Prevention of Back Bleeding During Carotid Endarterectomy: Analysis of Clamping Techniques

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OBJECTIVE: **Complete control of back bleeding during carotid endarterectomy (CEA) is important. We investigated the causes of back bleeding during CEA and techniques for the control of bleeding.**

METHODS: **A retrospective review was performed of 214 CEA procedures. We assessed the results of routine preoperative examinations, instruments used for arterial clamping (vessel loop and crude or bulldog clamps), and severity of carotid artery stenosis and arterial wall calcification. The study end point was incomplete control of back bleeding before arteriotomy. Factors associated with back bleeding were identified by univariate analysis. The culprit artery and intraoperative technique used in patients with back bleeding were also determined.**

RESULTS: **Transient back bleeding occurred in 19 CEA procedures (8.9%). Back bleeding was from the ascending pharyngeal artery in 9 cases, common carotid artery in 8 cases, and external carotid artery in 2 cases. Univariate analysis identified the following factors as being related to incomplete control of back bleeding: moderate carotid artery stenosis (20 mm thick) and use of bulldog clamps.**

CONCLUSIONS: **Transient back bleeding during CEA was uncommon, with the risk factors being severe carotid calcification and moderate carotid stenosis. Transient back bleeding was managed by clamping an undetected ascending pharyngeal artery or by additional clamping of the common carotid or external carotid artery. The vessel loop and crude were superior to the bulldog clamp for clamping the carotid artery.**

INTRODUCTION

During carotid endarterectomy (CEA), complete control of blood flow and prevention of back bleeding are very important because back bleeding interferes with smooth performance of surgery. It has been reported that back bleeding often arises from an ascending pharyngeal artery (AphA) with an anomalous origin or is caused by inadequate clamping.[1-7] We investigated the reasons for incomplete control of back bleeding and the effectiveness of our operative technique and arterial clamping instruments for CEA. To our knowledge, this is the first report on arterial clamping methods and blood flow control during CEA.

MATERIALS AND METHODS

Patients

From April 2010 to December 2018, 214 CEA procedures were performed at our institution. Demographic and clinical data on each patient were recorded in a standardized form, including the age, sex, atherosclerotic risk factors, clinical variables, preoperative medication, and intraoperative variables (Table 1).

Radiographic Evaluations

Patients underwent a standard preoperative diagnostic workup that included obtaining a medical history and performing

Key words
- Atherosclerosis
- Carotid endarterectomy
- Common carotid artery

Abbreviations and Acronyms
AphA: Ascending pharyngeal artery
CCA: Common carotid artery
CEA: Carotid endarterectomy
CI: Confidence interval
ECA: External carotid artery
ICA: Internal carotid artery
MDCT: Multidetector computed tomography
NASCET: North American Symptomatic Carotid Endarterectomy Trial
OR: Odds ratio

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Table 1. Demographic and Clinical Features of the Patients

Variable	Value
CEA procedures	214
Age (years)	76.4 ± 9.4
Male	176
Risk factors	
Hypertension	206
Diabetes	107
Smoking	94
Hyperlipidemia	134
Cardiac disease	64
Chronic kidney disease	93
Pulmonary disease	37
Symptoms	
Symptomatic	154
No symptoms	60
Anticoagulant	
Warfarin	18
DOAC	33
Antiplatelet medication	
Acetylsalicylic acid	185
Clopidogrel	79
Cilostazol	165
Severe calcification artery	42
Severe carotid artery stenosis	76
Operative variables	
Shunt placement	32
Quantity of heparin (IU)	3750 ± 232
ACT highest value (seconds)	288 ± 45
First bulldog clamps using	124

Values are number or mean ± SD.
CEA, carotid endarterectomy; DOAC, direct oral anticoagulant; IU, International Units; ACT, activated clotting time.

neurologic examination, cerebral computed tomography scan and/or magnetic resonance imaging, laboratory tests, electrocardiography, and echocardiography. The severity of carotid stenosis (defined according to the North American Symptomatic Carotid Endarterectomy Trial [NASCET]) and arterial wall calcification were assessed by preoperative multidetector computed tomography (MDCT) scan and MDCT angiography.[8] MDCT scan was performed in all patients with a 64-detector row computed tomography scanner (Revolution CT [General Electronics, Boston, Massachusetts, USA]) and the following parameters: spiral mode gantry rotation, 0.33 seconds; collimation, 32 × 0.6 mm; pitch factor, 1.5; slice thickness, 1.0 mm; reconstruction interval, 0.5 mm; and image acquisition, 120 kVp and 350 mA. Fifty milliliters of nonionic contrast medium (iohexol; Omnipaque 300 [Daiichi Sankyo, Tokyo]) was injected at a flow rate of 3.5 mL/s, followed by 25 mL of saline at the same rate. Images were obtained from the aortic arch to the inferior orbital margin, after which data were transferred to a workstation (Workstation Vincent [Fujifilm, Tokyo, Japan]) for postprocessing, and the extent of carotid plaque calcification was quantified. Patients were divided into 2 groups based on the severity of NASCET stenosis shown by MDCT angiography, including a severe stenosis group (NASCET ≥80%; range, 80%–99%; mean stenosis level, 89% ± 6%) and a moderate stenosis group (NASCET <80%; range, 56%–79%; mean stenosis level, 71% ± 8%). Arterial wall calcification was defined as vessel wall structures that showed hyperdensity on MDCT angiography (density >130 Hounsfield units).[9-11] Severe calcification was defined as any site of arterial wall calcification >20 mm thick in the region from the origin of the common carotid artery (CCA) to the internal carotid artery (ICA) at the skull base (Figure 1). Therefore, calcification of the ICA (e.g., cavernous), external carotid artery (ECA), and aortic arch was excluded.

Operative Techniques

CEA was performed under general anesthesia according to a strict protocol. Routine intraoperative monitoring of the upper arm sensory evoked potential was performed, and selective shunting (Pruitt-Inahara carotid shunt with T-port [LeMaitre Vascular Inc., Burlington, Massachusetts, USA]) was done immediately in patients with sensory evoked potential changes even a little.[12] Patients were administered intravenous unfractionated heparin (3000 U) before clamping of the ICA, and the activated clotting time was maintained at >250 seconds. The CCA and ICA were temporarily clamped, while avoiding atherosclerotic lesions in the ICA. The CCA was clamped by using bulldog clamps or vessel loop and crude (Argon Medical Devices Inc., Athens, Texas, USA) selected randomly, whereas the ICA was clamped with bulldog clamps or Sugita cerebral aneurysm clip (MIZUHO corporation, Tokyo, Japan) and the ECA was clamped by bulldog clamps or vessel loop and crude. In addition, the superior thyroid artery and AphA (possible confirmation before CEA) were clamped by using the Sugita clip. To confirm complete control of back bleeding before arteriotomy, we punctured the CCA with a 27-gauge needle. If blood flowed out of the needle, this was defined as incomplete control of back bleeding (Figure 2). The site of back bleeding was classified as the CCA, ICA, ECA, or AphA, and the procedure and instruments used to achieve complete control of back bleeding were recorded. The carotid cross-clamping time was also measured. After performing endarterectomy, the arterial sutures were covered with polytetrafluoroethylene patches. The study end point was incomplete control of back bleeding. In the patients with incomplete control of back bleeding, we investigated general factors, MDCT angiography findings, intraoperative factors, and clamping instruments.

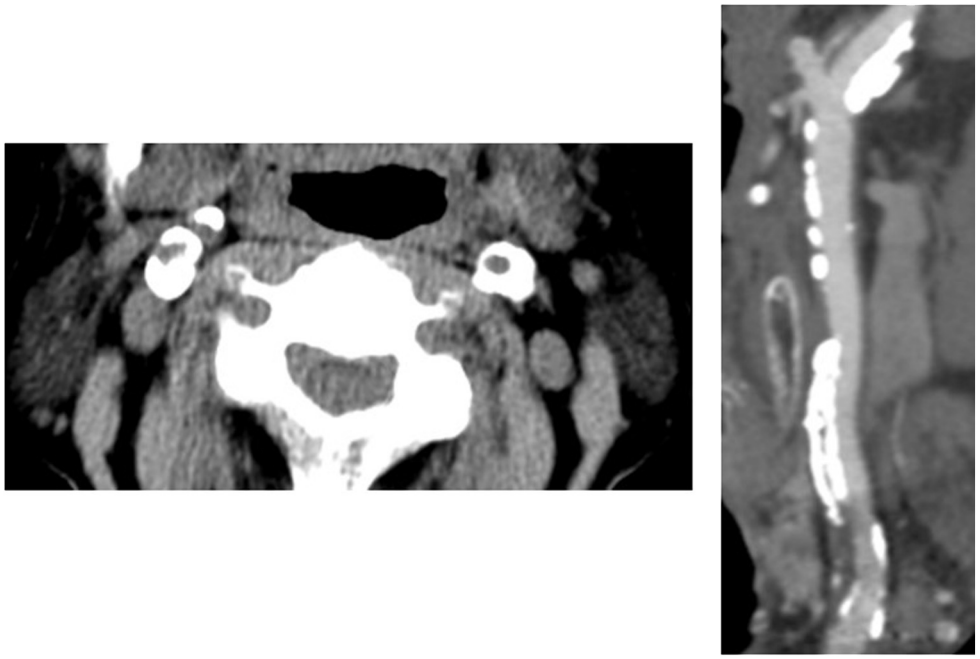

Figure 1. Example of severe calcification, defined as arterial wall calcification >20 mm thick.

Statistical Analysis

Univariate analysis was performed to identify the predictors of incomplete control of back bleeding. Results are expressed as mean ± SD. Continuous variables were compared with Student t test (2-tailed), and categorical variables were compared using Pearson χ^2 test (2-tailed) or Fisher exact test, as appropriate. Statistical significance was accepted at $P \leq 0.05$. The odds ratio (OR) and 95% confidence interval (CI) were calculated for relevant parameters.

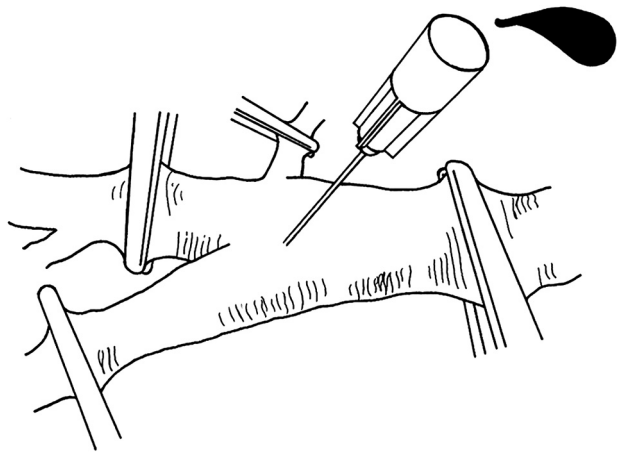

Figure 2. Using the Sugita clip to clamp the anterior pharyngeal artery and control back bleeding.

RESULTS

Outcomes and Complications

During the 30-day postoperative period after CEA, 2 patients developed minor stroke (0.9%). None of the procedures were aborted or incomplete because of technical issues during surgery. Three patients required blood transfusion because they had preoperative anemia because of chronic kidney disease, but there was no need for blood transfusion because of back bleeding.

Preoperative Data

Table 2 summarizes the preoperative demographic data, risk factors, symptoms, antiplatelet agents, anticoagulant therapy, and intraoperative variables.

Results of Back Bleeding

Transient back bleeding was noted in 19 CEA procedures (8.9%), with the culprit artery being the AphA in 9 cases, CCA in 8 cases, and ECA in 2 cases. In the 9 cases of AphA back bleeding, the vessel was too small to be identified by preoperative MDCT angiography. Figure 3 shows use of the Sugita clip for clamping the AphA to control back bleeding. The clip was inserted from behind the ECA to avoid disturbing the CEA procedure, especially the arterial wall suture. When back bleeding was from the CCA, a bulldog clamp was applied first. In 3 cases, another bulldog clamp was added and complete control of back bleeding was achieved. In 5 cases, back bleeding continued after adding another bulldog clamp; therefore, the bulldog clamps were removed and only vessel loop and crude were used. There was back bleeding from the ECA in 2 cases,

Table 2. Univariate Analysis of Incomplete Back Bleeding Control

Characteristics	Odds Ratio	95% CI	*P* Value
Age >75 years	1.82	0.16–4.03	0.53
Male	1.14	0.28–2.05	0.64
Hypertension	1.65	0.08–5.08	0.56
Diabetes	1.34	0.35–6.23	0.34
Smoking	1.24	0.37–5.76	0.42
Hyperlipidemia	0.65	0.32–3.54	0.47
Cardiac disease	1.22	0.14–4.32	0.63
Chronic kidney disease	0.87	0.34–3.24	0.56
Pulmonary disease	0.80	0.22–3.04	0.32
Symptomatic	0.86	0.25–2.33	0.43
Anticoagulant medication	1.39	0.59–4.27	0.39
Antiplatelet medication	1.05	0.65–4.94	0.63
Moderate carotid artery stenosis NASCET <80%	2.93	0.86–8.43	0.05
Sever calcification artery	3.14	1.20–8.24	0.02
Shunt placement	1.14	0.54–8.45	0.47
Maximum ACT (>300 seconds)	1.86	0.36–8.32	0.74
Bulldog clamps using	2.71	1.02–7.21	0.05

CI, confidence interval; NASCET, North American Symptomatic Carotid Endarterectomy Trial; ACT, activating clotting time.

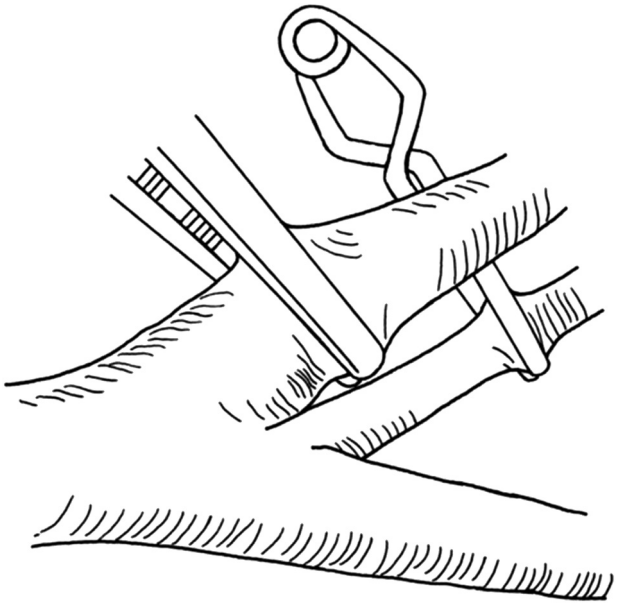

Figure 3. Before arteriotomy, the common carotid artery was punctured using a 27-gauge needle to confirm complete control of back bleeding. If blood was detected via the needle, this was defined as incomplete control.

with one case being managed by applying a bulldog clamp at a different site and vessel loop and crude being used to clamp the ECA in the other case. If back bleeding persisted, we rapidly dissected the carotid bifurcation around the carotid sinus to search for an AphA and clamped it. Patients with moderate carotid stenosis had a 2.9-fold (OR, 2.93; 95% CI, 0.86–8.43; $P = 0.05$) higher risk of incomplete control of back bleeding than those with severe stenosis. In addition, patients with severe calcification had a 3.1-fold (OR, 3.14; 95% CI, 1.20–8.24; $P = 0.02$) higher risk of incomplete control of back bleeding than those without severe calcification. Using a bulldog clamp rather than a vessel loop and crude for CCA clamping was also associated with a higher risk of incomplete control of back bleeding (OR, 2.71; 95% CI, 1.02–7.21; $P = 0.05$). However, other variables were not risk factors of incomplete control of back bleeding, including the maximum anticoagulant time, use of antiplatelet agents, and anticoagulant therapy (Table 2). There was also no statistically significant difference in carotid cross-clamping time (with back bleeding: 53 ± 17 minutes, without back bleeding: 57 ± 18 minutes; $P = 0.35$).

DISCUSSION

Transient Back Bleeding During CEA

During CEA, complete control of blood flow and prevention of back bleeding are very important. This study showed that transient back bleeding occurred in 8.9% of CEA procedures. Although back bleeding was not correlated with mortality, morbidity, carotid cross-clamping time, or major blood loss, incomplete control of back bleeding may interfere with surgery. There were 2 causes of transient back bleeding, which were incomplete occlusion of the main arteries (CCA or ECA) or bleeding from an AphA that was undetectable on MDCT angiography.

AphA

The AphA frequently originates from the posterior surface of the ECA near the bifurcation of the CCA, and anomalous origin of the AphA has previously been reported as an adverse factor for blood flow control,[4,13] If the AphA arises near the origin of the ECA and proximal to the lingual artery, carotid bifurcation, and ICA, it must be identified to prevent troublesome back bleeding during plaque removal.[3,5,6] Hayashi[13] reported that the AphA arose from the carotid bifurcation in 2% of specimens and from the ICA in 2%. Preoperative MDCT angiography is important for evaluation of anomalous small arteries, including the AphA.[3,13] It is usually recommended to avoid circumferential dissection around the carotid sinus to avoid injury to the baroreceptors/chemoreceptors; therefore, the AphA is not always identified.[7,14-16] If unexpected back bleeding occurs during CEA, we should consider an undetected AphA as the possible source. We propose clamping the AphA with a clip applied from behind the ECA to prevent interfering with arterial suture. The AphA typically divides into 2 main branches, which are the pharyngeal and neuromeningeal trunks.

Branches of the pharyngeal trunk run through the submucosal tissue of the pharynx and may anastomose with branches of the internal maxillary artery and ICA system.[7,15] The neuromeningeal trunk gives rise to hypoglossal and jugular divisions, and usually the inferior tympanic artery, which may also be an independent vessel.[3,15] The hypoglossal division supplies the vasa nervorum of the hypoglossal nerve and the high cervical spine, while anastomosing with the vertebral artery system. The jugular division supplies the vasa nervorum of the glossopharyngeal, vagus, and accessory nerves, entering the posterior fossa through the jugular foramen.[7,15] Although there is no evidence of a relationship between lower cranial nerve injury and clamping the AphA or ECA, preservation of the AphA during CEA may be important because it supplies the lower cranial nerves.[5,7,15] We found that patients with severe carotid stenosis (>80% stenosis) had less back bleeding than those with moderate stenosis. There was no back bleeding from the AphA in patients with severe stenosis, suggesting that more extensive plaque (severe stenosis) may occlude this small artery.

Carotid Intimal Calcification

We also found a relationship between transient back bleeding and severe carotid calcification. Intimal calcification is almost invariably an indicator of atherosclerotic disease, and there is a linear relationship between the calcium area and total plaque area. Vascular calcium deposits have recently become a major field of research because calcification increases the atherosclerotic burden and influences clinical outcomes.[17,18] Although the mechanisms leading to calcification remain poorly understood, the presence of apoptotic cells, extracellular matrix, and necrotic core material may promote microcalcification of plaque, with subsequent formation of more extensive calcium deposits.[19] To control arterial blood flow, a clamp needs to exert higher pressure than the blood pressure plus resistance from arterial hardness.[19] Calcified intima and plaque increase the vessel hardness and can lead to incomplete clamping.

Artery Clamping

It should also be recognized that strongly clamping a vessel is inextricably linked to an increased risk of damaging the intima and plaque.[9,20,21] For example, clamping a severely atherosclerotic aorta during vascular surgery should be avoided to prevent intimal injury and exposure of cholesterin crystals.[9,18] Vascular surgeons consider the following 3 points to avoid atheroembolism and damage to atherosclerosis plaque in high-risk patients: 1) selection of a safe arterial perfusion site and method, 2) no touch surgery for the diseased aorta, and 3) debridement of the anastomotic site. These points also apply to CEA.[9,18] Various arterial clamps are available, including bulldog clamps, Satinsky forceps, Debakey forceps, and vessel loop and crude. Our experience suggested that the vessel loop and crude was superior for prevention of back bleeding. A bulldog clamp applies pressure to both sides of an artery, whereas the vessel loop and crude applies circumferential pressure. In an atherosclerotic carotid artery, the intima is irregular and shows uneven hardness; therefore, a single bulldog clamp may be insufficient for clamping the CCA (Figure 4). We should use preoperative MDCT angiography to select an appropriate site and avoid clamping the artery where there is severe calcification. There have been many reports about vessel wall damage during temporary clipping in patients with a cerebral aneurysm.[22-24] The goal of temporary clipping is to block blood flow in the parent artery without causing permanent damage to the vessel wall. However, several studies investigating the effects of temporary aneurysm clips by light microscopic analysis have identified some pathologic changes of the arterial wall because of clip trauma, including endothelial cell damage, endothelial breaks, vascular crater defects, subendothelial platelet clumps, medial damage, and vascular stenosis.[22,23] In addition, intimal thickening can develop because of smooth muscle proliferation, fibrosis, and inflammation at the clip site.[20,22,23] Mitochondrial swelling and dilation of the endoplasmic reticulum have also been noted in vascular cells.[23] Development of arterial wall damage caused by temporary clipping is dependent on the duration of clip application and the closing pressure.[22,23] Sugita et al.[21] reported that arterial damage caused by temporary clipping is less severe when the clip closing pressure is <80 g, but there have been no reports about carotid artery damage after clamping. The closing pressure of our bulldog clamps was 150–170 g.[25] A higher closing pressure stops blood flow more effectively, but also increases the risk of arterial wall injury. Accordingly, we should determine the optimum closing pressure to control back bleeding without damaging the arterial wall.

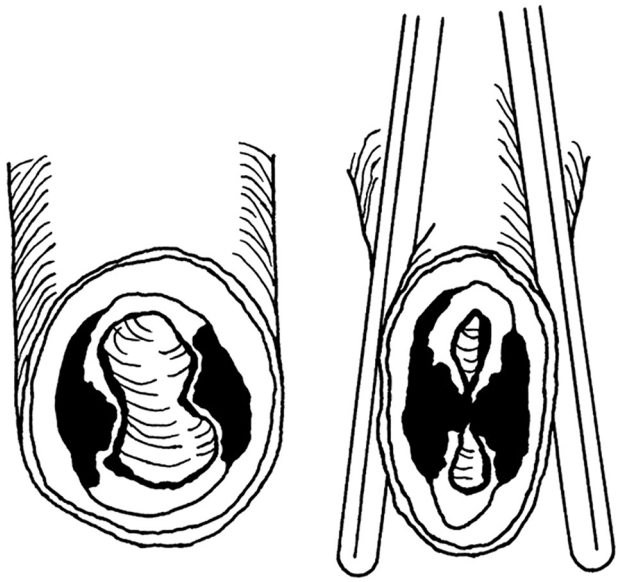

Figure 4. Severe calcification of the carotid intima is irregular and uneven in hardness. A single bulldog clamp may be insufficient for clamping a severely calcified common carotid artery.

CONCLUSIONS

This study showed that transient back bleeding is uncommon during CEA, and can be managed by clamping an undetected AphA or by additional clamping of the CCA or ECA. Severe carotid calcification and moderate carotid stenosis were risk factors for transient back bleeding. The vessel loop and crude were superior to the bulldog clamp for clamping the CCA.

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