Factors associated with hemodynamic instability following carotid artery stenting

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ABSTRACT

Objective: Carotid artery stenting (CAS) is a major treatment option for carotid artery stenosis, and a recognized alternative to carotid endarterectomy (CEA). However, CAS-related hemodynamic instability occurs frequently and is a known major risk factor of associated complications. This study was undertaken to identify the risk factors of hemodynamic instability associated with CAS.

Methods: We analyzed the medical records of 128 patients with carotid artery stenosis treated by CAS at our institution from 2014 to 2019 to identify the risk factors of hemodynamic instability after CAS. In addition, the incidences of hemodynamic instability, including bradycardia and hypotension, during and after the procedure were investigated.

Results: Overall, periprocedural bradycardia requiring atropine occurred in 18 (14.1%) of the 128 study subjects, and postprocedural persistent hypotension requiring vasopressors occurred in 15 (11.7%). Risk-adjusted analysis showed carotid bulb involvement of a stenotic lesion was an independent risk factor of periprocedural bradycardia (OR 4.25, 95% CI 1.34–13.40) and postprocedural persistent hypotension (OR 7.36, 95% CI 1.86–29.12). However, though a preoperative regimen of ≥2 antihypertensives was found to be an independent protective factor against postprocedural persistent hypotension (OR 0.17, 95% CI 0.04–0.81), it was not associated with periprocedural bradycardia (OR 0.37, 95% CI 0.08–1.60).

Conclusions: The risk of hemodynamic instability development is greater when a carotid stenotic lesion involves the carotid bulb, which cautions that careful evaluation is necessary. In addition, the receipt of antihypertensive regimens before CAS had a protective effect on persistent hypotension after CAS, but did not affect bradycardia.

1. Introduction

Based on the conclusions of several comparative studies on carotid artery stenting (CAS) and carotid endarterectomy (CEA), the two modalities are similar in terms of prognosis and complications, and as a result, CAS is now considered an alternative to CEA for the treatment of carotid artery stenosis [1–3]. Furthermore, CAS is a preferred treatment strategy for many patients due to its less invasive nature and greater patient satisfaction. Despite this trend, hemodynamic instability, including hypotension and bradycardia, occurs relatively frequently after CAS. This phenomenon is commonly attributed to a baroreceptor reflex caused by direct compression of the carotid bulb during balloon angioplasty and stenting, and requires attention because it can temporarily reduce cerebral perfusion and impair ability to wash out microemboli [1,4–6].

The risk factors of hemodynamic instability occurring during or after CAS remain controversial, and thus, in this study, we sought to determine the incidence and identify the risk factors of hemodynamic instability occurring during and after CAS.

2. Material and methods

2.1. Patient selection and data collection

One hundred and twenty-eight patients that underwent CAS due to carotid stenosis by two neuro-interventionists at a single institution...
between January 2014 and December 2019 were studied retrospectively. Patient eligibility for CAS was determined by neurologists and neuro-interventionists using diagnostic digital subtraction angiography images to determine degrees of carotid artery stenosis. Patients that received atropine for bradycardia during the procedure and patients administered vasopressin for hypotension during closed monitoring after the procedure were examined, and the effects of medical history on outcomes in these two patient groups were analyzed. In addition, we investigated the drugs patients were taking before the procedure, especially antihypertensive regimens. The presence of symptoms related to carotid stenosis was also investigated, and carotid stenosis location, carotid bulb involvement (defined as a distance between the carotid bifurcation and the center of the stenotic lesion of < 10 mm), and stenosis ratio were examined.

This retrospective study was approved by the Institutional Review Board of Wonju Severance Christian Hospital (CR320080).

### 2.2. CAS procedure

CAS procedures were performed by two neuro-interventionists with at least 5 years’ experience. All patients received a neurological assessment by neurologists within 24 h prior to and after CAS using the National Institutes of Health Stroke Scale and the modified Rankin Scale. In addition, all patients received dual antiplatelet therapy with aspirin and clopidogrel at least 7 days before the procedure. CAS was performed under local anesthesia, and an intraarterial bolus of heparin (5000 International units) was injected immediately after femoral sheath puncture. A distal embolic protection device (Spider FX; EV3, Plymouth, Minnesota, USA) was used for intra-procedural neuroprotection, and an open-cell stent (Protégé; EV3, Plymouth, Minnesota, USA) was selected in all cases. Balloon inflation was performed by selecting a balloon that does not exceed the diameter of the distal area of the stenotic lesion for minimal angioplasty. Blood pressure and heart rate were closed monitored by continuous monitoring of the arterial line and by electrocardiography during micro-catheter access, balloon inflation, and stent deployment around the carotid stenotic lesion. When heart rate fell below 20 beats/min, atropine (0.25 mg) was immediately injected intravenously, and in all patients heart rates recovered within seconds. Periprocedural hemodynamic instability was defined as the requirement for intravenous atropine injection for bradycardia (heart rate < 20 beats/min) during CAS. After the procedure, patients were moved to an intensive care or stroke unit to maintain closed monitoring, and vasopressor infusion was performed when hypotension (a systolic blood pressure of < 80 mmHg) persisted. Postprocedural hypotension was defined as persistent systolic blood pressure at < 80 mmHg, requiring intravenous vasopressor infusion which lasted more than 1 h.

### 2.3. Statistical analysis

The two-sample t-test or the Chi-square test (Fisher’s exact test) were used, as appropriate, to compare hemodynamic instability occurrences. Logistic regression analysis was used to identify factors that predicted CAS-associated hemodynamic instability; results are expressed as odds ratios (ORs) and 95 % confidence intervals (CIs). Continuous variables are presented as means and standard deviations, and categorical variables as frequencies and percentages. The analysis was conducted using SPSS version 24 (IBM, Armonk, NY, USA), and statistical significance was accepted for P values of < 0.05.

### 3. Results

The study subjects were 110 men and 18 women of mean age 71.5 years (range 50–90). CAS was performed on 79 symptomatic (61.7 %) and 49 asymptomatic (38.3 %) cases. Eighteen of the study subjects (14.1 %) were administered intravenous atropine for bradycardia during the procedure, and 15 (11.7 %) received vasopressor infusion due to persistent hypotension post-CAS. The baseline characteristics of the study subjects are summarized in Table 1.

During CAS, bradycardia requiring atropine occurred significantly more frequently when a lesion involved the carotid bulb (p = 0.020). However, taking antihypertensive regimens for hypertension prior to CAS was not related to bradycardia occurring during the procedure (p = 0.891). The number of types of antihypertensive regimens was greater in those that experienced bradycardia (1.2 ± 1.3) than in those that did not (0.9 ± 1.2), but this difference was not significant (Table 2).

In patients with persistent hypotension requiring vasopressor infusion post-procedurally, lesions significantly involved carotid bulbs (p = 0.006). Furthermore, the incidence of persistent hypotension was significantly lower for those on an antihypertensive regimen before CAS (p = 0.048). Furthermore, the incidence of postprocedural persistent hypotension tended to decrease as the number of types of antihypertensive regimens increased (p = 0.044) (Table 3).

Patient age, sex, medical history (e.g., hypertension, diabetes mellitus, or dyslipidemia) did not significantly influence the occurrence of hemodynamic instability during or after CAS. In addition, the occurrence of symptoms related to lesions and stenosis ratio did not influence hemodynamic instability (Tables 2 and 3).

Multivariate logistic regression analysis showed a distance between the carotid bifurcation and the center of the stenotic lesion of < 10 mm was an independent risk factor of perioperative bradycardia (OR 4.25, 95% CI 1.34–13.40) and of persistent hypotension after CAS (OR 7.36, 95% CI 1.86–29.12). On the other hand, two or more types of antihypertensive regimen taken before the procedure was found to be an independent protective factor of postprocedural persistent hypotension as compared with not taking a antihypertensive regimen (OR 0.17, 95% CI 0.04–0.81). However, the number of antihypertensive regimens received prior CAS was not found to be related to perioperative bradycardia (Tables 4 and 5).

### Table 1

Characteristics of 128 Patients Who Underwent Carotid Artery Stenting.

| Characteristics                        | No (%) of Patients (N = 128) |
|----------------------------------------|-------------------------------|
| Age                                    | 71.5 ± 8.1                    |
| Age, >70                               | 81 (63.3)                     |
| Age, >80                               | 19 (14.8)                     |
| Gender, male                           | 110 (85.9)                    |
| History                                |                               |
| Hypertension                           | 92 (71.9)                     |
| Diabetes mellitus                      | 44 (34.4)                     |
| Hyperlipidemia                         | 52 (40.6)                     |
| Smoking                                | 40 (31.3)                     |
| Coronary artery disease                | 35 (27.3)                     |
| Pulmonary disease                      | 18 (14.1)                     |
| Renal disease                          | 11 (8.6)                      |
| Transient ischemic attack              | 19 (14.8)                     |
| Stroke                                 | 72 (56.3)                     |
| Previous CAS* history                  | 4 (3.1)                       |
| Radiotherapy                           | 3 (2.3)                       |
| Cancer                                 | 12 (17.2)                     |
| Antihypertensive regimen               | 73 (57.0)                     |
| 0 antihypertensive regimen             | 55 (43.0)                     |
| 1 antihypertensive regimen             | 20 (15.6)                     |
| >1 antihypertensive regimen            | 53 (41.4)                     |
| Count of antihypertensive regimen      | 1.2 ± 1.2                     |
| Symptomatic                            | 79 (61.7)                     |
| Stenosis site                          |                               |
| Right side                             | 73 (57.0)                     |
| Left side                              | 55 (43.0)                     |
| Carotid bulb involved                  | 60 (46.9)                     |
| Lesion stenosis (%)                    | 78.0 ± 7.3                    |
| Periprocedural bradycardia             | 18 (14.1)                     |
| Postprocedural persistent hypotension  | 15 (11.7)                     |

* Carotid artery stenting.
Hypotension that occur during catheter-based carotid artery procedure [7, 11, 12, 18]. Hemodynamic changes like bradycardia and titial baroreceptors and the carotid sinus are manipulated during the intervention are relatively well-known physiological responses and in most cases are transient and self-limiting [8, 12]. However, when these hemodynamic responses are severe enough, periprocedural cardiopulmonary and neurological adverse effects may occur [8, 19, 20] that increase the risk of hospital complications and long-term death after CAS [7, 11]. In clinical practice, bradycardia and hypotension are relatively common during CAS, and urgent medications, such as atropine or epinephrine, are sometimes required. Due to these concerns, some intervention are relatively well-known physiological responses and in most cases are transient and self-limiting [8, 12]. However, when these hemodynamic responses are severe enough, periprocedural cardiopulmonary and neurological adverse effects may occur [8, 19, 20] that increase the risk of hospital complications and long-term death after CAS [7, 11]. In clinical practice, bradycardia and hypotension are relatively common during CAS, and urgent medications, such as atropine or epinephrine, are sometimes required. Due to these concerns, some

4. Discussion

The diagnostic rate of carotid artery stenosis is increasing in parallel with societal aging and diagnostic technique developments. Previously, CEA was routinely performed, but due to endovascular procedural and device developments, CAS results have matched CEA results over recent years, and as a result, CAS is being increasingly adopted as an alternative to CEA [7].

Nonetheless, it is widely known that CAS is a high-level procedure that presents risks of complications [8–10]. Neurological complications, such as stroke, are the most feared complications of this procedure, and efforts to reduce these complications continue to drive research on improved neuroprotective devices and treatment methods. Other associated physiological phenomena may also affect treatment outcomes, and hemodynamic instabilities, such as bradycardia or hypotension, have been reported to occur with incidences of 5%–76% after CAS [8, 11–13]. In addition, persistent hypotension requiring vasopressor infusion has been reported in 12%–40% of cases [11,14–17]. In this study, 14.1% of patients required an atropine injection for bradycardia during CAS, and 11.7% were administered vasopressors for persistent hypotension after CAS.

Hemodynamic instability associated with CAS can trigger neuronal reactions that cause bradycardia and hypotension and is considered to be caused by catheter manipulations (e.g., balloon dilatation) that stimulate the carotid bulb [8]. Several studies have shown that periprocedural hypotension is more common during CAS, because adventitial baroreceptors and the carotid sinus are manipulated during the procedure [7,11,12,18]. Hemodynamic changes like bradycardia and hypotension that occur during catheter-based carotid artery stenting.
administer prophylaxis IV atropine before balloon dilatation [1], though this was not performed in any of our study subjects. Also, in many cases, vasopressors are required for hypotension that occurs during closed monitoring after CAS. Identifying patients at high-risk of procedure-related hemodynamic instability can result in more aggressive treatment and improve prognosis, and thus, several studies have identified predictors of CAS-related hemodynamic instability [7,11,12, 18].

### 4.1. Carotid bulb involvement of stenosis lesion

Tsurumi, et al. [7] concluded a stenotic lesion involving the common carotid artery and the internal carotid artery (OR 98.38 95 % CI 4.36–2220) was a risk factor for periprocedural hypotension associated with CAS. In addition, they reported that a distance between the carotid bifurcation and the center of the stenotic lesion of < 10 mm (OR 13.66 95 % CI 2.42–77.2) was a risk factor of prolonged periprocedural hypotension. Rubio, et al. [14] reported that a carotid stenotic lesion involving the carotid bifurcation/bulb (OR 4.59 95 % CI 1.1–18.5) was an independent risk factor of hypotension requiring vasopressors after CAS. Nonaka, et al. [21] also reported that a distance between the carotid bifurcation and the center of the stenotic lesion of < 10 mm was a risk factor of prolonged hypotension. Nanto, et al. [1] argued that a distance between the carotid bifurcation and the center of the stenotic lesion of < 10 mm (OR 3.4 95 % CI 1.15–10.1) is an independent risk factor of hypotension post-CAS. Similarly, we found involvement of the carotid bulb is a major risk factor of CAS-associated hemodynamic instability. Operators should be aware of the risk of periprocedural hypotension when performing CAS on such lesions.

### 4.2. Antihypertensive regimens administration before the procedure

Rubio, et al. [14] reported that the administration of more than two antihypertensive regimens before surgery (OR 4.2 95 % CI 1.1–16.0) was an independent risk factor of hypotension requiring vasopressors after CAS and claimed the risk of reflex hypotension after CAS is greater in patients with refractory hypertension requiring two or more antihypertensive regimens. On the other hand, Nanto, et al. [1] reported preprocedural hypertension history was an independent protective factor against hemodynamic instability after CAS (OR 0.26 95 %CI 0.26–0.92). We attribute this inconsistency to methodologic differences. In the present study, the number of antihypertensive regimens received was found to be protective against postprocedural persistent hypertension, but there was no relation observed between number of antihypertensive regimens and the risk of periprocedural bradycardia. We suggest that patients who had received a number of antihypertensive regimens with different action mechanisms were relatively insensitive to physiological changes that induce hypotension. Additional research is required to elucidate the mechanism involved.

### 4.3. Other factors related to hemodynamic instability after CAS

Several studies have been conducted on several other factors related to hemodynamic instability after CAS. Lin, et al. [8] reported an age of ≥78 years (OR 5.25 95 % CI 2.32–15.25) and a low cardiac ejection fraction (25 %) (OR 3.25 95 % CI 0.58–6.58) were risk factors of hemodynamic instability after CAS, and that prior ipsilateral CEA history (OR 0.21 95 % CI 0.12–0.69) protected against hemodynamic depression after CAS. In addition, Tsurumi, et al. [7] reported the absence of a diabetes mellitus (DM) history (OR 16.60 95 % CI 1.55–178) was a risk factor of periprocedural hypotension, and that underlying DM protects against hypotension during balloon inflation and stent deployment due to autonomic functional impairment in DM patients. On the other hand, in the present study, underlying medical conditions including DM did not show any relationship with hemodynamic instability related to CAS.

The effect of degree of calcification of the carotid stenosis lesion on the occurrence of hypotension after CAS is controversial. Tsurumi, et al. [7] reported a lesion fibrous tissue content of > 60 % as determined by virtual histology intravascular ultrasound (Volcano Corporation, Rancho Cordova, California, USA) (OR 12.69 95 % CI 1.30–124) was a risk factor for periprocedural hypotension. Jeon, et al. [22] also concluded the presence of calcified plaque was related to hemodynamic instability after CAS (OR 8.57 95 % CI 1.32–55.62) and argued carotid bulb baroreceptors in patients with large calcified plaque were more directly and severely compressed by the radial force of balloon dilatation or stents and that this caused hypotension [7,14,22]. However, Rubio, et al. [14] reported that degree of calcification of stenotic lesions was not correlated with the occurrence of hypotension requiring vasopressors after CAS (p = 0.68), and a meta-analysis performed by Mylonas, et al. [15] also reported no association between prolonged hemodynamic instability, including hypotension and bradycardia, and calcified plaque. We did not investigate the association between degree of lesion calcification and the occurrence of hemodynamic instability, and recommend further research be conducted on the topic.

Nanto, et al. [1] found the use of proximal protection as a flow reversal technique (OR 3.06 95 % CI 1.09–8.62) was an independent risk factor of hypotension after CAS. However, distal filter protective devices were used in all of our cases, which prevented evaluation.

### 4.4. Limitations

Because this study was conducted using a retrospective study design and a relatively small sample size at a single institution, our results cannot be readily generalized. In particular, it would be inadvisable to assume that taking many different types of antihypertensive regimens necessarily means uncontrolled hypertension, since patients may have been prescribed antihypertensive regimens by several physicians at different institutions before CAS. In addition, since we only investigated the number of types of antihypertensive regimens received, we cannot comment on the different characteristics of antihypertensive regimens. Unlike previous studies, we evaluated risk factors using periprocedural hemodynamic instability and postprocedural persistent hemodynamic instability, and we believe both be considered useful outcome variables for further studies. In particular, because several previous studies have reported different results for the effects of antihypertensive regimens, we suggest a large-scale multicenter prospective study be undertaken to clarify inconsistencies.

### 5. Conclusion

CAS is increasingly being used to treat carotid artery stenosis, but the number of cases associated with hemodynamic instability is also increasing. In a carotid stenotic lesion involves the carotid bulb, the risk of hemodynamic instability, including periprocedural bradycardia and
postprocedural persistent hypotension, associated with CAS may also increase, which cautions that careful evaluation is necessary before procedures. In addition, the intake of various types of antihypertensive regimens before CAS was found to have a protective effect on postprocedural persistent hypotension, but not periprocedural bradycardia.

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CRediT authorship contribution statement

Jongwook Choi: Conceptualization, Methodology, Formal analysis, Investigation, Data curation, Writing - original draft. Ji Yong Lee: Conceptualization, Data curation, Supervision. Kum Whang: Validation. Sungmin Cho: Visualization. Jongyeon Kim: Conceptualization, Writing - review & editing, Project administration.

Declarations of Competing Interest

The authors have no conflict of interest to declare.

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