

Altered blood pressure responses of carotid endarterectomy in patients with contralateral carotid artery lesion

Kontralateral karotis arter lezyonlu hastalarda karotis endarterektominin değişmiş kan basıncı yanıtları

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ABSTRACT

Background: In this study, we evaluated altered blood pressure responses within the first three days following carotid endarterectomy in patients with contralateral carotid artery lesion.

Methods: A total of 111 patients (52 males, 59 females; mean age 67.7±8.6 years; range 46 to 83 years) who underwent carotid endarterectomy between March 2009 and November 2012 were retrospectively analyzed. The patients were divided into two groups: those with a patent contralateral carotid artery (group 1, n=65) and those with a contralateral carotid artery stenosis (50% to 99%) or occlusion (group 2, n=44). The changes of the blood pressure compared to the baseline during the postoperative course were analyzed and compared between the groups.

Results: In the postoperative period, group 2 patients had significantly higher systolic, diastolic, and mean arterial blood pressure values, compared to group 1 (p<0.05). During the postoperative period, the need for intravenous antihypertensive drugs was significantly higher in group 2, compared to group 1 (p<0.05). The dose of existing antihypertensive medications during hospitalization increased or additional antihypertensive medication was prescribed in 25 patients (56.8%) in group 2 and 14 patients (21.5%) in group 1 after carotid endarterectomy (p<0.05). In group 2, one patient experienced a neurological complication involving a transient ischemia attack. No significant postoperative neurological, surgical, or cardiac complications developed in any patient in either group.

Conclusion: The present study showed that the patients scheduled for carotid endarterectomy were at high risk for postoperative hypertension in the presence of a stenosis (50 to 99%) or occlusion of the contralateral internal carotid artery.

Keywords: Carotid endarterectomy; contralateral stenosis; occlusion; postoperative hypertension.

ÖZ

Amaç: Bu çalışmada kontralateral karotis arter lezyonu olan hastalarda karotis endarterektomiye takiben ilk üç gün içinde değişmiş kan basıncı yanıtları değerlendirildi.

Çalışma planı: Mart 2009 - Kasım 2012 tarihleri arasında karotis endarterektomi yapılan toplam 111 hasta (52 erkek, 59 kadın; ort. yaş 67.7±8.6 yıl; dağılım 46-83 yıl) retrospektif olarak incelendi. Hastalar iki gruba ayrıldı: kontralateral karotis arter patent olan (grup 1, n=65) ve kontralateral karotis arter darlığı (%50-99) veya tıkanıklığı olan (grup 2, n=44). Ameliyat sonrası izlem sırasında başlangıca kıyasla kan basıncındaki değişiklikler incelendi ve gruplar arasında karşılaştırıldı.

Bulgular: Ameliyat sonrası dönemde, grup 1'e kıyasla, grup 2 hastalarında sistolik, diastolik ve ortalama arteriyel basınç değerleri istatistiksel olarak daha yüksekti (p<0.05). Ameliyat sonrası dönemde intravenöz antihipertansif ilaç ihtiyacı, grup 1'e kıyasla, grup 2'de anlamlı düzeyde daha fazla idi (p<0.05). Karotis endarterektomi sonrası yatış sırasında grup 2'de 25 hastada (%56.8) ve grup 1'de 14 hastada (%21.5) mevcut antihipertansif tedavinin dozu artırıldı veya antihipertansif tedaviye ilaç eklendi (p<0.05). Grup 2'de bir hastada geçici iskemi atak şeklinde nörolojik komplikasyon görüldü. İki grupta da hiçbir hastada ameliyat sonrası anlamlı nörolojik, cerrahi veya kardiyak komplikasyon gelişmedi.

Sonuç: Bu çalışma, karotis endarterektomi planlanan hastaların kontralateral internal karotis arterde darlık (%50-99) veya tıkanma varlığında ameliyat sonrası hipertansiyon açısından yüksek riskli olduğunu gösterdi.

Anahtar sözcükler: Karotis endarterektomi; kontralateral darlık; tıkanma; ameliyat sonrası hipertansiyon.



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Carotid artery stenosis is an atherosclerotic disease which usually affects the carotid artery bifurcation and internal carotid artery (ICA) and also encompasses the carotid baroreceptors an important anatomic structure in this area.^[1,2] Baroreceptors are one of the main components of the physiological blood pressure control mechanism, which play an active role in the autoregulation of the blood flow.^[1] Therefore, carotid surgical procedures have the potential to affect the baroreceptors.^[2]

The well-defined conventional and eversion carotid endarterectomy (C-CEA, E-CEA) techniques used in the carotid artery stenosis treatment often reduce the physiological baroreflex reserve, and, therefore, disrupt the blood pressure homeostasis.^[3-6] In addition, the presence of contralateral carotid artery stenosis is also one of the important variables which affects the blood pressure homeostasis during carotid artery surgery.^[2] In this study, we evaluated altered blood pressure responses within the first three days following carotid endarterectomy in patients with contralateral carotid artery lesion.

PATIENTS AND METHODS

Between March 2009 and November 2012, a total of 182 patients who underwent CEA for carotid artery stenosis were screened for the evaluation of early postoperative blood pressure changes and responses. The exclusion criteria were as follows: redo CEA, CEA simultaneous with coronary artery bypass grafting, peripheral vascular surgery or valve surgery, presence of a short interval between symptomatic neurological event and the date of surgery (within two weeks), surgery in the carotid area for any reason, and uncontrolled preoperative hypertension. A total of 111 patients (52 males, 59 females; mean age 67.7 ± 8.6 years; range 46 to 83 years) were included in this retrospective study. The patients were divided into two groups: those with a patent contralateral carotid artery (group 1, $n=65$) and those with a contralateral carotid artery stenosis (50% to 99%) or occlusion (group 2, $n=44$). The choice of the CEA technique depended on the anatomy of the ICA. Eversion CEA technique was used in patients with the high carotid bifurcation or an extensive arterial stenosis (long-segment stenosis) of the ICA and in presence of pathological elongation of the ICA (coiling or kinking), particularly distal smaller caliber internal carotid artery.

Operative procedures were performed under general anesthesia. Cerebral oximetry (near infrared spectroscopy: NIRS) was used to determine the presence of inadequate cerebral perfusion and the

use of shunting during carotid clamping. Stump pressure was considered reliable and cost-effective method of measuring cerebral ischemia during CEA. We adopted a practice of selective shunting based on stump pressure <50 mmHg. Of note, we do not routinely shunt on the basis of the status of the contralateral carotid artery or on the symptom status of the patient. A combination of NIRS and stump pressure was used in our study.

In all our cases, carotid artery systemic pressure was measured by inserting a 22-gauge needle into the common carotid artery (CCA), proximal to the carotid bifurcation and stenosis. Then, the CCA and the external carotid arteries (ECA) were occluded and the carotid systolic, diastolic, and mean stump pressures were recorded after the CCA systolic, diastolic, and mean pressures were recorded. Shunting was only used, if the systolic stump pressure was <50 mmHg. Prior to the induction, the anesthesiologist devoted considerable efforts during the entire period of carotid clamping to keep the systemic systolic pressure close to, or preferably 10 mmHg higher than, the baseline pressure.

The preoperative assessment of carotid artery disease included carotid Duplex ultrasonography. Complex bifurcation diseases with long, multifocal lesions or an angulated ICA, extensive aortic or brachiocephalic trunk plaque, or ring-like heavy calcifications of the carotid bifurcation were confirmed by magnetic resonance angiography. Blood pressure was assessed on the day of admission within the first six hours following surgery, and on the first and third postoperative days. On admission, baseline blood pressures values were recorded using three non-invasive blood pressure measurements on each arm. During the first six hours in the recovery room, post-endarterectomy blood pressure values were recorded at one-hour interval using the intra-arterial blood pressure monitoring. Then, non-invasive blood pressure measurements were performed on each arm three times a day until discharge. For patients who received antihypertensive agents which could potentially affect the blood pressure values, blood pressure measurements were standardized performing one hour after the administration of the antihypertensive agent. Values were presented in mean values. Hypertension was defined when the systolic pressure (SP) exceeded 140 mmHg or the diastolic pressure (DP) exceeded 90 mmHg. Postoperative hypertension (HTN) was defined as elevated systolic pressures >180 mmHg or $>40\%$ increase above normal. Postoperative hypotension

was defined as reduced systolic pressures <90 mmHg or <40% decline below normal requiring pharmacotherapy.

A written informed consent was obtained from each patient. The study protocol was approved by the Katip Çelebi University Atatürk Training and Research Ethics Committee. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Surgical technique

Standard CEA: The CCA, ICA, and ECA were exposed through an oblique incision parallel to the anterior border of the sternocleidomastoid muscle. Manipulation of the carotid body at the carotid bifurcation was avoided. After systemic heparinization, the CCA, ECA, and ICA were clamped. A longitudinal arteriotomy was made in the CCA and extended to the ICA distal to the end of the atherosclerotic plaque. It was followed by a meticulous CCA, ECA, and ICA endarterectomy. Based on the discretion of the surgeon, the ICA intima was removed or a patch was used for closure. In case of complete hemostasis, the incision was closed.

Eversion CEA: The E-CEA technique involved an oblique transection of the ICA from the CCA at the bulb. After division, the ICA appeared redundant; a cephalad incision from the heel of the transected ICA was used to shorten the artery. The ICA was everted over the atheroma core. It is important to remove the most external layers of the media to maintain the eversion of the artery over the end of the atheroma. The endpoint was directly visualized and loose fragments were removed; 6-0 or 7-0 monofilament sutures were placed distally, if necessary.

After completion of the ICA endarterectomy, the arteriotomy was able to be extended to the CCA to facilitate removal of the CCA and ECA plaque. The ICA was, then, tailored and shortened, if necessary for re-anastomosis to the CCA. In case of complete hemostasis, the incision was closed.

Statistical analysis

Data were saved in an electronic database (Microsoft Excel, Redmond). The StatDirect statistical software version 2.7.3 (The StatDirect Ltd, Cheshire, United Kingdom) was used for statistical analysis. Differences among systolic arterial pressure (SAP), diastolic arterial pressure (DAP), and mean arterial pressure (MAP) were calculated using an unpaired t-test. The chi-square test was used to compare the differences between the variables. A two-sided *p* value

was computed. A *p* value of <0.05 was considered statistically significant.

RESULTS

Demographic and clinical characteristics of the patient groups are similar and shown in Table 1. The mean age of the patients was 69.2±9.2 years in group 1 and 66.2±7.9 years in the CEA group 2. Of all patients, 44.6% and 52.2% were males in group 1 and group 2, respectively. Twenty-seven patients in group 1 (84.3%) and 25 patients in group 2 (89.2%) had a normal blood pressure (systolic blood pressure <140 mmHg and diastolic pressure <90 mmHg) on antihypertensive medication. During the first six hours in the recovery room and after step-down to the surgical ward until discharge, the need for intravenous and oral vasodilators in group 2 was significantly higher than in group 1 (*p*<0.05). The dosage of existing antihypertensive medications during hospitalization was increased or additional antihypertensive medication was prescribed in 25 patients (56.8%) in group 2 and 17 patients (26.1%) in group 1 after CEA (*p*<0.05). The preoperative baseline arterial pressures were the same in both groups and changes in postoperative arterial pressures are shown in Table 2 and Figure 1.

There were significant differences in all preoperative and postoperative arterial pressures (SAP, DAP, and MAP) after CEA between the two groups (Table 2, Figure 1). In group 1, neurological complications were observed only in one patient in the form of a transient ischemic attack. None of the patients with postoperative HTN requiring pharmacological treatment had a cardiac complication. The other complications are summarized in Table 1. There was no difference in the length of hospitalization of patients in either group.

DISCUSSION

Postoperative blood pressure alteration immediately following CEA is a well-known clinical manifestation.^[6,10] Postoperative hypertension is common after CEA with a prevalence in 11% to 56% of patients with 40% or more patients requiring specific therapeutic intervention.^[6,11,12] Several studies have demonstrated that hypertension caused by baroreflex dysfunction is associated with higher levels of cardiovascular and neurological mortality and morbidity.^[1-3,13] Untreated hypertension is often considered to be one of the independent risk factors for poor outcome following CEA. While certain studies describe a positive relation between perioperative hypertension and the risk of poor neurological outcome or death after CEA, other studies have not

Table 1. Demographic and clinical characteristics of patients undergoing carotid endarterectomy

	Group 1 (n=65)			Group 2 (n=44)			p
	n	%	Mean±SD	n	%	Mean±SD	
Age (year)			69.2±9.2			66.2±7.9	NS
Gender							
Male	29	44.6		23	52.2		NS
Body surface area (m ²)		25.2			26.1		NS
Surgical site (left)	25	78.1		19	67.8		NS
Surgical technique							
Eversion carotid endarterectomy	35			28			NS
Conventional carotid endarterectomy	30			16			0.04
Hypertension	27	84.3		25	89.2		NS
Coronary artery disease	9	28.1		10	35.7		NS
Diabetes mellitus	12	37.5		12	42.8		NS
Hyperlipoproteinemia	25	78.1		24	85.7		NS
Peripheral artery disease	3	9.3		2	7.1		NS
Chronic obstructive pulmonary disease	4	12.5		3	10.7		NS
Nicotine use	15	46.8		14	50		NS
Alcohol use	5	15.6		6	21.4		NS
Stenosis			82.1±6.8			76.8±5.5	0.06
Contralateral stenosis (%)							
50-69	-	-		27	61.3		
70-99	-	-		12	27.4		
Contralateral occlusion	-	-		5	11.3		
On antihypertensive medication	28	87.5		25	89.2		NS
Complications							
Mortality	0			0			NS
Stroke	1			0			NS
Dysesthesia wound areas	8			4			0.03
Cranial nerve injury	1			0			NS
Neck hematoma	2			1			NS
Shunting	8	12.3		5	11.3		NS

SD: Standard deviation; NS: Non-significant; Group 1: Contralateral internal carotid artery lesion (defined as more than 50% stenosis or occlusion); Group 2: Contralateral carotid artery patent.

found such a relationship.^[14,15] Due to the current discussions and uncertainty concerning the effect of preoperative and postoperative hypertension on postoperative outcomes, patients with uncontrolled preoperative hypertension were excluded.

Previous studies have also demonstrated that atheromas located in the carotid sinus area may impair the baroreceptors sensitivity, and, eventually, result in perioperative and postoperative hypertension.^[1,3,16,17] A several variety of causes described in numerous studies as factors which significantly contribute to the development of hypertension include the severity and side of carotid stenosis; prior ipsilateral hemispheric neurological symptoms (stroke or TIA); the presence of contralateral disease; history of previous ipsilateral or contralateral carotid surgery; and the surgical techniques being used.^[6,16,18-21]

Furthermore, several studies have defined preoperative neurological deficits as independent predictors of hypertension following CEA.^[5,6,22] Hypertension is common following ischemic stroke; however, target values of the arterial pressure following stroke still remain a matter of debate.^[23] Increased perioperative arterial pressure lability may be also observed in patients who have recently had TIA.^[2] As recent stroke is one of the common risk factors for adverse neurological outcomes following CEA, patients who are at a higher risk will require urgent surgery, along with the fact that there will be less time to check their other risk factors, such as hypertension.^[2] As a result, the risk of delaying surgery may exceed the benefits gained from delaying surgery, until an acceptable arterial pressure is achieved. Therefore, the exclusion criteria, as

Table 2. Mean arterial pressures at baseline and postoperatively

	Preoperative		Postoperative 0-6 h		Postoperative 6-24 h		Postoperative 72 h	
	Median	IQR	Median	IQR	Median	IQR	Median	IQR
SAP								
Group 1	133.42	101.32-162.43	113.78	96.23-135.98	125.56	103.43-158.71	124.94	106.18-147.82
Group 2	130.95	105.17-155.33	129.45	102.23-152.98	137.66	108.43-162.71	135.97	106.78-160.22
<i>p</i>		NS		0.01		0.01		0.02
DAP								
Group 1	75.84	59.76-86.17	66.74	55.08-80.14	71.86	54.96-85.82	72.91	58.78-87.44
Group 2	72.55	55.56-84.27	70.14	53.68-81.93	79.90	62.68-89.88	77.07	60.04-92.25
<i>p</i>		NS		NS		0.03		0.05
MAP								
Group 1	94.53	79.29-103.17	86.16	78.09-100.76	90.01	75.06-103.08	91.27	70.09-105.98
Group 2	90.47	77.11-100.34	90.92	80.36-104.55	98.05	82.17-111.18	96.47	70.09-110.88
<i>p</i>		NS		0.04		0.02		0.03

IQR: Interquartile range; SAP: Systolic arterial pressure; DAP: Diastolic arterial pressure; MAP: Mean arterial pressure.

discussed in the present study, were considered to be the presence of a short interval (within two weeks) between the symptomatic neurological event and the date of surgery.

Several studies have investigated technique-related blood pressure effects.^[1,3,16,24,25] Eversion CEA is associated with a higher incidence of postoperative hypertension, higher levels of vasodilator use, and lower levels of vasopressor use after surgery compared to standard longitudinal endarterectomy with or without patch angioplasty.^[1,3] This finding is most likely to be attributed to the baroreceptor apparatus

and the almost certain need for carotid sinus nerve transection using the eversion technique.^[3] On the other hand, increased carotid bulb diameters in patch angioplasty, following C-CEA, may cause an increased wall tension, which is at the same ratio as that of the intraluminal arterial pressure.^[3]

In this study, the presence of these important variables was excluded as a possible risk factor for postoperative hypertension, while the presence of lesions of the contralateral carotid artery was identified as a significant risk factor for postoperative hypertension. The fact that the presence of contralateral

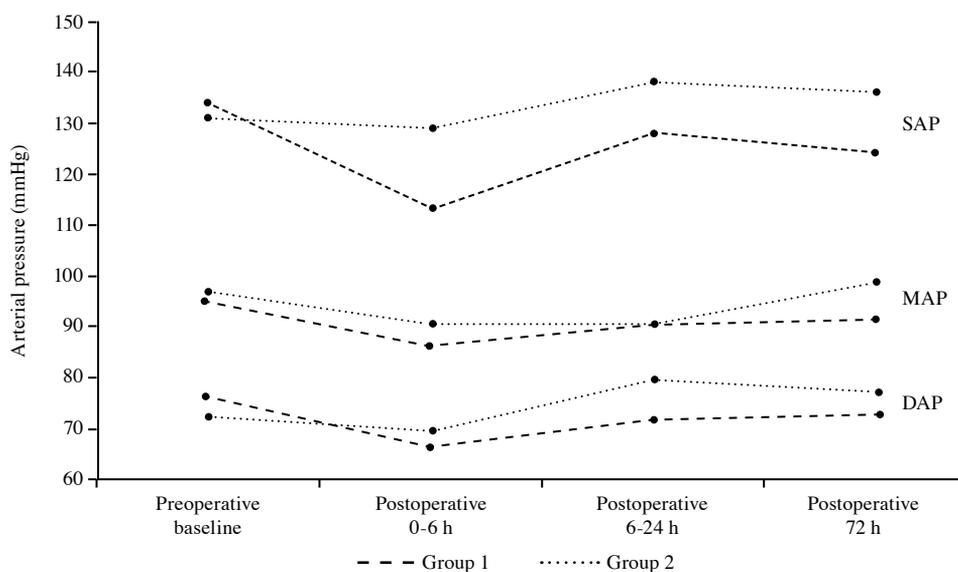


Figure 1. Systolic, diastolic, and mean arterial pressures change from baseline at 72 h after surgery.

SAP: Systolic arterial pressure; MAP: Mean arterial pressure; DAP: Diastolic arterial pressure.

stenosis, which is independent from many of the other contributors to the development of hypertension, is associated with the increased sympathetic activity during the early perioperative period, and is most likely to be attributed to an atheroma in the carotid sinus region related to baroreceptor impairment. Postoperative patients, who are at risk of contralateral stenosis, are clinically more prone to blood pressure derailment than those who are not. The former may also require a closer monitoring of blood pressure.

Surgery to the carotid arteries often results in baroreceptor impairment.^[1,3,17,18] Surgical removal of a carotid plaque partially disrupts the baroreceptor activity, immediately leading to hypertension, as well as increased instability in the arterial pressure.^[17,18] This effect, which may continue for several hours or days following surgery,^[17] may result from the stripping of sensory nerve endings from the arterial lumen.^[17] Patients with an advanced contralateral carotid atheroma tend to have a higher incidence of both intraoperative and postoperative hypertension episodes than patients with normal contralateral carotid arteries.^[16,18] This is mainly due to the dysfunction in the bilateral baroreceptor and the reduced baroreflex reserve.^[18] In addition, baroreceptor insensitivity has also been observed among hypertensive patients.^[11,12] Even after unilateral CEA, baroreflex failure syndrome can be a potential complication for hypertensive patients with severe bilateral atherosclerotic lesions.^[26] Although there are numerous risk factors associated with the development of postoperative hypertension, it is important to exercise particular caution during the dissection of the CCA in patients with bilateral baroreceptor dysfunction and reduced baroreceptor reserve in order to avoid damaging the vagus nerve and the carotid sinus and also to prevent carotid baroreceptor dysfunction.

Postoperative hypertension is usually transient and peaks within the first few hours after surgery and the pathophysiology of this usually episodic hypertension might be related to induce abnormalities of carotid baroreceptor sensitivity.^[2] The results of the current study showed that the baroreflex dysfunction decreased significantly on postoperative day one and remained noticeably lower on postoperative day three; however, on postoperative day three, the baroreflex dysfunction also seemed to show a gradual trend (or increase) towards recovery.^[26] The transience of the reduced baroreflex dysfunction might be associated with the recovery of the baroreflex dysfunction through the baroreflex apparatus located on the contralateral side,

and the aortic arch a compensatory mechanisms, which may require several days for adaptation.^[26]

In this study, SAP, MAP and DAP were statistically significantly higher in the contralateral lesion group, compared to the patent contralateral carotid artery. In other words, we observed that the presence of a contralateral carotid artery lesion contributed to the baroreflex dysfunction and its associated postoperative hypertension. In addition, while a continuous and gradual increase was observed in the early postoperative blood pressure following endarterectomy, this blood pressure values began to descend towards to baseline values on postoperative day four.

On the other hand, our study has some limitations. The main limitations were small sample size and the retrospective design of the study. In addition, during follow-up, the baroreceptor sensitivity was unable to be evaluated. Despite these drawbacks, after carotid endarterectomy, we believe that our findings are highly relevant to development of postoperative hypertension in presence of contralateral carotid lesion.

In conclusion, the present study demonstrates that compared to the presence of contralateral carotid artery stenosis, this pathology is associated with increased postoperative hypertension during the early perioperative period, mainly because of the loss of the baroreceptor reflex due to the presence of atheroma in the carotid sinus region. Considering that hypertension following carotid endarterectomy is multi-factorial and that patients with this condition have a higher risk of complications due to uncontrolled hypertension, the physician's goal with such patients must be to analyze the relevant preoperative risk factors in detail and to bring the hypertension under control in a reliable and effective manner.

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REFERENCES

1. Demirel S, Attigah N, Bruijnen H, Macek L, Hakimi M, Able T, et al. Changes in baroreceptor sensitivity after eversion carotid endarterectomy. *J Vasc Surg* 2012;55:1322-8.
2. Stoneham MD, Thompson JP. Arterial pressure management and carotid endarterectomy. *Br J Anaesth* 2009;102:442-52.
3. Demirel S, Macek L, Bruijnen H, Hakimi M, Böckler D, Attigah N. Eversion carotid endarterectomy is associated with decreased baroreceptor sensitivity compared to

- the conventional technique. *Eur J Vasc Endovasc Surg* 2012;44:1-8.
4. Hans SS, Glover JL. The relationship of cardiac and neurological complications to blood pressure changes following carotid endarterectomy. *Am Surg* 1995;61:356-9.
 5. Towne JB, Bernhard VM. The relationship of postoperative hypertension to complications following carotid endarterectomy. *Surgery* 1980;88:575-80.
 6. Wong JH, Findlay JM, Suarez-Almazor ME. Hemodynamic instability after carotid endarterectomy: risk factors and associations with operative complications. *Neurosurgery* 1997;41:35-41.
 7. Aksun M, Girgin S, Kuru V, Sencan A, Yılık L, Aran G, et al. Cerebral oximetry monitoring method for the evaluation of the need of shunt placement during carotid endarterectomy. *Turk Gogus Kalp Dama* 2013;21:1152-5.
 8. Bowyer MW, Zierold D, Loftus JP, Egan JC, Inglis KJ, Halow KD. Carotid endarterectomy: a comparison of regional versus general anesthesia in 500 operations. *Ann Vasc Surg* 2000;14:145-51.
 9. Chiriano J, Abou-Zamzam AM Jr, Nguyen K, Molkara AM, Zhang WW, Bianchi C, et al. Preoperative carotid duplex findings predict carotid stump pressures during endarterectomy in symptomatic but not asymptomatic patients. *Ann Vasc Surg* 2010;24:1038-44.
 10. Cafferata HT, Merchant RF Jr, DePalma RG. Avoidance of postcarotid endarterectomy hypertension. *Ann Surg* 1982;196:465-72.
 11. Tu JV, Wang H, Bowyer B, Green L, Fang J, Kucey D. Risk factors for death or stroke after carotid endarterectomy: observations from the Ontario Carotid Endarterectomy Registry. *Stroke* 2003;34:2568-73.
 12. Halm EA, Hannan EL, Rojas M, Tuhim S, Riles TS, Rockman CB, et al. Clinical and operative predictors of outcomes of carotid endarterectomy. *J Vasc Surg* 2005;42:420-8.
 13. Taurino M, Filippi F, Persiani F, Tirrotti C, Dito R, Brancadoro D, et al. Hemodynamic changes in Chevalier eversion versus conventional carotid endarterectomy. *Eur J Vasc Endovasc Surg* 2014;48:514-20.
 14. Bond R, Narayan SK, Rothwell PM, Warlow CP. Clinical and radiographic risk factors for operative stroke and death in the European carotid surgery trial. *Eur J Vasc Endovasc Surg* 2002;23:108-16.
 15. Rothwell PM, Slattery J, Warlow CP. Clinical and angiographic predictors of stroke and death from carotid endarterectomy: systematic review. *BMJ* 1997;315:1571-7.
 16. Mehta M, Rahmani O, Dietzek AM, Mecnas J, Scher LA, Friedman SG, et al. Eversion technique increases the risk for post-carotid endarterectomy hypertension. *J Vasc Surg* 2001;34:839-45.
 17. Sigauco-Roussel D, Evans DH, Naylor AR, Panerai RB, London NL, Bell P, et al. Deterioration in carotid baroreflex during carotid endarterectomy. *J Vasc Surg* 2002;36:793-8.
 18. Nouraei SA, Al-Rawi PG, Sigauco-Roussel D, Giussani DA, Gaunt ME. Carotid endarterectomy impairs blood pressure homeostasis by reducing the physiologic baroreflex reserve. *J Vasc Surg* 2005;41:631-7.
 19. Azuma Y, Imai K, Oda K, Niwa F, Makino M, Oshima F. Case of baroreflex failure after bilateral revascularization of the cervical carotid artery. *Rinsho Shinkeigaku* 2007;47:657-61. [Abstract]
 20. Rothwell PM, Warlow CP. Timing of TIAs preceding stroke: time window for prevention is very short. *Neurology* 2005;64:817-20.
 21. Tang TY, Walsh SR, Gillard JH, Varty K, Boyle JR, Gaunt ME. Carotid sinus nerve blockade to reduce blood pressure instability following carotid endarterectomy: a systematic review and meta-analysis. *Eur J Vasc Endovasc Surg* 2007;34:304-11.
 22. Robinson TG, Dawson SL, Eames PJ, Panerai RB, Potter JF. Cardiac baroreceptor sensitivity predicts long-term outcome after acute ischemic stroke. *Stroke* 2003;34:705-12.
 23. Robinson TG, Potter JF. Blood pressure in acute stroke. *Age Ageing* 2004;33:6-12.
 24. Ballotta E, Da Giau G, Saladini M, Abbruzzese E, Renon L, Toniato A. Carotid endarterectomy with patch closure versus carotid eversion endarterectomy and reimplantation: a prospective randomized study. *Surgery* 1999;125:271-9.
 25. Robertson D, Hollister AS, Biaggioni I, Netteville JL, Mosqueda-Garcia R, Robertson RM. The diagnosis and treatment of baroreflex failure. *N Engl J Med* 1993;329:1449-55.
 26. Demirel S, Bruijnen H, Attigah N, Hakimi M, Böckler D. The effect of eversion and conventional-patch technique in carotid surgery on postoperative hypertension. *J Vasc Surg* 2011;54:80-6.