PRACTICAL ADVICE FOR PHYSICIANS

UDC: 616.133-089-06
https://doi.org/10.2298/VSP170910154R

How to identify risk for cerebral hyperperfusion syndrome after carotid revascularization procedures

Kako identifikovati rizik od nastanka sindroma cerebralne hiperperfuzije nakon procedura karotidne revaskularizacije

Djordje M. Radak*†, Milorad Ševković*, Srdjan Babić*†

**Cardiovascular Institute "Dedinje", *Vascular Surgery Clinic, Belgrade, Serbia;
University of Belgrade, †Faculty of Medicine, Belgrade, Serbia**

Key words:
endarterectomy, carotid; cerebral revascularization; risk factors; hypertension; vascular headaches; cerebrovascular disorders.

Ključne reči:
endarterektomija a.carotis; mozak, revaskularizacija; faktori rizika; hipertenzija; glavobolja, vaskularna; cerebrovaskularni poremećaji.

Introduction

Atherosclerotic lesions of the extracranial part of carotid arteries are one of the most common causes of stroke [1]. Carotid endarterectomy (CEA) and carotid stenting (CAS) are therapeutic options in the prevention of primary and secondary stroke in the patients with significant stenosis of internal carotid arteries (ICA) [2]. Although both of these procedures are considered to be relatively safe, there are certain neurological and non-neurological complications. The more serious one is the cerebral hyperperfusion syndrome (CHS). Albeit rare, CHS is a potentially devastating event that can even be fatal if intracranial hemorrhage (ICH) occurs [3–7]. In this review, we will summarize available data regarding this phenomenon and focus on its pathophysiology, prevention, diagnostics and management.

Definition of hyperperfusion and cerebral hyperperfusion syndrome

First and foremost, we must differentiate between the concept of hyperperfusion and CHS. In general, hyperperfusion occurs when cerebral blood flow (CBF) in the revascularized territory increases by 100%, or more, with respect to the baseline values [8, 9]. However, not every patient with hyperperfusion develops CHS. The term CHS was used to describe the clinical entity consisting of symptoms triad: ipsilateral migraine-like headache, seizure, and transient focal neurologic deficits in the absence of cerebral ischemia in combination with the high post-procedural blood pressure (BP). This was first described by Sundt et al. [10].

Pathophysiology

Although this phenomenon is well described in the relevant literature, not much is known about the pathophysiological mechanisms which lead to CHS. As mentioned before, not all patients with increased CBF develop CHS. In the series conducted by Ogasawara et al. [11], 16.7%–28.6% of the patients with an increase in CBF 100% developed CHS. Also, in some cases with slightly elevated CBF, CHS can be developed [11, 12]. That leads to the conclusion that other factors play a role in the occurrence of CHS.

All authors agree that two interlinked and synergized mechanisms lead to increased CBF; first, impaired cerebral autoregulation, and second, the increased postprocedural BP [13, 14].

The main autoregulatory mechanism is the cerebrovascular reactivity (CVR), the ability of the arterioles to constrict or dilate in response to the alterations of blood flow, or to other stimuli (i.e., hypocapnia) [13]. In order to compensate the reduced blood flow to the brain in the patients with severe ICA, stenosis arteriolae remain in the state of maximal dilation to maintain the sufficient cerebral blood supply.

The severity of CVR impairment is likely due to several different factors – a degree of ipsilateral and contralateral ICA stenosis, an incomplete circle of Willis and insufficient collateral flow [3, 15, 16]. However, the syndrome was described even in the patients without contralateral lesions, thereby gi-

Correspondence to: Milorad Sevković, Cardiovascular Institute "Dedinje", Vascular Surgery Clinic, Heroja Milana Tepica 1 street, 11 000 Belgrade, Serbia. E-mail: dr_msevkovic@yahoo.com

ving even more significance to the disturbed autoregulation mechanisms that can develop in the region of ipsilateral stenosis even without severe contralateral ICA lesion. After CEA, the increased nitric oxide levels during clamping of ICA and increased oxygen-derived free radicals produced during the restoration of the perfusion pressure are involved in the endothelium dysfunction and the deterioration of autoregulatory mechanisms [17]. Besides, several studies have demonstrated significant elevations in malondialdehyde, diene conjugates, or lipoperoxides, the products of free radical-induced lipid peroxidation, in jugular vein plasma immediately after declamping of the ICA [18].

Increased BP after CEA is largely attributed to the baroreceptor reflex failure after denervation during the procedure. This is especially expressed after bilateral CEA; the baroreflex breakdown induced hypertension leading to an increase of CBF. In contrast, the autoregulation mechanisms are diminished and thus lead to hyperperfusion in the previously hypoperfused tissue. Both cerebral hyperperfusion associated with cerebral edema and the elevated intracranial pressure may lead to an increase in central and peripheral norepinephrine levels and a subsequent further elevation of the systemic blood pressure.

Increased CBF, which cannot be controlled by the autoregulatory mechanisms, leads to the transudation of fluid into the pericapillary astrocytes and interstitium. This results in vasogenic white matter edema, especially in the vertebrobasilar circulation territory of the posterior parietal and occipital regions. New studies on rodent models are trying to shed more light on the mechanisms of occurrence of CHS [19].

Clinical presentation, risk factors and diagnostics

CHS can develop at any time; immediately after the procedure to up to a month later, but the majority of patients develop symptoms within the first few days (mean 5 days) [7, 20, 21]. Although this most commonly appears after CAS and CEA, CHS was described and after the subclavian artery stenting [20] and after the endovascular reconstruction of carotid artery in a high-flow carotid-jugular fistula [21]. The reported incidence rate of CHS and ICH after CEA is 1.9% and 0.37% and 1.16% and 0.74% after CAS [3, 6, 7, 10–12, 22]. The most common symptoms include: headaches, fluctuation of consciousness, confusion and focal neurologic deficit (Table 1).

Table 1
Clinical symptoms of cerebral hyperperfusion syndrome

Symptoms	Incidence (%)
Deterioration of consciousness, confusion	37.1
Headache	30.6
Epileptic disturbances, focal seizures	25.8
Motor disturbances (hemiparesis, hemiplegia)	17.7
Abnormal speech, aphasia	6.4
Nausea	4.8
Intracranial hemorrhage	4.8
Psychotic disorders	3.2
Visual disturbances (hemianopsia)	3.2
Ataxia	1.6

Headaches are usually moderate to severe, ipsilateral to the revascularised artery, pounding and migrenous [23]. Focal neurologic deficit is a result of cerebral edema and usually is transient [6, 15, 24, 25]. It includes the cortex derived symptoms – hemiplegia, hemiparesis, hemianopsia, dysphasia, seizures and less commonly ataxia and visual disorders. Seizures can sometimes present even as status epilepticus [26].

By far the most devastating complication of CHS is ICH. It is a very rare complication, as previously stated, but it is often fatal (36%–63%), and up to 80% of survived patients are left with significant morbidity [15, 27, 28]. Since ICH is associated with CHS, the symptoms of increased intracranial pressure can be present (nausea, vomiting, or altered sensorium) [29]. There is a form of hyperacute ICH that occurs within hours after CAS, and it is almost always unpreventable since it occurs without prodromal signs. It could be a result of rupture of perforating arteries in basal ganglia which are exposed with suddenly normalized perfusion pressure after CAS [30].

The occurrence of CHS is multifactorial, while the cerebral perfusion and autoregulation are individualized in each patient. CBF changes in each patient are variable, and there is no proof that a degree of stenosis is directly linked with the CBF variations [31]. This could be explained by the presence of collateral circulation and the degree of cerebral autoregulation impairment in each patient. Various studies indicated a potential role for risk factors, definitive prediction of subgroups of patients with an increased risk of developing CHS after CEA, or CAS, is not feasible. This point expresses not the ambiguity of the risk factors, but the complexity and the multifactorial contribution in the pathogenesis of the syndrome. The risk factors that were described to be significantly involved in developing CHS are shown in Table 2.

Table 2
Risk factors for developing cerebral hyperperfusion

Risk factors
Preoperative
Long standing hypertension with cerebral microangiopathy
Diabetes mellitus
Older age
Recent contralateral carotid endarterectomy
High-grade ipsilateral stenosis
Contralateral occlusion/high grade stenosis
Incomplete circle of Willis
Attenuated cerebrovascular reactivity after acetazolamide challenge
Perioperative
Intraoperative distal carotid pressure of < 40 mmHg
High doses of volatile halogenated hydrocarbon anesthetics
Periprocedural cerebral infarction
Intraoperative ischemia
Refractory postoperative cerebral hyperperfusion
Postoperative
Postoperative hypertension
Administration of anticoagulants or antiplatelet agents

Some risk factors are identified as more significant than the others in the increase the risk of CHS: low pulsatility index, severe ipsilateral or contralateral carotid disease, bilateral carotid artery stenosis and incomplete circle of Willis [15, 32, 33].

Additionally, one study suggested that interval between two procedures should be no less than 3 months [3]. That study reported 6.6% of patients developing CHS after bilateral CEA within less than 3 months. The authors suggested that inconsistencies in baroreceptor function may be a causative factor for CHS.

The use of anticoagulants and antiplatelet agents is routine after CEA and CAS. In the absence of sufficient data, it remains uncertain whether the use of post-procedure anticoagulation therapy may be associated with an increased risk of developing CHS and ICH [27, 28].

There were several techniques suggested to identify the patients at risk for developing CHS. The most widely available method is transcranial color Doppler (TCD). It is used to determine CBF changes via monitoring cerebral blood flow velocities (CBFV) changes in intracranial vessels. TCD is used to assess cerebrovascular reactivity using vasodilator agents such as acetazolamide, CO_2 inhalation, or the breath holding test [34–36]. The blood flow is severely restricted when there is a critical ICA stenosis present and after being removed, the blood flow increases dramatically. In the patients with badly impaired cerebral autoregulation, this dramatically increases the mean flow velocity in the middle cerebral artery (MCA) [34–36]. The preoperative drop in CBFV is indicative of hypoperfusion and can lead to postoperative hyperperfusion and thus to the CHS.

However, TCD has several limitations. The first one is an insufficient cranial window and the second one is the experience of operater [24, 37]. Despite these drawbacks, the TCD findings should always be evaluated carefully. Results in the TCD studies demonstrate that the blood flow redistribution through the anterior communicating pathway and the ophthalmic artery is achieved, in case of contralateral ICA stenosis, and in the patients with contralateral ICA occlusion through the posterior communicating pathway. Asher et al. [4] reported a significant increase in the mean internal carotid artery volume flow (MICAVF) in all patients with CHS during the symptomatic period. After the symptoms receded, the flow volume returned to normal.

Standard computed tomography (CT) has limited value preoperatively and can be completely normal postoperatively. It can still be useful as a quick tool to remove a suspicion of ICH. Also, the brain edema that can be seen early can be indicative of CHS (Figure 1). A recent study showed the pretreatment CT perfusion imaging (CTP) with acetazolamide challenge could identify the patients at risk for CHS after CAS. CTP maps were assessed for the absolute and relative cerebral blood flow (CBF), cerebral blood volume (CBV), mean transit time (MTT). Although the CTP parameter that the most accurately identified the patients at risk for HPS was the absolute value of post-acetazolamide MTT, resting MTT was sufficiently accurate [37–47]. Single photon emission CT (SPECT) can detect alterations in the preoperative cerebral perfusion (after administering acetazolamide) [47]. It is also very useful in differentiating between cerebral ischemia and hyperperfusion, and identifying the patients at risk of CEA. However, some studies did not find any correlation between preoperative asymmetry in brain perfusion in rest and CHS [39–42]. Ogasawara et al. [43] suggested that hyperperfusion lasting at least up to three postoperative days on SPECT predisposes to the CHS development.

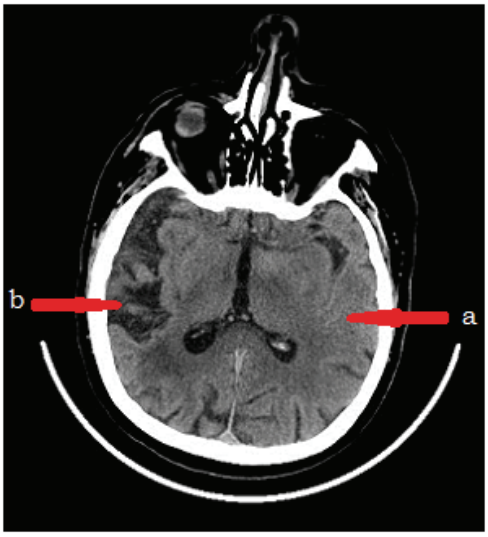

Fig. 1 – Brain computed tomography showing: a) brain swelling as a result of cerebral hyperperfusion syndrome; b) older date ischemic lesion after a stroke.

The magnetic resonance imaging (MRI) techniques also proved useful in diagnosis of CHS, and especially power-weighted (PW) MRI that can reveal intrahemispheric differences in CBF in the patients after CEA [11]. However, as it is not quantitative method, PWI MRI can only be used in absence of contralateral steno-occlusive ICA disease. The conventional MRI findings in the patients with CHS include white matter edema, focal infarction and a local, or a massive hemorrhage. These abnormalities, however, are not pathognomonic for CHS.

Alternative methods such as intraoperative electroencephalography (EEG) and ocular pneumoplethysmography were proposed for diagnosing CHS. However, these methods are yet to prove their worth [48, 49].

Managment of hyperperfusion syndrome

The most important stage in the prevention of CHS is the aggressive BP management postoperatively. This needs to be performed in order to prevent the most dangerous complication of CHS, the ICH. Further reduction of BP even in the normotensive patients should be considered if hyperperfusion is detected, as they can develop hypertension later. Drugs like labetalol and clonidine should be used. Vasodilating drugs with hydralazine, nitrate and Ca^+ channel blockers should be avoided since they can add to already existing brain swelling [23, 42–44]. Beyond this criteria, there is no evidence favoring any other specific drug. Also, beta blockers should be limited [43, 44].

The cerebral edema treatment includes adequate sedation, hyperventilation, manitol administration and hypertonic saline solution [10, 41, 45]. Corticosteroids were tried, but their effectiveness remains uncertain [10, 41, 45].

Oxygen-derived free radicals produced during ischemia were implicated in the ischemia-reperfusion injury. In cerebral tissue, these radicals could lead to the endothelial dysfunction and a break in the blood brain barrier, leading to the post-ischemic hyperperfusion, edema and hemorrhage. In one small case series with historical controls, edaravone, a free-radical scavenger that inhibits lipid peroxidation and vascular endothelial injury decreased the incidence of hyperperfusion following CEA, mainly in the patients with decreased CVR [50].

Conclusion

CHS is a rare, but potentially deadly complication of brain revascularization procedures. Two key, interlinked and synergistic mechanisms play part in its occurrence – impaired cerebral autoregulation and the elevated BP after procedure. It is important to understand the complexity and multiple factors that contribute to the symptom appearance. Although different studies developed risk factors, the determination of subgroups of patients in danger of developing CHS after CAS or CEA is still not feasible. If not treated promptly and properly, CHS can lead to fatal ICH. The treatment strategies were developed towards regulating the BP, reducing brain swelling, and most importantly, limiting the extreme blood flow rising in the patients at potential risk.

R E F E R E N C E S

1. *Radak D, Tanasković S, Matić P, Babić S, Aleksić N, Ilijevski N.* Eversion carotid endarterectomy--our experience after 20 years of carotid surgery and 9897 carotid endarterectomy procedures. Ann Vasc Surg 2012; 26(7): 924–8.
2. *Radak Dj, Ilijevski N, Djukic N.* Carotid surgery today: an update after 14000 carotid endarterectomy procedures. Vojnosanit Pregl 2016; 73(5): 472–9.
3. *Hobson RW 2nd, Mackey WC, Ascher E, Murad MH, Calligaro KD, Comerota AJ,* et al. Society for Vascular Surgery. Management of atherosclerotic carotid artery disease: clinical practice guidelines of the Society for Vascular Surgery. J Vasc Surg 2008; 48(2): 480–6.
4. *Ascher E, Markevich N, Schutzer RW, Kallakuri S, Jacob T, Hingorani AP.* Cerebral hyperperfusion syndrome after carotid endarterectomy: Predictive factors and hemodynamic changes. J Vasc Surg 2003; 37(4): 769–77.
5. *Ogasawara K, Sakai N, Kuroiwa T, Hosoda K, Iihara K, Toyoda K,* et al. Japanese Society for Treatment at Neck in Cerebrovascular Disease Study Group. Intracranial hemorrhage associated with cerebral hyperperfusion syndrome following carotid endarterectomy and carotid artery stenting: retrospective review of 4494 patients. J Neurosurg 2007; 107(6): 1130–6.
6. *Abou-Chebl A, Yadav JS, Reginelli JP, Bajzer C, Bhatt D, Krieger DW.* Intracranial haemorrhage and hyperperfusion syndrome following carotid artery stenting. J Am Coll Cardiol 2004; 43(9): 1596–601.
7. *Solomon RA, Loftus CM, Quest DO, Correll JW.* Incidence and etiology of intracerebral hemorrhage following carotid endarterectomy. J Neurosurg 1986; 64(1): 29–34.
8. *Piepgras DG, Morgan MK, Sundt TM Jr, Yanagihara T, Mussman LM.* Intracerebral hemorrhage after carotid endarterectomy. J Neurosurg 1988; 68(4): 532–6.
9. *Van Mook WN, Rennenberg RJ, Schurink GW, van Oostenbrugge RJ, Mess WH, Hofman PA,* et al. Cerebral hyperperfusion syndrome. Lancet Neurol 2005; 4(12): 877–88.
10. *Sundt TM, Sandok BA, Whisnant JP.* Carotid endarterectomy. Complications and preoperative assessment of risk. Mayo Clin Proc 1975; 50(1): 301–6.
11. *Ogasawara K, Inoue T, Kobayashi M, Endo H, Yoshida K, Fukuda T,* et al. Cerebral hyperperfusion following carotid endarterectomy: diagnostic utility of intraoperative transcranial Doppler ultrasonography compared with single-photon emission computed tomography study. AJNR Am J Neuroradiol 2005; 26(2): 252–7.
12. *Mitrasinović A, Kolar J, Radak S, Nenezić D, Kupresanin I, Aleksić N,* Ultrasonografic monitoring of hemodynamic parameters in symptomatic and asymptomatic patients with high-grade carotid stenosis prior and following carotid endarterectomy. Vojnosanit Pregl 2012; 69(5): 399–404. (Serbian)
13. *Henderson RD, Phan TG, Piepgras DG, Wijdicks EF.* Mechanisms of intracerebral hemorrhage after carotid endarterectomy. J Neurosurg 2001; 95(6): 964–9.
14. *Krdžić I, Čovišković-Šternić N, Katsiki N, Isenović ER, Radak Đ.* Correlation of carotid artery disease severity and vasomotor response of cerebral blood vessels. Angiology 2015; 66(5): 481–7.
15. *Reigel MM, Hollier LH, Sundt TM Jr, Piepgras DG, Sharbrough FW, Cherry KJ.* Cerebral hyperperfusion syndrome: a cause of neurologic dysfunction after carotid endarterectomy. J Vasc Surg 1987; 5(4): 628–34.
16. *Tang SC, Huang YW, Shieh JS, Huang SJ, Yip PK, Jeng JS.* Dynamic cerebral autoregulation in carotid stenosis before and after carotid stenting. J Vasc Surg 2008; 48(1): 88–92
17. *Suga Y, Ogasawara K, Saito H, Komoribayashi N, Kobayashi M, Inoue T,* et al. Preoperative cerebral hemodynamic impairment and reactive oxygen species produced during carotid endarterectomy correlate with development of postoperative cerebral hyperperfusion. Stroke 2007; 38(10): 2712–7.
18. *Soong CV, Young IS, Hood JM, Rowlands BJ, Trimble ER, Barros D'Sa AA.* The generation of byproducts of lipid peroxidation following carotid endarterectomy. Eur J Vasc Endovasc Surg 1996; 12(4): 455–8.
19. *Schwartz RB.* Hyperperfusion encephalopathies: hypertensive encephalopathy and related conditions. Neurologist 2002; 8(1): 22–34.
20. *Ito K, Yonaha H, Kai Y, Hokama Y, Nagamine H, Miyagi T,* et al. Hyperperfusion syndrome after stent placement for subclavian artery stenosis: case report. Neurol Med Chir (Tokyo) 2012; 52(12): 902–5.
21. *Mondel PK, Udare AS, Anand S, Kulkarni AV, Kapadia FN, Modhe JM,* et al. Cerebral hyperperfusion syndrome after endovascular reconstruction of carotid artery in high-flow carotid-jugular fistula. Cardiovasc Intervent Radiol 2014; 37(5): 1369–75.
22. *Ziaja D, Biolik G, Kocełak P, Sznapka M, Janas P, Czajka A,* et al. Neurological symptoms associated with cerebral hyperperfusion syndrome after CEA and CAS - one centre study. Eur Rev Med Pharmacol Sci 2014; 18(8): 1176–80.
23. *Coutts SB, Hill MD, Hu WY.* Hyperperfusion syndrome: toward a stricter definition. Neurosurgery 2003; 53(5): 1053–8; discussion 1058–60.
24. *Sundt TM Jr, Sharbrough FW, Piepgras DG, Kearns TP, Messick JM Jr, O'Fallon WM.* Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy:

with results of surgery and hemodynamics of cerebral ischaemia. Mayo Clin Proc 1981; 56(9): 533–43.
25. *Bernstein M, Ross Fleming JF, Deck JH.* Cerebral hyperperfusion after carotid endarterectomy: a cause of cerebral hemorrhage. Neurosurgery 1984; 15(1): 50–6.
26. *Oh SI, Lee SJ, Lee YJ, Kim HJ.* Delayed cerebral hyperperfusion syndrome three weeks after carotid artery stenting presenting as status epilepticus. J Korean Neurosurg Soc 2014; 56(5): 441–3.
27. *Abou-Chebl A, Yadav JS, Reginelli JP, Bajzer C, Bhatt D, Krieger DW.* Intracranial haemorrhage and hyperperfusion syndrome following carotid artery stenting. J Am Coll Cardiol 2004; 43(9): 1596–601.
28. *Wagner WH, Cossman DV, Farber A, Levin PM, Cohen JL.* Hyperperfusion syndrome after carotid endarterectomy. Ann Vasc Surg 2005; 19: 479–86.
29. *Adhiyaman V, Alexander S.* Cerebral hyperperfusion syndrome following carotid endarterectomy. QJM 2007; 100: 239–44.
30. *Buhk JH, Cepek L, Knauth M.* Hyperacute intracerebral hemorrhage complicating carotid stenting should be distinguished from hyperperfusion syndrome. AJNR Am J Neuroradiol 2006; 27(7): 1508–13.
31. *Ko NU, Achrol AS, Chopra M, Saha M, Gupta D, Smith WS,* et al.Cerebral blood flow changes after endovascular treatment of cerebrovascular stenoses. AJNR Am J Neuroradiol 2005; 26(3): 538–42.
32. *Jovic M, Unic-Stojanovic D, Isenovic E, Manfredi R, Cekic O, Ilijevski N,* et al. Anesthetics and cerebral protection in patients undergoing carotid endarterectomy. J Cardiothorac Vasc Anesth 2015; 29(1): 178–84.
33. *Jansen C, Sprengers AM, Moll FL, Vermeulen FE, Hamerlijnck RP, van Gijn J,* et al. Prediction of intracerebral haemorrhage after carotid endarterectomy by clinical criteria and intraoperative transcranial Doppler monitoring: results of 233 operations. Eur J Vasc Surg 1994; 8(2): 220–5.
34. *Markus HS, Harrison MJ.* Estimation of cerebrovascular reactivity using transcranial Doppler, including the use of breath-holding as the vasodilator stimulus. Stroke 1992; 23: 668–73.
35. *Widder B, Paulat K, Hackspacher J, Mayr E.* Transcranial Doppler CO2-test for the detection of hemodynamically critical carotid artery stenoses and occlusions. Eur Arch Psychiatry Neurol Sci 1986; 236(3): 162–8.
36. *Ringelstein EB, Sievers C, Ecker S, Schneider PA, Otis SM.* Noninvasive assessment of CO2-induced cerebral vasomotor response in normal individuals and in patients with internal carotid artery occlusions. Stroke 1988; 19(8): 963–9.
37. *Ogasawara K, Inoue T, Kobayashi M, Endo H, Yoshida K, Fukuda T, et* al. Cerebral hyperperfusion following carotid endarterectomy: diagnostic utility of intraoperative transcranial Doppler ultrasonography compared with single-photon emission computed tomography study. AJNR Am J Neuroradiol 2005; 26(2): 252–7.
38. *Maltezos CK, Papanas N, Papas TT, Georgiadis GS, Dragoumanis CK, Marakis J,* et al. Changes in blood flow of anterior and middle cerebral arteries following carotid endarterectomy: a transcranial Doppler study. Vasc Endovascular Surg 2007; 41: 389–96.
39. *de Nie AJ, Blankensteijn JD, Visser GH, van der Grond J, Eikelboom BC.* Cerebral blood flow in relation to contralateral carotid disease, an MRA and TCD study. Eur J Vasc Endovasc Surg 2001; 21(3): 220–6.
40. *Hosoda K, Kawaguchi T, Shibata Y, Kamei M, Kidoguchi K, Koyama J,* et al. Cerebral vasoreactivity and internal carotid artery flow help to identify patients at risk for hyperperfusion after carotid endarterectomy. Stroke 2001; 32(7): 1567–73.
41. *Naylor AR, Evans J, Thompson MM, London NJ, Abbott RJ, Cheryman G,* et al. Seizures after carotid endarterectomy: hyperperfusion, dysautoregulation or hypertensive encephalopathy? Eur J Vasc Endovasc Surg 2003; 26(1): 39–44.
42. *Sfyroeras GS, Arsos G, Karkos CD, Liasidis C, Spyridis C, Boundas D,* et al. Interhemispheric asymmetry in brain perfusion before and after carotid stenting: a 99mTc-HMPAO SPECT study. J Endovasc Ther 2006; 13(6): 729–37.
43. *Ogasawara K, Yukawa H, Kobayashi M, Mikami C, Konno H, Terasaki K,* et al. Prediction and monitoring of cerebral hyperperfusion aftercarotid endarterectomy by using single-photon emission computerized tomography scanning. J Neurosurg 2003; 99(3): 504–10.
44. *Dalman JE, Beenakkers IC, Moll FL, Leusink JA, Ackerstaff RG.* Transcranial doppler monitoring during carotid endarterectomy helps to identify patients at risk of post-operative hyperperfusion. Eur J Vasc Endovasc Surg 1999; 18(3): 222–7.
45. *Hingorani A, Ascher E, Tsemekhim B, Markevich N, Kallakuri S, Schutzer R,* et al. Causes of early post carotid endartectomy stroke in a recent series: the increasing importance of hyperperfusion syndrome. Acta Chir Belg 2002; 102: 435–8.
46. *Yoshimoto T, Shirasaka T, Yoshizumi T, Fujimoto S, Kaneko S, Kashiwaba T.* Evaluation of carotid distal pressure for prevention of hyperperfusion after carotid endarterectomy. Surg Neurol 2005; 63(6): 554–7; discussion 557–8.
47. *Yoshie T, Ueda T, Takada T, Nogoshi S, Fukano T, Hasegawa Y.* Prediction of cerebral hyperperfusion syndrome after carotid artery stenting by CT perfusion imaging with acetazolamide challenge. Neuroradiology 2016; 58(3): 253–9.
48. *Jia B, Zhao L, Xiao W, Cai B, Wang TL, Li DG.* A new rodent model of cerebral hyperperfusion. Int J Clin Exp Med 2015; 8(10): 17441–50. eCollection 2015.
49. *Kim KH, Lee CH, Son YJ, Yang HJ, Chung YS, Lee SH.* Post-carotid endarterectomy cerebral hyperperfusion syndrome : is it preventable by strict blood pressure control?: J Korean Neurosurg Soc 2013; 54(3): 159–63.
50. *Ogasawara K, Inoue T, Kobayashi M, Endo H, Fukuda T, Ogawa A.* Pretreatment with the free radical scavenger edaravone prevents cerebral hyperperfusion after carotid endarterectomy. Neurosurgery 2004; 55(5): 1060–7.

Received on September 10, 2017.
Accepted on October 11, 2017.
Online First October, 2017.