carotid artery stenting seems to have a better effect than CEA on BP at the 1-year follow-up.
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Key Words • blood pressure • carotid artery stenosis • carotid artery stenting • carotid endarterectomy • vascular disorders

Hypertension remains the most important well-documented, modifiable risk factor for stroke, and treating hypertension is among the most effective treatment strategies for preventing both ischemic and hemorrhagic stroke.[6] Blood pressure instability in the immediate postprocedural period is a common occurrence after carotid artery stenosis treatment with either CEA or CAS. Hypotension or hypertension can occur, which may result in a prolonged hospital stay or neurological complications. However, to the best of our knowledge, the long-term effects of CEA or CAS on BP changes have not been well evaluated. The aim of this study was to evaluate and compare the long-term effects between CEA and CAS on BP.

Methods

This retrospective study was approved by our institutional review board, and the need for patient informed consent was waived. Between January 2003 and December 2009, 134 patients underwent 145 procedures for the treatment of carotid artery stenosis by either CEA or CAS. Among these patients, those who had at least 1 year of clinical and radiographic follow-up after treatment were included in this study. After exclusion of 32 patients (23 patients were lost to follow-up, 6 died, and 3 underwent balloon angioplasty only), a total of 102 patients constituted the population of this study. These patients were placed in the CEA group (n = 59) or the CAS group (n = 43) according to their treatment. Among them, 2 patients underwent bilateral CAS, 3 patients underwent bilateral CEA, and 1 patient underwent CAS on one side and CEA on the other. For the 6 patients who underwent bilateral procedures, 2 (4.7%) were included in the CAS group and 4 (6.8%) were included in the CEA group.

The SBP and DBP were measured at the following 4 different time points: pretreatment, posttreatment, the 1-month follow-up, and the 1-year follow-up. Noninvasive BP monitoring was used for routine BP measurements. The pretreatment, posttreatment, 1-month follow-up, and 1-year follow-up BPs were calculated by averaging the noninvasive BP values for 24 hours at the pretreatment

Abbreviations used in this paper: BP = blood pressure; CAS = carotid artery stenting; CEA = carotid endarterectomy; DBP = diastolic BP; SBP = systolic BP.

and the posttreatment time points, and for 1 month (twice a day; immediately after awaking in the morning and before sleep at night) at the 1-month follow-up period, and 1 month prior to the 1-year follow-up time point. Before the procedure, an assigned nurse measured BP every 2 hours for 24 hours using an automatic arm manometer. After the procedure, the nurse measured BP every hour for 8 hours and then every 2 hours for 16 hours. During the follow-up period, most of the patients were instructed on how to measure their BP in their left arm in the sitting position at home with the aid of commercial automatic BP measuring equipment.

The average SBP and DBP values at each time point were evaluated in each group. In addition, the percentage change in the SBP decrement at each time point was calculated using the following equation and was compared between the groups: percentage change in SBP decrement $= \{(SBP_n - SBP_0)/SBP_n\} \times 100$, (n = 1, 2, or 3), where SBP_0, SBP_1, SBP_2, and SBP_3 represent the pretreatment BP, the posttreatment BP, the 1-month follow-up BP, and the 1-year follow-up BP, respectively. The percentage change in the DBP decrement was calculated using the equivalent equation.

A normotensive BP was defined when the SBP/DBP decreased to lower than 120/80 mm Hg after the procedures. The number of patients with a normotensive BP and the change in the number of antihypertensive drugs taken by the patients at each time point were evaluated and compared. The antihypertensive drugs consisted of diuretics, calcium channel blockers, angiotensin receptor blockers, angiotensin-converting enzyme inhibitors, and/or beta blockers in a single or combined regimen.

Surgical Management

All CEA procedures were performed under general anesthesia with somatosensory evoked potential and/or electroencephalography monitoring. If preoperative transfemoral cerebral angiography showed that there was not enough cross-filling during the Matas Test and the Allcock Test, carotid shunts were used during CEA. A local lidocaine infiltration at the carotid sinus was performed only when the patient's heart rate was lower than 40 bpm. A local injection was performed in 8 (13.6%) of 59 patients in this study. When closing the carotid artery after plaque removal, we preferred continuous-suture closure for artery repair rather than carotid patch angioplasty.

Carotid artery stenting was routinely performed after administration of a local anesthetic and with strict BP monitoring. Among the 43 patients enrolled in this study, a distal embolic protection device was used in the 38 most recently treated patients. A self-expanding nitinol stent was used for all patients undergoing CAS.

Statistical Analysis

Statistical analyses were conducted using SPSS (version 13.0, SPSS, Inc.). We evaluated the significance of BP changes between the pretreatment and follow-up periods by using a paired t-test. The baseline characteristics, procedure-related complications, restenosis rates, changes in the number of drugs, and the percentage change in BP decrement between the groups were evaluated using the Student t-test. Probability values < 0.05 were considered statistically significant. Mean values are presented as the mean ± SD.

Results

The baseline characteristics of each group are shown in Table 1. The mean age was younger in the CEA group (63.6 ± 7.0 years) than in the CAS group (67.0 ± 8.1 years, $p = 0.032$). Symptomatic lesions were found in 45 patients (76.3%) in the CEA group and in 32 patients (74.4%) in the CAS group ($p = 0.832$). The mean stenosis rates were 81.3% ± 14.4% in the CEA group and 77.5% ± 12.9% in the CAS group ($p = 0.175$). There were no significant differences between the groups for the other baseline characteristics, including the presence of hypertension, diabetes, hypercholesterolemia, alcohol intake, body mass index, atrial fibrillation, or the proportion of the patients who underwent bilateral procedures. The mean number of antihypertensive drugs at the pretreatment time point was 1.29 ± 0.81 in the CEA group and 1.47 ± 0.85 in the CAS group ($p = 0.290$). Procedure-related complications (5.1% in the CEA group and 2.3% in the CAS group, $p = 0.431$) and the restenosis rate 1 year after the procedure (1.7% in the CEA group and 7.0% in the CAS group, $p = 0.483$) were not significantly different between the groups.

The mean SBPs and DBPs for each time point are shown in Fig. 1. Compared with the pretreatment BPs, the follow-up BPs were significantly decreased in each group after treatment. In the CEA group, the mean SBP/DBP values were 122.5/73.0, 125.4/75.5, and 129.5/77.7 mm Hg at the posttreatment time point, the 1-month follow-up, and the 1-year follow-up, respectively. Compared with 136.9/83.5 mm Hg at the pretreatment time point, the BPs in the CEA group were significantly decreased ($p < 0.01$). In the CAS group, the mean SBP/DBP values were 122.7/76.2, 127.2/78.5, and 128.5/77.3 mm Hg at the posttreatment time point, the 1-month follow-up, and the 1-year follow-up, respectively. Compared with 140.7/87.2 mm Hg at the pretreatment time point, the mean BPs in the CAS group were also significantly decreased ($p < 0.01$). In both groups, the SBP and DBP were significantly decreased after the procedure, and the values tended to increase slightly afterward until the 1-year follow-up. The follow-up BPs were significantly lower than the pretreatment BPs.

Table 2 shows the percentage changes in the BP decrement at each time point compared with the pretreatment BP. The patients in the CEA group had a 12.6%, 9.3%, and 5.9% SBP decrement and a 15.9%, 11.8%, and 8.1% DBP decrement at the posttreatment time point, the 1-month follow-up, and the 1-year follow-up, respectively. The patients in the CAS group had a 15.1%, 11.1%, and 9.6% SBP decrement and a 15.3%, 11.6%, and 12.8% DBP decrement at the posttreatment time point, the 1-month follow-up, and the 1-year follow-up, respectively. The percentage change in the SBP decrement at the 1-year follow-up in the CAS group was greater ($p = 0.035$) than that in the CEA group (Fig. 2). In addition, the percentage change in the DBP decrement at the 1-year follow-up in

TABLE 1: Baseline characteristics, procedure-related complications, and restenosis rate of each group*

Parameter	Value		p Value
	CEA Group (n = 59)	CAS Group (n = 43)	
mean age (yrs)	63.6 ± 7.0	67.0 ± 8.1	0.032
female	7 (11.9)	11 (25.6)	0.052
rt side	37 (62.7)	16 (37.2)	0.006
symptoms present	45 (76.3)	32 (74.4)	0.832
bruit	4 (6.8)	1 (2.3)	
retinal	6 (10.2)	4 (9.3)	
hemispheric	38 (64.4)	27 (62.8)	
previous stroke	8 (13.6)	6 (14.0)	
mean stenosis rate (%)	81.3 ± 14.4	77.5 ± 12.9	0.175
hypertension	53 (89.8)	33 (76.7)	0.091
diabetes	26 (44.1)	16 (37.2)	0.492
hypercholesterolemia	20 (33.9)	17 (39.5)	0.563
smoking	32 (54.2)	20 (46.5)	0.446
alcohol	23 (39.0)	19 (44.2)	0.602
mean BMI	24.5 ± 2.4	24.9 ± 2.8	0.486
atrial fibrillation	1 (1.7)	2 (4.7)	0.388
mean no. of pretreatment drugs	1.29 ± 0.81	1.47 ± 0.85	0.290
no. of procedure-related complications	3 (5.1)	1 (2.3)	0.431
minor	2 (3.4)	1 (2.3)	
major	1 (1.7)	0 (0.0)	
restenosis rate	1 (1.7)	3 (7.0)	0.483

* Values are presented as the number of patients (%) unless indicated otherwise. Mean values are presented as ± SD. Abbreviation: BMI = body mass index.

TABLE 2: Percentage changes of the BP decrements between groups*

BP & Time Points	% Change		p Value
	CEA Group	CAS Group	
SBP			
post-Tx	12.6	15.1	0.253
1 mo	9.3	11.1	0.340
1 yr	5.9	9.6	0.035
DBP			
post-Tx	15.9	15.3	0.854
1 mo	11.8	11.6	0.952
1 yr	8.1	12.8	0.049

* Tx = treatment.

the CAS group was greater (p = 0.049) than that in the CEA group.

Compared with pretreatment BP, the CAS group comprised a greater percentage of patients with a normotensive BP at the follow-up time points than the CEA group (Table 3). At the 1-month follow-up, 29 patients (49.2%) in the CEA group and 22 patients (51.2%) in the CAS group exhibited a decrease in SBP/DBP to lower than 120/80 mm Hg, which was not significantly different between the groups (p = 0.695). Yet at the 1-year follow-up, there were significantly more patients with a normotensive BP in the CAS group (46.5%) than in the CEA group (22.0%, p = 0.012). In contrast, 5 patients (8.5%) in the CEA group and 2 patients (4.7%) in the CAS group had hypertension (SBP/DBP ≥ 140/90 mm Hg) during the immediate postprocedural period. At the 1-year follow-up, postprocedural hypertension occurred in 2.9% of the patients overall (2 in the CEA group and 1 in the CAS

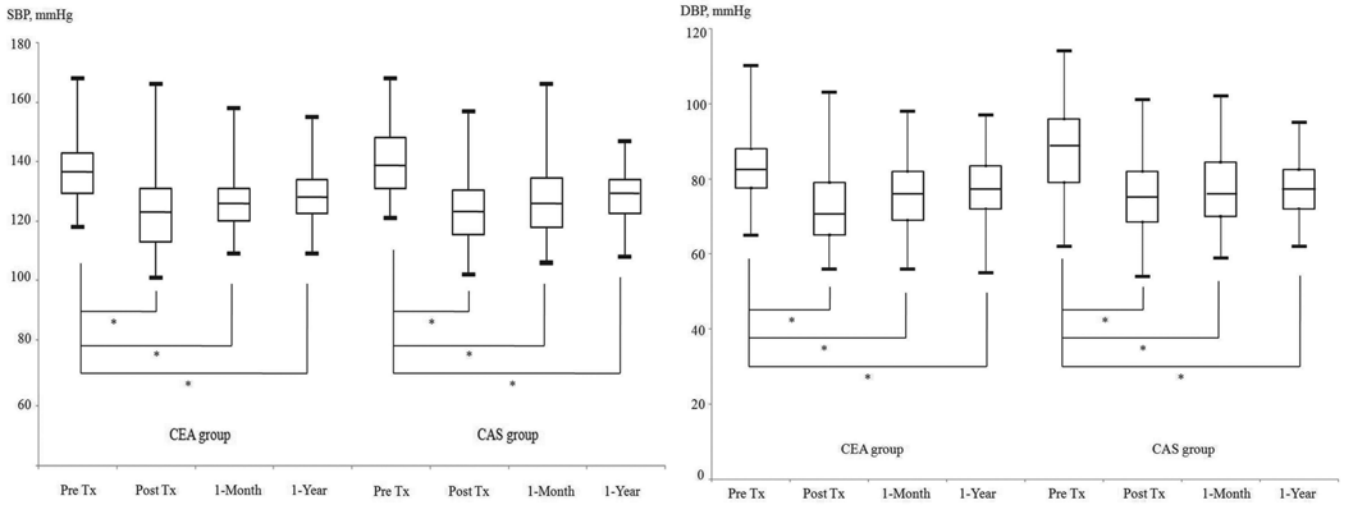

Fig. 1. Box-and-whisker plots showing the mean noninvasive BP at each time point in the 2 groups. Compared with the pretreatment BP, the follow-up BPs were significantly decreased after CEA or CAS. In both groups, the BPs were significantly decreased after the procedure, and they tended to increase slightly afterward until the 1-year follow-up. *p < 0.01. Tx = treatment. The *horizontal lines* in the boxes indicate the median. The *error bars* represent the ranges, and the *boxes* represent the lower quartile (25%) and upper quartile (75%) values.

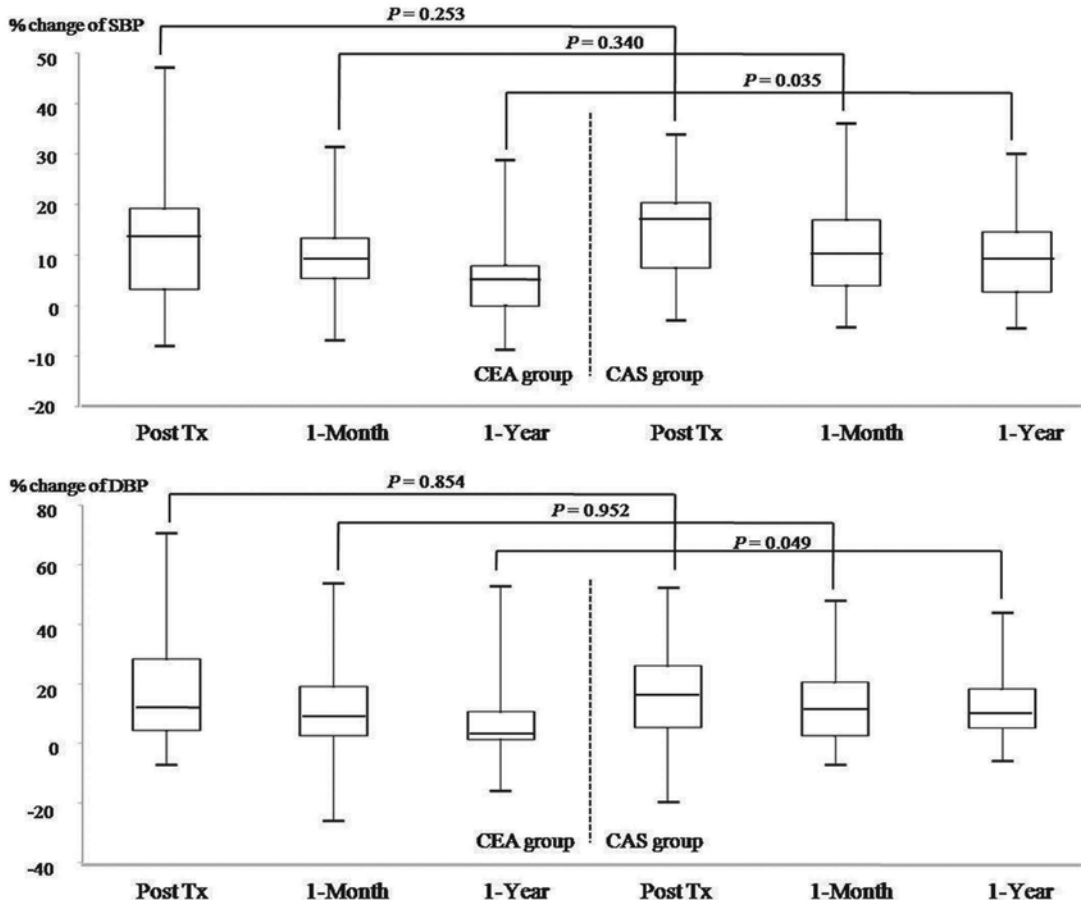

Fig. 2. Box-and-whisker plots showing the percentage change in BP decrement in the 2 groups. At the 1-year follow-up, the percent change of the BP in the CAS group was greater than that in the CEA group. The *horizontal lines* in the boxes indicate the median. The *error bars* represent the ranges, and the *boxes* represent the lower quartile (25%) and upper quartile (75%) values.

group). The number of antihypertensive drugs taken by the patients was not significantly changed at the follow-up time points.

Compared with the mean number of pretreatment drugs taken, there were no significant changes at the follow-up time points (1-month and 1-year follow-up) between the groups. However, 2 patients (4.7%) in the CAS group and 18 patients (30.5%) in the CEA group ($p = 0.043$) at the 1-year follow-up were taking more antihypertensive drugs (Table 4).

Discussion

Carotid artery stenosis can currently be treated using CEA or CAS. During these procedures, periprocedural hemodynamic instabilities, such as hypotension, bradycardia, or hypertension, are often encountered.[1,11–14,17,20] Hypertension has been noted with a frequency of 9%–38% after CEA[18,20] and 39% after CAS.[16] A wide variation in the frequency of hypotension has been observed after CEA (12%–50%)[2,20] and after CAS (18.8%–56.1%).[4,8] Since there are few reports for the long-term follow-up of BP changes after treatment of carotid artery stenosis, we evaluated the long-term effects of carotid artery stenosis treatment on BP, and we compared the effects between CEA and CAS.

In this study, a low frequency of episodes of increased BP was noted at the 1-year follow-up (2.9% of the patients with SBP/DBP > 140/90 mm Hg), but a much higher frequency of episodes of normotensive BP (SBP/DBP < 120/80 mm Hg) was observed at the 1-year follow-up (22.0% after CEA and 46.5% after CAS, Table 3). The CAS group showed a greater frequency of normotensive BP than the CEA group. Although the pretreatment BP was slightly lower in the CEA group, the 1-year follow-up BP was lower in the CAS group (Fig. 1). In addition, the percentage changes of SBP and DBP were greater in the CAS group at the 1-year follow-up than in the CEA group, indicating that CAS has a greater tendency for BP-lowering effects than CEA.

Damage to the carotid baroreceptor is an important cause of periprocedural hemodynamic instability. The adventitial baroreceptors in the carotid sinus, which is the dilated segment of the internal carotid artery at its origin from the common carotid artery, are stretch receptors. The arterial wall of the carotid sinus is thinner than that of other arteries of the same caliber. Increased firing of these baroreceptors inhibits sympathetic neurons in the tractus solitarius nucleus, which reduces the sympathetic

TABLE 3: Number of patients with normotensive BP at the follow-up time points*

Time Point	No. of Patients (%) CEA Group (n = 59)	CAS Group (n = 43)	p Value
1-mo follow-up	29 (49.2)	22 (51.2)	0.695
1-yr follow-up	13 (22.0)	20 (46.5)	0.012

* Normotensive BP is lower than 120/80 mm Hg.

TABLE 4: Changes in the number of antihypertensive drugs taken at the follow-up time points

Time Point & No. of Drugs	No. of Patients (%) CEA Group (n = 59)	CAS Group (n = 43)	p Value
1-mo follow-up			
same	34 (57.6)	33 (76.7)	0.116
increased	22 (37.3)	3 (7.0)	0.052
decreased	3 (5.1)	7 (16.3)	0.074
1-yr follow-up			
same	34 (57.6)	28 (65.1)	0.208
increased	18 (30.5)	2 (4.7)	0.043
decreased	7 (11.9)	13 (30.2)	0.085

tone in the peripheral blood vessels, leading to a reduction in systemic BP. Atherosclerosis and hypertension lower the baroreceptors' sensitivity because the carotid sinus becomes stiffer and more resistant to deformation. In this study, it might be suggested that CAS has a greater tendency for a BP-lowering effect, possibly because of greater compliance and an increase in the diameter of the arterial segment after the angioplasty. Furthermore, the radial force of the stent imparts chronic mechanical pressure over the carotid sinus.[1] On the other hand, even though CEA can result in optimal BP control in a patient with treatment-resistant hypertension,[9] CEA may induce the surgical denervation of the carotid sinus nerve or induce dysfunction of the carotid baroreceptors during the procedure, leading to less reduction in BP at the long-term follow-up.

In this study, more patients had carotid artery stenosis on the right side in the CEA group (62.7%) than in the CAS group (25.6%, p = 0.006). The difference in the laterality of the carotid artery stenosis might affect the results of this study since some studies in humans have suggested some laterality of the carotid sinus output. Williamson et al.[19] suggested that afferent input from the left carotid sinus might have a greater influence on efferent muscle sympathetic outflow. Diedrich et al.[5] suggested the presence of a right-sided lateralization of carotid baroreceptor output. These authors concluded that right lateralization of the sympathetic activity to the vessels is indicated by normalized burst strength parameters of bilateral muscle sympathetic nerve activity recordings. According to their study, treating one side of the carotid artery stenosis might have a greater BP-lowering effect than treating the other side. On the other hand, Furlan et al.[7] concluded that there was no asymmetry in the neural sympathetic discharge responses after single-sided carotid baroreceptor stimulation. In this study, BP-lowering effects were observed in both groups despite the fact that there were more left-sided lesions in the CAS group than the CEA group. In addition, there was insufficient evidence to explain why the lateralization of the carotid sinus output had a better long-term (1-year follow-up) BP-lowering effect in the CAS group. Overall, a complete understanding of the lateralization of the carotid sinus output is difficult to arrive at from the current data.

Blood pressure management plays a crucial role in the prevention of stroke. Any decrease in BP is associated with a decrease in the relative risk of stroke.[10] A study of antihypertensive treatment in patients with a history of cerebrovascular events found that a reduction of 9/4 mm Hg was associated with a reduced risk of stroke of 28%, so even small decreases in BP are highly effective.[15] In this study, the long-term effect of CAS is associated with greater sustained reduction of BP than that associated with CEA, which means that CAS may have better effects on BP control than CEA after treating carotid artery stenosis. Therefore, CAS may have a better effect on the secondary prevention of stroke than CEA, and it may be worth further studying the effect of this greater BP lowering of CAS than CEA on the secondary prevention of stroke.

Even though there were some changes in the number of antihypertensive drugs taken by the patients in each group during the follow-up periods, there were no significant differences between the groups except for the numbers of the drugs that were being taken at the 1-year follow-up (Table 4). More patients in the CEA group (30.5%) were taking antihypertensive drugs at the 1-year follow-up than those in the CAS group (4.7%, p = 0.043). However, CAS treatment had a longer-term effect on BP control than CEA treatment, which indicated that the number of antihypertensive drugs may not affect the BP-lowering effect of carotid artery stenosis treatment.

The limitations of this study include its retrospective design, which excluded randomization and access to certain data. It was very difficult, for example, to analyze the dosage changes of the antihypertensive drugs, and therefore such an analysis was not done in this study. Some previous studies excluded patients who changed their antihypertensive drugs.[11] However, we analyzed the changes in the number of drugs, and we attempted to evaluate the correlation between the number of antihypertensive drugs and the BP-lowering effects. Finally, we examined the average of the noninvasive BP values rather than ambulatory BP values.[3,21] Noninvasive BP measurements are less reliable than ambulatory BP measurements as a representative measure. We routinely educate patients to check their BP twice a day to control their BP during the follow-up period. To help eliminate factors that reduce the reliability of single blood pressure values, the BPs were calculated by averaging the noninvasive BP values for 24 hours pre- and posttreatment, and for 1 month (twice a day) at the 1-month follow-up and the 1-year follow-up.

Conclusions

Both CEA and CAS have BP-lowering effects. Carotid artery stenting seems to have a better effect than CEA on the BP decrement at the 1-year follow-up after the procedure. A randomized double-blind study is expected to demonstrate a certain association between the BP-lowering effect and carotid artery stenosis treatment.

Disclosure

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Author contributions to the study and manuscript preparation include the following. Conception and design: Chung. Acquisition of data: Chung, Paik, Hyun. Analysis and interpretation of data: Chung, Kim. Drafting the article: Chung, Paik. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Park. Statistical analysis: Chung. Study supervision: Park.

References

1. Bagshaw RJ, Barrer SJ: Effects of angioplasty upon carotid sinus mechanical properties and blood pressure control in the dog. **Neurosurgery 21:**324–330, 1987
2. Cafferata HT, Merchant RF Jr, DePalma RG: Avoidance of postcarotid endarterectomy hypertension. **Ann Surg 196:** 465–472, 1982
3. Calhoun DA, Jones D, Textor S, Goff DC, Murphy TP, Toto RD, et al: Resistant hypertension: diagnosis, evaluation, and treatment: a scientific statement from the American Heart Association Professional Education Committee of the Council for High Blood Pressure Research. **Circulation 117:**e510–e526, 2008
4. Dangas G, Laird JR Jr, Satler LF, Mehran R, Mintz GS, Larrain G, et al: Postprocedural hypotension after carotid artery stent placement: predictors and short- and long-term clinical outcomes. **Radiology 215:**677–683, 2000
5. Diedrich A, Porta A, Barbic F, Brychta RJ, Bonizzi P, Diedrich L, et al: Lateralization of expression of neural sympathetic activity to the vessels and effects of carotid baroreceptor stimulation. **Am J Physiol Heart Circ Physiol 296:**H1758–H1765, 2009
6. Furie KL, Kasner SE, Adams RJ, Albers GW, Bush RL, Fagan SC, et al: Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. **Stroke 42:**227–276, 2011
7. Furlan R, Diedrich A, Rimoldi A, Palazzolo L, Porta C, Diedrich L, et al: Effects of unilateral and bilateral carotid baroreflex stimulation on cardiac and neural sympathetic discharge oscillatory patterns. **Circulation 108:**717–723, 2003
8. Gil-Peralta A, Mayol A, Marcos JR, Gonzalez A, Ruano J, Boza F, et al: Percutaneous transluminal angioplasty of the symptomatic atherosclerotic carotid arteries. Results, complications, and follow-up. **Stroke 27:**2271–2273, 1996
9. Kirin I, Kirin M, Sicaja M, Missoni S, Pehar M, Prka Z, et al: Carotid endarterectomy unexpectedly resulted in optimal blood pressure control. **Coll Antropol 33:**1229–1231, 2009
10. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, et al: Blood pressure, stroke, and coronary heart disease. Part 1, prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. **Lancet 335:**765–774, 1990
11. McKevitt FM, Sivaguru A, Venables GS, Cleveland TJ, Gaines PA, Beard JD, et al: Effect of treatment of carotid artery stenosis on blood pressure: a comparison of hemodynamic disturbances after carotid endarterectomy and endovascular treatment. **Stroke 34:**2576–2581, 2003
12. Nonaka T, Oka S, Miyata K, Mikami T, Koyanagi I, Houkin K, et al: Prediction of prolonged postprocedural hypotension after carotid artery stenting. **Neurosurgery 57:**472–477, 2005
13. Nouraei SA, Al-Rawi PG, Sigaudo-Roussel D, Giussani DA, Gaunt ME: Carotid endarterectomy impairs blood pressure homeostasis by reducing the physiologic baroreflex reserve. **J Vasc Surg 41:**631–637, 2005
14. Pappadà G, Beghi E, Marina R, Agostoni E, Cesana C, Legnani F, et al: Hemodynamic instability after extracranial carotid stenting. **Acta Neurochir (Wien) 148:**639–645, 2006
15. PROGRESS Collaborative Group: Randomised trial of a perindopril-based blood-pressure-lowering regimen among 6,105 individuals with previous stroke or transient ischaemic attack. **Lancet 358:**1033–1041, 2001 (Erratum in **Lancet 358:**1556, 2001; erratum in **Lancet 359:**2120, 2002)
16. Qureshi AI, Luft AR, Sharma M, Janardhan V, Lopes DK, Khan J, et al: Frequency and determinants of postprocedural hemodynamic instability after carotid angioplasty and stenting. **Stroke 30:**2086–2093, 1999
17. Taha MM, Toma N, Sakaida H, Hori K, Maeda M, Asakura F, et al: Periprocedural hemodynamic instability with carotid angioplasty and stenting. **Surg Neurol 70:**279–286, 2008
18. Wade JG, Larson CP Jr, Hickey RF, Ehrenfeld WK, Severinghaus JW: Effect of carotid endarterectomy on carotid chemoreceptor and baroreceptor function in man. **N Engl J Med 282:**823–829, 1970
19. Williamson JW, Muzi M, Ebert TJ: Unilateral carotid sinus stimulation and muscle sympathetic nerve activity in man. **Med Sci Sports Exerc 28:**815–821, 1996
20. Wong JH, Findlay JM, Suarez-Almazor ME: Hemodynamic instability after carotid endarterectomy: risk factors and associations with operative complications. **Neurosurgery 41:**35–43, 1997
21. Zanchetti A: The role of ambulatory blood pressure monitoring in clinical practice. **Am J Hypertens 10:**1069–1080, 1997

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