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Hemodynamic instability after extracranial carotid stenting

G. Pappadà[1], **E. Beghi**[3], **R. Marina**[2], **E. Agostoni**[3], **C. Cesana**[1], **F. Legnani**[1], **M. Parolin**[1], **D. Petri**[4], and **E. P. Sganzerla**[1]

[1] Department of Neurosurgery, University of Milano-Bicocca, Ospedale San Gerardo, Monza-Milan, Italy
[2] Section of Neuroradiology, Ospedale San Gerardo, Monza-Milan, Italy
[3] Department of Neurology, University of Milano-Bicocca, Ospedale San Gerardo, Monza-Milan, Italy
[4] Department of Cardiology, Ospedale San Gerardo, Monza-Milan, Italy

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Summary

Objective. Hemodynamic instability (hypertension, hypotension and bradycardia) is a well-known complication of carotid endarterectomy. Carotid angioplasty and stenting (CAS) is becoming a valuable alternative treatment for patients with severe carotid stenosis and increased surgical risk. CAS implies instrumentation of the carotid bulb, so baroceptor dysfunction may provoke hemodynamic instability. The aim of this work was to calculate the incidence of this complication and to detect factors to predict it.

Methods. Medical records and angiograms of 51 consecutive patients submitted to CAS for severe atherosclerotic stenosis (40 cases) or post-surgical restenosis (11 cases) were retrospectively reviewed in order to detect the occurrence of intra- and post-procedural hypertension (systolic blood pressure >160 mmHg), hypotension (systolic blood pressure <90 mmHg) and bradycardia (heart rate <60 beats/min). The relationship between clinical, procedural and angiographic factors and the occurrence of hemodynamic instability was assessed with univariate and multivariate analysis (logistic regression).

Results. Transient mild systolic post-procedural hypertension occurred in five cases (10%); preprocedural hypertension, asymptomatic stenosis and ipsilateral post-surgical restenosis predicted this. Hypotension with bradycardia also occurred in five cases (10%), one with neurological sequelae. Transient periprocedural bradycardia occurred in 19 cases (37%). Severe bradycardia without hypotension arose in one case only. Factors predicting post-procedural hypotension included the presence of a fibrous plaque and the ratio between the pre- and post-stenting diameter of the internal carotid artery. Peri-procedural bradycardia predicted post-procedural bradycardia. None of these factors were confirmed by multivariate analysis as a significant prognostic predictor.

Conclusion. Mild systolic hypertension may occur after CAS, but is resolved by medical treatment. Prolonged hypotension and bradycardia may also arise and this can be dangerous because it may cause neurological deterioration due to hypoperfusion. These complications cannot be predicted by clinical, procedural, and angiographic factors.

Keywords: Carotid stenting; carotid sinus; hypotension; bradycardia; hypertension.

Introduction

Hemodynamic instability, consisting of hypertension, hypotension and bradycardia, after carotid endarterectomy (CEA) is well-known [3, 8, 27, 29, 34]. Dysfunction of adventitial baroceptors in the endarterectomized artery may lead to acute hypertension during the early post-operative period [11, 27, 34]. Changes of metabolic factors such as cerebral renin and vasopressin may also play a role [27, 28, 34]. Hypotension is another potential complication [29] because removal of the atheromatous plaque may increase the compliance and stretch of the arterial wall, increasing firing from adventitial baroceptors and the carotid sinus [3, 29, 31, 34].

Carotid angioplasty and stenting (CAS) is increasingly undertaken to treat recurrent carotid artery stenosis [9] and in symptomatic patients suffering from severe stenosis, with an increased surgical risk because of advanced age, comorbidities, contralateral occlusion, or bifurcation above C2 [15, 16, 18, 26, 30] CAS involves instrumentation of adventitial baroceptors and the carotid sinus, which may lead to severe hemodynamic instability. The aim of this study was to calculate the incidence of this complication and detect factors that may predict it.

Clinical material and methods

The study population comprised 51 consecutive patients aged 64 to 82 years (mean 73) who underwent CAS at the Department of Neurosciences in Monza hospital from January 1997 to January 2003. Medical

records and angiograms were retrospectively reviewed to find the prevalence of hypertension (systolic pressure >160 mmHg), hypotension (systolic pressure <90 mmHg) and bradycardia (<60 heart beats/min). Forty patients had atherosclerotic stenosis of the carotid bifurcation. Carotid stenosis was >70% (according to NASCET criteria) [19] in all but four cases, who had 50–60% stenosis with large type C ulcers. There were 33 symptomatic and 8 asymptomatic patients. All were considered at high risk for surgery because of advanced age, associated intracranial aneurysm, bifurcation above C2, previous oncological neck surgery, contralateral carotid occlusion, or important comorbidities. All symptomatic patients were in stable neurological conditions (grade 0 according to the Rankin scale). Asymptomatic patients underwent CAS because of contralateral carotid occlusion (four cases), previous contralateral hemispheric and left cerebellar ischemia with left vertebral occlusion and left posterior cerebral artery arising from the carotid artery on the side of the stenosis (one case), or coronary artery disease scheduled for myocardial revascularization (three cases). The sample also included 11 cases with postsurgical tight stenosis (>70%).

All patients underwent eco-color-Doppler at our institution. Carotid plaques were classified as fibro-lipidic, fibrous and fibro-calcific. Plaques with an extensive calcific content were excluded from endovascular treatment. All patients were evaluated by a neurologist, a cardiologist, a neurosurgeon and a neuroradiologist according to our institutional protocol [20, 21]. Each patient gave informed consent for angiography and possible endovascular treatment. All arteriographies and endovascular procedures were performed by the transfemoral route under local anesthesia, with light conscious sedation with remiphentanil hydrochloride and/or propofol in three cases only.

The mean degree of stenosis was 78% (SD ±10, range 50–99%). Patients with atherosclerotic stenosis had 27 concentric and 14 asymmetric stenoses. The length of the plaques ranged from 1.1 to 3.2 cm. On angiography the surface of the plaque was smooth in 10 cases, irregular in 15, with small ulcers in 12, and large ulcers in 4 cases. All restenoses compromised 70% or more of the lumen. Antiplatelet drugs (ticlopidine 250–500 mg/day or aspirin 300 mg/day) were not withdrawn. Patients treated with anti-hypertensive medications received their morning doses before surgery. During baseline angiography and endovascular treatment, heart and respiratory rates were monitored throughout the procedure. At the beginning of the procedure all patients were in normal hemodynamic conditions. Blood pressure was measured at 5-min intervals in one arm (usually the left) by an automated-cuff-inflation sphygmomanometer (Datex AS/3). After diagnostic angiography and before the endovascular procedure a periprocedural bolus of 5000 U of heparin was given in order to achieve an activated clotting time of 250/300 seconds. Atropine (0.5 mg i.v.) was also injected. The same neuroradiologist (R.M.) performed the endovascular procedures in all cases.

Tight stenoses were first crossed with microcatheters and microwires using a road mapping technique. After predilatation a stent was advanced over the guide-wire using a stent delivery system. Self-expandable devices were used in all cases (Wallstent or Acculink in more recent cases). Balloon postdilatation was then performed in order to mold residual stenosis and to obtain the so-called embedding of the stent in the vessel wall. Patients were then transferred to the neurosurgical intensive care unit. Heart and respiratory rates were monitored and blood pressure was measured at 15-min intervals from the same arm (Datex AS/3). If patients were hemodynamically stable, monitoring was discontinued the day after the procedure. Unstable patients continued monitoring until they became hemodynamically stable.

The patients'medical records were reviewed to detect any episodes of hypertension (systolic blood pressure >160 mmHg), hypotension (systolic blood pressure <90 mmHg) and bradycardia (heart rate <60 beats/min). To analyze factors associated with the development of post-procedural hemodynamic instability, the following clinical variables were taken into account: age, sex, history of angina or myocardial infarct (MI), coronary by-pass or angioplasty, dysrhythmias and atrio-ventricular blocks, hypertension (including anti-hypertensive treatment), heart failure, pulmonary disease, any neurological symptoms related to the ipsilateral or contralateral carotid, previous ipsilateral or contralateral endarterectomy, and the echographic characteristics of the plaques. Angiographic variables included the degree of stenosis (measured according to Nascet criteria) [19], the length of the stenosis, the involvement of the common carotid artery, and the lateral or medial wall of the carotid bulb. Procedural variables were the diameter of the largest balloon employed, the pressure and duration of pre and post dilatation, and the type, length and diameter of the stent. Finally, the change in the vessel diameter after stenting was also assessed, measured as the ratio on lateral angiograms between the unstented and the stented post-stenotic segments.

Statistical analysis

The effects of clinical, periprocedural and angiographic variables were correlated with the occurrence of hypertension, hypotension and bradycardia. Univariate analysis was done on each variable using the chi-square test, Fisher's exact test or Student's test, as appropriate. Logistic regression analysis models were used to assess the independent role of each variable on the occurrence of hypertension and, separately, of hypotension or bradycardia. A p value <0.05 was considered significant.

Results

One or more complications of the stent procedure arose in 11 patients (22%); these included hypertension (5 cases or 10%), hypotension with bradycardia (5 cases or 10%), bradycardia (one case). Transient mild hypertension (180/190 mmHg) was observed in 5 cases during the first hours after CAS. These episodes were all promptly and successfully treated with i.v. clonidine. No adverse cardiac events occurred and the hospital stay was not prolonged. In all cases systolic blood pressure was sometimes >160 mmHg in the 24-hour pre-procedural period. All patients were treated with beta-blockers, ace-inhibitors, diuretics or beta-blockers as polytherapy. Two of these patients suffered from ipsilateral restenosis.

Significant predictors of post-procedural hypertension included history of hypertension, episodes of systolic blood pressure above 160 mmHg, asymptomatic stenosis and ipsilateral restenosis, the latter being of borderline significance (Table 1). However, multivariate analysis did not confirm any of these factors as predicting post-procedural hypertension. No relationship was found for age, sex or any other clinical, angiographic and procedural factor.

Hypotension occurred in five cases (10%), lasted from 2 to 72 hours, and always arose immediately after the release of the stent; in no case was there delayed onset of hypotension. Dopamine infusion followed by oral norepinephrine was necessary in all four cases to raise blood pressure above 100 mmHg. Serial ECG and cardiological evaluation ruled out MI and atrioventricular block.

Table 1. *General characteristics of the sample*

Variable	No.	Total (%)	Post-procedural hypertension (%)	Post-procedural hypotension/bradycardia (%)
Total	51	100.0	9.8	
Sex				
– M	41	80.4	12.2	9.8
– F	10	19.6	–	10.0
Age, year				
– <65	5	10	–	20.0
– 65–74	30	59	16.7	3.3
– >74	16	31	–	18.8
Symptomatic				
– Yes	33	65	3.0	9.1
– No	18	35	22.2*	11.1
Side				
– Right	23	45	–	17.4
– Left	28	55	17.9**	3.6
Risk factors for surgery^				
– None	4	7.8		
– Heart disease	18	35.3	8.3	8.3
– Vertebral-basilar occlusion	1	2.0	–	20.0
– Controlateral carotid occlusion	6	12	–	16.7
– Older age	10	19.6	–	25.0
– Intracranial aneurysm	3	6	–	–
– High carotid bifurcation	2	4	50.0	–
– Previous neck surgery	2	4	–	–
– Other comorbities	1	2	–	–
– Hypertension before surgery	3	6	66.7***	–
Carotid stenosis (%)				
– ≤60	4	8	–	–
– 61–80	15	29	20.0	–
– >80	32	63	6.3	15.6
Source of carotid stenosis				
– Atherosclerosis	40	78	5.0	12.5
– Recurrent stenosis	11	22	27.3	–
Plaque				
– Fibrolipidic	16	31	6.3	–
– Fibrous	23	45	4.3	21.7
– Fibro-calcific	2	4	–	–
Stenosis length (mm)				
– Internal carotid	14	27	–	21.4
– Internal and common carotid	37	73	13.5	5.4
Stenosis type				
– Concentric	42	82	9.5	9.5
– Asymmetric	9	17	11.1	11.1

^ In seven cases more than one risk factor was present.
* $p = 0.047$ (Fisher's exact test); ** $p = 0.056$; *** $p = 0.023$ (Fisher's exact test).

All cases had severe and persistent bradycardia. In one case hypotension was followed by left cerebellar ischemia. Hypertension did not cause neurological deficits. Mean age was 74.6 years in the hypotensive group and 71.1 years in the non-hypotensive group.

As preprocedural antihypertensive therapy, two patients took diuretics or alpha-blockers. A Wall stent was positioned in three patients and a nitinol Acculink stent in two. Factors predicting post-procedural hypotension included the presence of a fibrous plaque and a higher ratio of before-to-after stenting internal carotid artery (ICA) diameter (Table 2). Logistic regression analysis did not confirm any of the above factors as predictors of post-procedural hypotension.

Table 2. *Technical approach*

Variable	No.	Total (%)	Post-procedural hypertension (%)	Post-procedural hypotension/bradycardia (%)
Type of stent (diameter × length, mm)				
– 120	1	2	–	100.0****
– 150	1	2	–	–
– 180	19	37	10.5	10.5
– 210	8	16	25.0	25.0
– 240	15	30	6.7	–
– 280	5	10	–	–
– 320	2	4	–	–
Characteristics of balloon				
– 18	21	42	4.8	9.5
– 21	1	2	–	–
– 24	1	2	–	–
– 36	22	44	13.6	9.1
– 42	4	8	–	25.0
– 48	1	2	100	–
– NS	1			
Pre-dilatation	23	45	8.7	4.3
Post-procedural transient bradycardia	30	59	6.7	13.3
Before/after stenting diameter ratio				
– 0.50	4	8	–	–
– 0.55	10	20	10.0	–
– 0.60	7	14	–	–
– 0.65	13	25	15.4	15.4
– 0.70	12	23	8.3	25.0
– 0.75	3	6	33.3	–
– 0.80	2	4	–	–

**** $p = 0.046$ (Cochran-Mantel-Haenszel chi-square).

Table 3. *Response of blood pressure to before/after stenting ICA diameter ratio*

	Ratio							
	0.50	0.55	0.60	0.65	0.70	0.75	0.80	Total
	N (%)							
BP unchanged	4 (100.0)	10 (100.0)	6 (85.7)	11 (84.6)	8 (66.7)	3 (100.0)	2 (100.0)	44 (86.3)
BP depression	– (–)	– (–)	1 (14.3)	2 (15.4)	4 (33.3)	– (–)	– (–)	7 (13.7)

ICA Internal carotid artery, *BP* Blood pressure, $P = 0.185$ (Cochran-Mantel-Haenszel chi-square).

Finally, transient, mild bradycardia arose concomitantly with predilatation, the stent release or postdilatation in 19 cases (37%).

To verify whether relief of carotid stenosis could be associated with blood pressure depression, the before-to-after stenting ICA diameter ratio was correlated with the response of blood pressure (patients remaining hypertensive or normotensive vs those shifting from hypertension to normal pressure or from normal pressure to hypotension) (Table 3). No significant correlations were detected.

During follow-up patients with hypertension continued to suffer from episodes of systolic pressure above 160 mmHg apparently unresponsive to treatment. Of the five cases with hypotension and bradycardia, four remained normotensive on single-drug antihypertensive treatment instead of their preprocedural polytherapy; the fifth case had hypertension in the fourth postprocedural week, but pressure returned to normal with the treatment schedule taken before stenting.

Discussion

Adventitial baroceptors in the carotid sinus (the dilated segment of the ICA at its origin from the common carotid artery) are stretch receptors. The arterial wall of the

carotid sinus is thinner than in other arteries of the same caliber; especially on the ventro-medial side, where the sinus nerve emerges, the medial layer lacks smooth muscles which are almost completely replaced by elastic fibers. Their impulses travel through the sinus and glossopharyngeal nerves to the tractus solitarius nucleus in the caudal medulla oblongata. Increased firing of these baroceptors inhibits sympathetic neurons in the tractus solitarius nucleus and reduces sympathetic tone in the peripheral blood vessels, leading to a reduction of systemic blood pressure. In contrast, a decrease in sinus activity stimulates sympathetic neurons, leading to a rise in blood pressure. In vivo baroceptors, in the carotid sinus and aortic arch play a key role in short-term regulation of blood pressure in response to abrupt pressure changes due to changes in peripheral resistances, blood volume and cardiac output [4]. Nevertheless, classical experimental studies on isolated carotid sinus demonstrated that if a prolonged and stable pressure distends the carotid sinus, its firing is prolonged, with no signs of fatigue for a long period [5]. Atherosclerosis and hypertension lower the baroceptors' sensitivity because the carotid sinus becomes stiffer and more resistant to deformation [10].

Instrumentation of the carotid sinus segment, as during PTA and stenting, may cause dysfunction of adventitial baroceptors in the arterial segment that is distended and covered by the stent.

Experimental data [2] indicated that angioplasty of the carotid sinus in normotensive and hypertensive dogs can cause chronic hypotension, possibly because of greater compliance and an increase in the diameter of the arterial segment after PTA. Furthermore, the radial force of the stent implies chronic mechanical pressure over the carotid sinus.

Transient and mild bradycardia with lowering of systolic blood pressure is observed in 5–10% of cases after carotid stenting [22] despite prophylaxis with atropine. However, severe hemodynamic instability after CAS has not been assessed in detail. Wholey [33] observed bradycardia requiring temporary pace-maker placement in 11 (9.6%) of 114 patients who underwent CAS, with hypotension requiring i.v. vasopressor in one case. Yadav [35] reported bradycardia in 71% of 107 patients, one patient requiring a permanent pace-maker. Waigand [32] reported sino-atrial block in 40 of 50 patients with severe coronary artery disease during high-pressure dilatation for CAS. Mendelson [17] reported hemodynamic instability in 37% of 19 patients undergoing carotid stenting with auto-expandable devices; in one case severe hypotension was followed by adverse neurological complications. This author defined hypotension as systolic blood pressure $\leq$100 mmHg and bradycardia as heart rate $\leq$60 beats/min. Most of the adverse events occurred during stent post-dilatation. Harrop and co-workers [9] reported on a series of 37 procedures in 33 patients, routinely using temporary venous cardiac pace-makers to prevent bradycardia. Pace-maker activation occurred in 73% of the procedures; in two cases severe hypotension arose despite pace-maker activation. The incidence of bradycardia in these series seems high because of a preponderance of radiation-induced stenoses and post-surgical restenosis with atherosclerotic lesions; furthermore the balloon used for pre and post-dilatation seems to be oversized [13] and non-self-expandable stents were used in many cases. Cases of restenoses had a lower incidence of pace-maker activation. Lopes [14] reported one case of severe hypotension with fatal myocardial infarction among 49 patients with concomitant coronary artery disease submitted to CAS. Qureshi [25], in a multicenter study enrolling 71 cases of carotid stenosis submitted to CAS with nitinol self-expanding stents encountered no hemodynamic instability. Qureshi and co-workers [23, 24] investigated hemodynamic instability in patients undergoing CAS for atherosclerotic stenosis and post-surgical restenosis, and found the incidence of hypotension, bradycardia and hypertension was 22.4, 27.5 and 38.8% respectively. Multivariate analysis indicated that MI predicted hypotension and bradycardia and previous ipsilateral endarterectomy and hypertension predicted post-procedural hypertension. Brooks in 2004 observed 5 (11.6%) cases of marked hypotension and bradycardia among 43 cases submitted to CAS [6].

In a fairly similar series our findings were as follows: hypotension 9.8%, bradycardia 11.8% and hypertension 9.8%. Univariate analysis indicated asymptomatic stenosis, preprocedural hypertension and previous ipsilateral endarterectomy as predicting post-procedural hypertension; the presence of a fibrous plaque and the ratio of before-to-after stenting ICA diameter were factors predicting post-procedural hypotension, and peri-procedural bradycardia predicted post-procedural bradycardia. Nevertheless, we could not confirm these findings as multivariate analysis failed to detect any independent prognostic predictor. However, the higher prevalence of post-procedural hypertension in hypertensive individuals (66.7%) and in those with recurrent stenosis (27.3%) is worth noting. The small numbers in our series are the most likely explanation of our findings.

Hypotension may be caused by pressure on the carotid sinus, stretched by the radial force of the stent. Sinus

physiology means the vagal response leading to bradycardia when systolic pressure is low. Bradycardia arising concomitantly with hypotension is due to the fact that baroceptors read the pressure of the stent against the vessel wall. According to us and others [13], severe hypotension and bradycardia seem to occur less frequently with improved stent technology and the low pressure values used during stent dilatation.

Ischemic coronary artery disease is frequently associated with hypotension after CAS. Alpman *et al.* [1] found increased carotid sinus baroceptor sensitivity in these patients, in whom acute or chronic ischemia may activate the receptors of the atrio-ventricular node, leading to vagal activation or sensitisation and to an exaggerated carotid sinus response. A fibrous plaque and a stiffer vessel wall counteracting the radial force of the stent, were prognostic indicators of post-procedural hypotension and bradycardia.

Hypertension after CAS is related to pre-treatment hypertension and ipsilateral endarterectomy. This may be because of reduced sinus activity caused by post-surgical periarterial fibrosis and loss of compliance of the vessel. In these circumstances CAS probably further reduces sinus sensitivity.

Endovascular treatment for atherosclerotic stenosis of the carotid bifurcation is undoubtedly less invasive than carotid endarterectomy; nevertheless these patients must be carefully evaluated from the cardiovascular standpoint, as is currently done with surgery. Antihypertensive drugs (e.g., alpha-blockers) and premedication with drugs that may induce hypotension should be avoided. These patients do not receive enteral nutrition from the night before the procedure and this may be another factor exacerbating hypovolemia. It is our policy to give intravenous hydration the day before carotid endarterectomy in order to avoid volume depletion [7] and the same policy is adopted when carotid stenting is scheduled. Even slightly elevated systolic pressure is best avoided in the preprocedural days but, if present, it should be controlled, especially in cases of post-surgical restenosis. During and after the procedure, patients must be followed closely in order to permit immediate management of cardiovascular emergencies. Routine admission of CAS patients to an intensive care unit seems unnecessary and a sub-intensive ward is adequate for the majority of cases in our opinion.

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Correspondence: Ettore Beghi, Department of Neurology, Ospedale Nuovo San Gerardo, Via Donizetti 106, 20052 Monza-Milan, Italy. e-mail: beghi@marionegri.it