

Stroke prevention in asymptomatic carotid artery disease: revascularization of carotid stenosis is not the solution

Robert G. Hart¹, Kuan H. Ng²

¹ Stroke Program, Department of Medicine (Neurology), Population Health Research Institute, McMaster University, Hamilton Health Sciences, Hamilton, Ontario, Canada

² Stroke Program, Department of Medicine (Neurology), McMaster University, Hamilton Health Sciences, Hamilton, Ontario, Canada

KEY WORDS

asymptomatic carotid stenosis, carotid artery atherosclerosis, carotid artery revascularization, carotid endarterectomy

ABSTRACT

Asymptomatic carotid stenosis (ACS) exceeding 50% is present in about 2% of 60-year-old patients and an even higher fraction of older individuals. The major independent risk factors include advancing age, male sex, tobacco smoking, and a history of vascular disease. The best available evidence does not support either population screening for ACS or routine carotid revascularization when ACS is discovered. There is an urgent need to identify patients with ACS and a sufficiently high risk of ipsilateral stroke (despite contemporary medical management) to warrant invasive treatment. The mainstays of medical management are antiplatelet therapy (usually low-dose aspirin), high-dose statins, blood pressure control, and smoking cessation. Patients with ACS should be periodically educated about symptoms of transient ischemic attack and stroke that require emergent medical attention. Current guidelines vary widely in recommendations regarding revascularization (ie, endarterectomy or carotid stenting). The benefits of revascularization strategies remain uncertain for patients with ACS who receive contemporary medical management.

Introduction Cervical carotid artery atherosclerosis as a cause of stroke was initially emphasized 65 years ago,¹ and the search for effective and safe preventive strategies has vexed clinicians ever since. About 15% of ischemic strokes occurring among patients in Europe and North America are associated with cervical carotid artery atherosclerotic stenosis (FIGURE).^{2,3} Consistent results of large randomized trials conducted in the 1980s and 1990s demonstrated that carotid endarterectomy roughly halves the risk of ipsilateral stroke in patients with asymptomatic carotid stenosis (ACS),^{4,5} and population screening for ACS followed by revascularization would at first thought seem a reasonable approach to reducing the burden of stroke associated with ACS. However, it has proven not to be so straightforward.

A vast number of publications continue to be published yearly concerning management of ACS, testifying to ongoing uncertainties and controversies. In this commentary, we review recent studies and current concepts and guidelines concerning the management of ACS. We conclude

that revascularization of ACS is not the solution to substantially reducing the burden of stroke from cervical carotid artery atherosclerosis and that decisions regarding revascularization of ACS based solely on the degree of stenosis are poorly informed.

Frequency of asymptomatic carotid stenosis A recent pooled analysis of 4 population-based studies from Europe and North America involving 23 706 people screened with ultrasonography yielded a frequency of ACS exceeding 50% in 2% and of ACS exceeding 70% in 0.5% of the combined cohort at a mean age of 61 years old.⁶ ACS exceeding 70% was present in 0.3% of this cohort in the absence of a history of vascular disease and in 1.9% of those with clinical coronary artery disease. Age, male sex, current tobacco smoking, and a history of vascular disease were the strongest independent predictors of severe ACS.⁶ Considering 29 published studies (not restricted to population-based cohorts), the prevalence of ACS of 50% or higher was 5% among men vs. 2% for women

Correspondence to:

Robert G. Hart, MD, Population Health Research Institute, DBCVSR1 C4-105, 237 Barton Street East, Hamilton, Ontario L8L 2X2, Canada, tel.: +1-905-521-2100, fax: +1-905-577-1427, e-mail: robert.hart@phri.ca

Received: April 5, 2015.

Revision accepted: April 13, 2015.

Published online: April 17, 2015.

Conflict of interest: none declared.

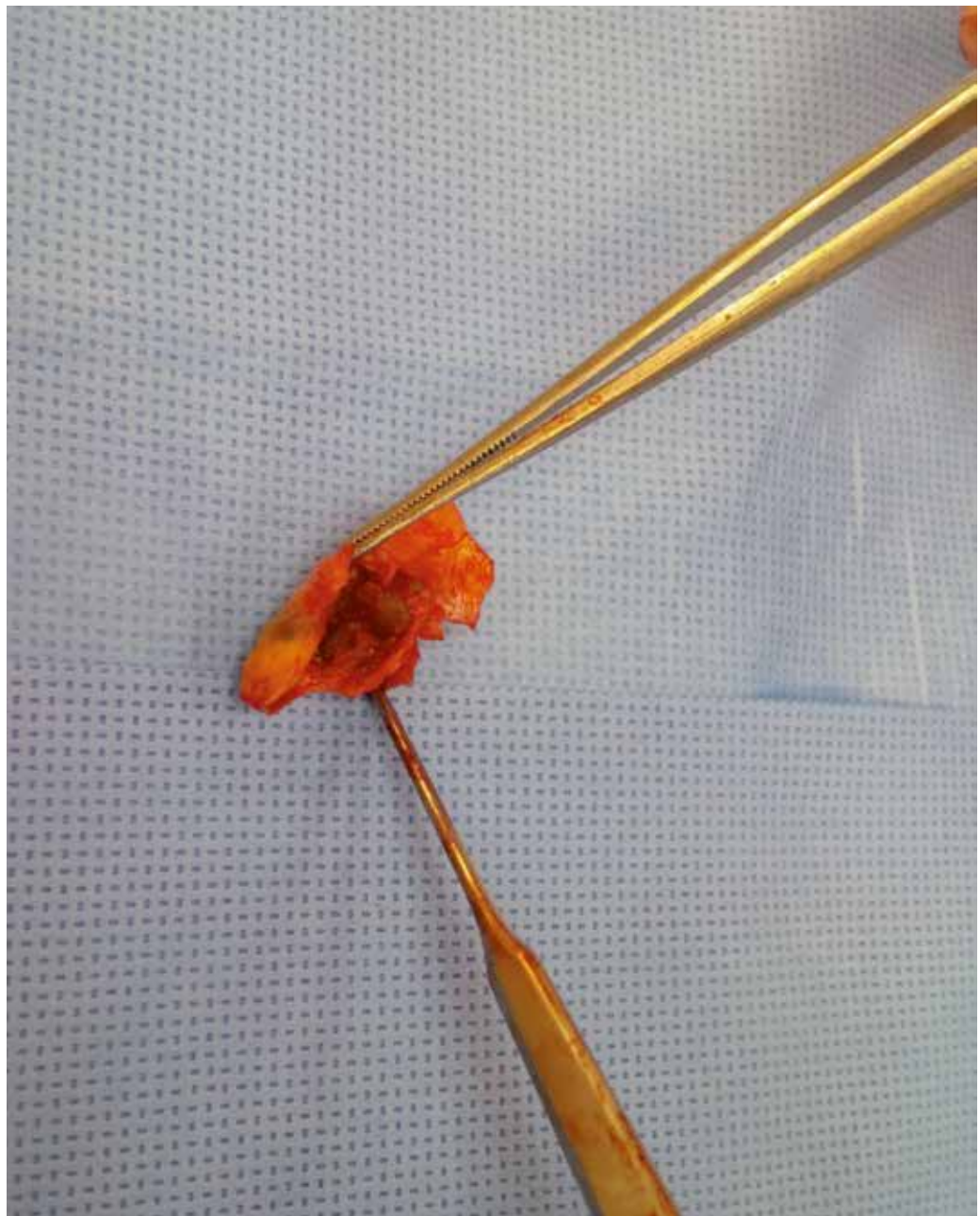
Pol Arch Med Wewn. 2015;

125 (5): 363-369

Copyright by Medycyna Praktyczna,

Kraków 2015

FIGURE Example of a cervical carotid artery plaque that caused a high-grade carotid artery stenosis removed by carotid endarterectomy (courtesy of Dr. Jacques Tittley, Hamilton, Canada)



under the age of 70 years.⁷ Among a cohort of patients with manifest arterial disease or diabetes mellitus with a mean age of 58 years, the frequency of ACS of 50% or higher was 8%.⁸ However, in many of these studies, screening with carotid artery ultrasonography was not confirmed by other imaging modalities, and false-positive results are likely to have inflated prevalence estimates for ACS (see “Population screening for asymptomatic carotid stenosis” below).

Risk of stroke and other vascular events associated with asymptomatic carotid stenosis Based on consistent evidence, the absolute rate of ipsilateral stroke is low in contemporary patients with ACS, averaging 1% per year or lower among unselected patients^{3,4,9,10} and those with vascular disease.⁸ Current stroke rates associated with ACS are consistently lower than those reported in the 1980s and 1990s (which averaged 2%–4% per year) and are lower than stroke rates reported in

the medically-treated arms of randomized trials testing carotid endarterectomy.¹¹ The most likely explanation for the decline in stroke rates is an interval improvement in medical management, particularly the use of “statin” medications that have been proved to remarkably reduce stroke risk in patients with carotid artery atherosclerosis.¹² Consequently, the modest absolute benefit conferred by carotid endarterectomy documented by randomized trials conducted 20 years ago is likely to be even smaller for contemporary patients with ACS managed aggressively with blood pressure control, lipid lowering using statins, and antiplatelet therapy. In short, because of low ipsilateral stroke rates in contemporary patients with ACS, many have questioned the relevance of these earlier trials to current practice.^{2-4,13} On the other hand, the complication rates associated with revascularization of ACS may also be declining.¹⁴

ACS is a risk factor for covert/silent brain infarcts detected by computed tomography in

TABLE 1 Predictors of stroke in asymptomatic cervical carotid stenosis (adapted from Paraskevas et al.)²²

Predictor	Comments
degree of maximal stenosis ²³⁻²⁵	statistically significant, but relatively weak predictor of ipsilateral stroke
progression of stenosis by serial ultrasound imaging ^{24,26}	incidence of progression 10%–25% (varying by method of assessment and criteria) over 2–4 years and associated with double, but still relatively low (2% per year), ipsilateral stroke risk
history of stroke or TIA ^{18,23}	hazard ratio for stroke, ~3
microembolic signals detected by transcranial Doppler ultrasound ²⁷	abnormal signals detected distal to carotid stenosis independently associated with subsequent ipsilateral stroke
carotid plaque characteristics defined by ultrasonography ²⁸⁻³²	plaque echolucency or ulceration; new techniques include 3-dimensional contrast-enhanced ultrasound
carotid plaque characteristics (including intraplaque hemorrhage) detected by high-resolution MRI with special sequences ³³⁻³⁷	intraplaque hemorrhage, plaque rupture, and luminal thrombus associated with higher risks of stroke in nonstenotic carotid plaques
silent ipsilateral brain infarcts on CT or MRI ³⁸	prevalence 10%–20% by CT ^{8,17} associated with about double the stroke risk ³⁸
¹⁸ FDG-PET-CT features of plaque ³⁹	
cerebral blood flow reserve / intracranial collateral circulation ⁴⁰	assessed by transcranial Doppler with acetazolamide challenge/carbon dioxide challenge or positron emission tomography; “isolated hemisphere” due to incomplete Circle of Willis

Abbreviations: CT, computed tomography; ¹⁸FDG, fluorodeoxyglucose; MRI, magnetic resonance imaging; PET, positron emission tomography; TIA, transient ischemic attack

patients without a history of stroke^{8,15,16}; however, covert/silent infarcts are equally distributed ipsilaterally and contralaterally to the side of the ACS.¹⁷ Patients with ACS also have a substantial frequency of coronary artery atherosclerosis: myocardial infarction is about half as frequent as stroke, while the risk of cardiovascular death exceeds that of stroke.^{8,18} Aggressive medical management of ACS offers the additional benefit of prevention of coronary events. There are too few high-quality studies to determine whether ACS is an independent risk factor for cognitive dysfunction,¹⁹ and the effect of revascularization on measures of cognition have been mixed.²⁰

Can patients with asymptomatic carotid stenosis at high risk for stroke be identified? The degree of stenosis alone is a relatively weak predictor of ipsilateral stroke and does not identify medically treated patients with carotid artery atherosclerosis who have sufficient absolute rates of ipsilateral stroke to justify revascularization.²¹ It has been estimated that with contemporary medical management only about 10% of unselected patients with ACS could benefit from additional revascularization.²² Can high-risk subgroups be identified?

This critical issue has recently received considerable attention (TABLE 1). ACS patients with other clinical manifestations of atherosclerosis (ie, coronary artery disease, peripheral vascular disease) or previous stroke have higher stroke risks, but the absolute rates have not been well characterized. High-resolution magnetic resonance imaging has been promising for identification of “vulnerable plaques” associated with higher risk of stroke (TABLE 1).^{36,37,41} However, many experts believe that there are as yet no externally validated, reliable indicators that can identify ACS patients at an increased risk of stroke.⁴²⁻⁴⁴

Revascularization for asymptomatic carotid stenosis: current guidelines

Despite the concern that carotid revascularization is of no substantial benefit for the majority of ACS patients who are treated with contemporary medical management, current guidelines endorse revascularization for many patients with ACS (TABLE 2). Although guidelines attempt to synthesize the best available management recommendations, they are not without limitations.^{52,53} Guideline recommendations favoring revascularization of ACS have been criticized on the basis that revascularization does not make sense for ACS patients with an annual risk of ipsilateral stroke of 1% to 2% because of the inherent upfront risk of opening the carotid arteries.^{11,22} The most recent European Stroke Organisation guideline is perhaps the most cautious regarding revascularization: “There is uncertainty about the benefit of revascularization for asymptomatic carotid stenosis ... there is not enough data to recommend [stenting] as an alternative to [carotid endarterectomy].”⁴⁶

Population screening for asymptomatic carotid stenosis

There is a general agreement in the recent literature that population screening for ACS is not warranted.^{2,42,51,54-56} Using optimistic estimates for the sensitivity (95%) and specificity (92%) of carotid ultrasonography, screening of 100 000 adults assuming a true prevalence of ACS of 1% would yield 940 true-positive results and 7920 false-positive results, or a positive predictive value of about 10%.² Of note, the positive predictive value of ultrasonography is much higher in patients presenting to clinicians with asymptomatic bruits or other clinical manifestations of atherosclerosis (eg, peripheral vascular disease, contralateral ischemic stroke). The U.S. Preventive Services Task Force has recently reaffirmed its recommendation against screening for ACS in the general adult population based on

TABLE 2 Selected guideline recommendations for management of asymptomatic carotid artery stenosis

Guideline	Summary of recommendations
European Stroke Organisation (ESO) (2008) ⁴⁵	low-dose aspirin for >50% stenosis; CEA not recommended for 60%–99% stenosis “except in those at high risk of stroke” ^a ; angioplasty/stenting not recommended
ESO Karolinska Stroke Update: Consensus Statements 2012 ⁴⁶	“There is uncertainty about the benefit of revascularization for asymptomatic carotid stenosis ... not enough data to recommend [stenting] as an alternative to CEA.”
European Society of Cardiology (2011) ⁴⁷	antiplatelet therapy and statin; “CEA should be considered” if life expectancy >5 years and perioperative stroke/death rate <3%; stenting may be considered at centers with documented stroke/death rate <3%
National Institute for Clinical Excellence (2011) ⁴⁸	medical control of cardiovascular risk factors; if severe, “sometimes treated by CEA”; caution against routine use of stenting (“well-documented risks ... evidence on efficacy is inadequate”)
American Heart Association / American Stroke Association (2014) ⁴⁹	aspirin and statin; CEA “reasonable” for >70% stenosis if perioperative risk is <3%; angioplasty/stenting “might be considered in highly selected patients ... but effectiveness compared with medical therapy alone not well established”; it is reasonable to repeat duplex ultrasonography annually to assess progression in CAS >50%
Canadian Best Practice Recommendations (2014) ⁵⁰	antiplatelet therapy and statin; CEA for selected patients with 60%–99% stenosis if life expectancy >5 years and <3% perioperative risk; stenting may be considered for nonoperative candidates
Society for Vascular Surgery (2011) ⁵¹	antiplatelet therapy; consider CEA for ≥60% diameter stenosis if ≥3 year life expectancy and perioperative stroke/death rate <3%

a high risk of stroke not defined

Abbreviations: CAS, coronary artery stenosis; CEA, carotid endarterectomy

TABLE 3 Management of patients with asymptomatic carotid artery stenosis

antiplatelet therapy (usually low-dose aspirin)
high-dose statin therapy
control of hypertension (target blood pressure <140/90 mmHg)
smoking cessation (if applicable)
lifestyle modification for cardiovascular health (physical activity, diet)
patient education: – recognition of symptoms of transient ischemic attack warranting urgent reevaluation – importance of seeking emergent care for symptoms of stroke
revascularization (carotid endarterectomy or carotid artery stenting): – recommended as an option for >70% stenosis or ill-defined “high-risk” or “selected” patients by many guidelines – about 10% of patients with acute coronary syndrome estimated to benefit from revascularization – no generally accepted criteria to define those at high-risk – occasionally justified by strong patient preferences

the uncertain absolute benefits of revascularization with either endarterectomy or stenting if added to current medical management.⁴²

Ongoing randomized trials involving patients with asymptomatic carotid stenosis The Carotid Revascularization and Medical Management for Asymptomatic Carotid Stenosis Trial (CREST-2)(NCT02 089 217) is comparing medical management with carotid revascularization (endarterectomy and stenting separately) added to medical management in 2480 patients with asymptomatic carotid artery stenosis of 70% or higher. The Stent-Protected Angioplasty in Asymptomatic Carotid Artery Stenosis vs. Endarterectomy (SPACE2)(ISRCTN 78 592 017) is enrolling patients with asymptomatic (within the previous 6 months) carotid artery stenosis of 50% or higher, comparing two revascularization options

with medical management alone. The Aggressive Medical Treatment Evaluation for Asymptomatic Carotid Artery Stenosis (AMTEC) trial (NCT00 805 311) compared carotid endarterectomy with medical therapy in 400 patients and was recently terminated, but results have not been published.⁵⁷ The Cardiovascular Outcomes for People Using Anticoagulation Strategies (COMPASS)(NCT01 776 424) is anticipated to include about 1200 patients with ACS randomized to receive aspirin, rivaroxaban, or their combination and followed for about 4 years, but it will likely be underpowered to meaningfully assess relative effects of the antithrombotic agents on prevention of ipsilateral stroke.

At least 3 additional randomized trials are comparing carotid endarterectomy with stenting in ACS patients: (Asymptomatic Carotid Surgery Trial-2 (ACST-2)(NCT00 883 402)⁵⁸; Randomized Evaluation of Short Term and Long Term Outcome After Endovascular Repair by Stenting of Carotid Artery Stenosis in Patients with Severe (70% and Higher) Asymptomatic Carotid Stenosis (NCT00 772 278); Carotid Angioplasty and Stenting Versus Endarterectomy in Asymptomatic Subjects Who Are at Standard Risk for Carotid Endarterectomy With Significant Extracranial Carotid Stenotic Disease (ACT 1)(NCT00 106 938) (terminated, but as yet unpublished).

It appears unlikely that addition of revascularization to medical management will show worthwhile absolute benefits for ACS patients chosen for inclusion based only on the degree of stenosis. In our view, restricting inclusion to ACS patients who have additional stroke risk factors will be required for randomized trials to have “positive”, clinically meaningful results. For example, atrial fibrillation is a more powerful risk factor for stroke than ACS, and recent large randomized

trials testing antithrombotic therapies were restricted to atrial fibrillation patients with additional stroke risk factors. The inclusion of patients with ACS in future randomized trials assessing revascularization should not be based solely on the degree of stenosis. Including otherwise unselected patients with ACS will bias results toward negative trials owing to the inclusion of low-risk patients with little potential to benefit from any intervention. While subgroup analyses of high-risk participants included in these trials are likely to be undertaken, even if planned a priori, interpretation in the setting of negative overall trial results is problematic. We endorse the recommendation of Naylor et al.¹¹ that “It is, therefore, essential that any new . . . randomized trials in asymptomatic patients include analysis of one or more . . . imaging strategies” or other features (TABLE 1) to identify the subgroups at high risk for stroke who will benefit substantially from revascularization.⁴⁴ Nevertheless, definitively negative comparisons of invasive versus noninvasive treatment of ACS could usefully impact management in parts of the world such as the United States where revascularization of ACS patients is widely undertaken.^{11,22}

Management of asymptomatic carotid stenosis in 2015 Carotid artery stenosis of any degree is a relatively weak predictor of ipsilateral stroke, and it is time to move beyond ACS to focus research on defining high-risk carotid plaques (with stenosis as only one component). It is clear that nonstenotic carotid artery plaques can be a source of brain ischemia.³³ Medical management of ACS should include smoking cessation, antiplatelet therapy, high-dose “statin” therapy, and optimizing blood pressure (TABLE 3). Educating and periodically reeducating of ACS patients about symptoms of transient ischemic attack and stroke is important. We occasionally obtain ultrasonography in patients with asymptomatic cervical bruits to demonstrate ACS to further motivate patients to stick with prevention strategies (especially the difficult challenge of cessation of tobacco smoking).

Enthusiasm for revascularization is driven in part by the worst nightmare of clinicians that their patient with known ACS who is being treated medically experiences disabling stroke and that they “should have done more.” This faulty reasoning is rarely in the best interest of their patient, exposing the overwhelming majority (95%) of ACS patients to unnecessary procedures that are not without risk.^{59,60} Revascularization is not the right thing to do for most ACS patients, and restraint is warranted^{3,4,13,22,42,44,61} (although not everyone agrees).⁶² Until high-risk subgroups of ACS patients receiving contemporary medical therapy who could benefit substantially from revascularization are reliably identified (or the much larger low-risk subgroups who cannot), and revascularization is shown to be beneficial in contemporary randomized trials in

high-risk ACS patients, the management controversies surrounding ACS will continue. Alas, there is no end in sight.

REFERENCES

- 1 Fisher M. Occlusion of the internal carotid artery. *AMA Arch Neurol Psych.* 1951; 65: 346-377.
- 2 Jonas DE, Feltner C, Amick HR, et al. Screening for asymptomatic carotid artery stenosis: A systematic review and meta-analysis for the U.S. Preventive Services Task Force. *Ann Intern Med.* 2014; 161: 336-346.
- 3 Abbott AL. Medical (nonsurgical) intervention alone is now best for prevention of stroke associated with asymptomatic severe carotid stenosis. Results of a systematic review and analysis. *Stroke.* 2009; 40: e573-e583.
- 4 Raman G, Moorthy D, Hadar N, et al. Management strategies for asymptomatic carotid stenosis. *Ann Intern Med.* 2013; 158: 676-685.
- 5 Halliday A, Harrison M, Hayter E, et al. Asymptomatic Carotid Surgery Trial (ACST) Collaborative Group. 10-year stroke prevention after successful carotid endarterectomy for asymptomatic stenosis (ACST-1): a multicenter randomized trial. *Lancet.* 2010; 376: 1074-1084.
- 6 de Weerd M, Grieving JP, Hedblad B, et al. Prediction of asymptomatic carotid artery stenosis in the general population. Identification of high-risk groups. *Stroke.* 2014; 45: 2366-2371.
- 7 de Weerd M, Grieving JP, de Jong AW, et al. Prevalence of asymptomatic carotid artery stenosis according to age and sex. *Stroke.* 2009; 40: 1105-1113.
- 8 Goessen BM, Visseren FL, Kappelle LJ, et al; The SMART Study Group. Asymptomatic carotid artery stenosis and the risk of new vascular events in patients with manifest arterial disease. The SMART study. *Stroke.* 2007; 38: 1470-1475.
- 9 Marquardt L, Geraghty OC, Methta Z, Rothwell PM. Low risk of ipsilateral stroke in patients with asymptomatic carotid stenosis on best medical treatment: a prospective, population-based study. *Stroke.* 2010; 41: e11-e17.
- 10 Spence JD, Coates C, Li H, et al. Effects of intensive medical therapy on microemboli and cardiovascular risk in asymptomatic carotid stenosis. *Arch Neurol.* 2010; 67: 180-186.
- 11 Naylor AR. What is the current status of invasive treatment of extracranial carotid artery disease? *Stroke.* 2011; 42: 2080-2085.
- 12 Sillesen H, Amarencu P, Hennerici MG, et al. Atorvastatin reduces the risk of cardiovascular events in patients with carotid atherosclerosis: a secondary analysis of the Stroke Prevention by Aggressive Reduction in Cholesterol Levels (SPARCL) trial. *Stroke.* 2008; 39: 3297-3302.
- 13 Naylor AR. Time to rethink management strategies in asymptomatic carotid artery disease. *Nat Rev Cardiol.* 2012; 9: 116-124.
- 14 ACST-2 Collaborative Group; Halliday A, Bulbulia R, Gray W, et al. Status update and interim results from the Asymptomatic Carotid Surgery Trial – 2 (ACST-2). *Euro Vasc Endovasc Surg.* 2013; 46: 510-518.
- 15 Das RR, Seshardi S, Beiser AS, et al. Prevalence and correlates of silent cerebral infarcts in the Framingham Offspring Study. *Stroke.* 2008; 39: 2929-2935.
- 16 Uehara T, Tabuchi M, Mori E. Risk factors for silent cerebral infarcts in subcortical white matter and basal ganglia. *Stroke.* 1999; 30: 378-382.
- 17 Brott T, Tomsick T, Feinberg W, et al; the Asymptomatic Carotid Atherosclerosis Study Investigators. Baseline silent cerebral infarction in the Asymptomatic Carotid Atherosclerosis Study. *Stroke.* 1994; 25: 1122-1129.
- 18 Aichner R, Topkian R, Alberts MJ, et al. High cardiovascular event rates in patients with asymptomatic carotid stenosis: the REACH Registry. *Eur J Neurol.* 2009; 16: 902-908.
- 19 Chang X-L, Zhou H-Q, Lei C-Y, et al. Association between asymptomatic carotid stenosis and cognitive function: A systematic review. *Neurosci Biobehav Reviews.* 2013; 37: 1493-1499.
- 20 Siddiqui AH, Hopkins LH. Asymptomatic carotid stenosis: The not-so-silent disease. *J Am Coll Cardiol.* 2013; 61: 2510-2513.
- 21 Paraskevas KI, Spence JD, Weith FJ, Nicolaides AN. Identifying which patients with asymptomatic carotid stenosis could benefit from intervention. *Stroke.* 2014; 45: 3720-3724.
- 22 Spence JD, Pelz D, Veith FJ. Asymptomatic carotid stenosis. Identifying patients at high enough risk to warrant endarterectomy or stenting. *Stroke.* 2014; 45: 655-657.
- 23 Nicolaides AN, Kakkos SK, Kyriacou E, et al; the Asymptomatic Carotid Stenosis and Risk of Stroke (ACSRS) Study Group. Asymptomatic internal carotid artery stenosis and cerebrovascular risk stratification. *J Vasc Surg.* 2010; 52: 1486-1496.
- 24 Kakkos SK, Nicolaides AN, Charalambous I, et al; the Asymptomatic Carotid Stenosis and Risk of Stroke (ACSRS) Study Group. Predictors and clinical significance of progression or regression of asymptomatic carotid stenosis. *J Vasc Surg.* 2014; 59: 956-967.
- 25 den Hartog AG, Achterberg S, Moll FL, et al; the SMART Study Group. Asymptomatic carotid artery stenosis and the risk of ischemic stroke

- according to subtype in patients with clinical manifest arterial disease. *Stroke*. 2013; 44: 1002-1007.
- 26 Hirt LS. Progression rate and ipsilateral neurological events in asymptomatic carotid stenosis. *Stroke*. 2014; 45: 702-706.
 - 27 Markus HS, King A, Shipley M, et al. Asymptomatic embolization for prediction of stroke in the Asymptomatic Carotid Emboli Study (ACES): a prospective observational study. *Lancet Neurol*. 2010; 9: 663-671.
 - 28 Madani A, Beletsky V, Tamayo A, et al. High-risk asymptomatic carotid stenosis: Ulceration on 3D ultrasound vs. TCD microemboli. *Neurol*. 2011; 77: 744-750.
 - 29 Partovi S, Loebe M, Aschwanden M, et al. Contrast-enhanced ultrasound for assessing carotid atherosclerotic plaque lesions. *AJR Am J Roentgenol*. 2012; 198: W13-W19.
 - 30 Kakkos SK, Griffen MB, Nicolaides AN, et al; the Asymptomatic Carotid Stenosis and Risk of Stroke (ACSRS) Study Group. The size of juxtaluminal hypoechoic area in ultrasound images of asymptomatic carotid plaques predicts the occurrence of stroke. *J Vasc Surg*. 2013; 57: 609-618.
 - 31 Gupta A, Kesavabhotla K, Baradham H, et al. Plaque echolunacy and stroke risk in asymptomatic carotid stenosis. A systematic review and meta-analysis. *Stroke*. 2015; 46: 91-97.
 - 32 Pedro LM, Sanchez JM, Seabra J, et al. Asymptomatic carotid disease – a new tool for assessing neurological risk. *Echocardiography*. 2014; 31: 353-361.
 - 33 Freilinger TM, Schindler A, Schmidt C, et al. Prevalence of nonstenosing, complicated atherosclerotic plaques in cryptogenic stroke. *J Am Coll Cardiol Imag*. 2012; 5: 397-405.
 - 34 Cai JM, Hatsukami TS, Ferguson MS, et al. Classification of human carotid atherosclerotic lesions with in vivo multicontrast magnetic resonance imaging. *Circ*. 2002; 106: 1368-1373.
 - 35 Sun J, Balu N, Hippe DS, et al. Subclinical carotid atherosclerosis: Short-term natural history of lipid-rich necrotic core – a multicenter study with MR imaging. *Radiol*. 2013; 268: 61-68.
 - 36 Underhill HR, Hatsukami TS, Cai J, et al. A noninvasive imaging approach to assess plaque severity: the carotid atherosclerosis score. *AJNR Am J Neuroradiol*. 2010; 3: 1068-1075.
 - 37 Xu D, Hippe DS, Underhill HR, et al. Prediction of high-risk plaque development and plaque progression with the Carotid Atherosclerosis Score. *J Am Coll Cardiol Imag*. 2014; 7: 366-373.
 - 38 Kakkos SK, Sabetai M, Tegos T, et al; the Asymptomatic Carotid Stenosis and Risk of Stroke (ACSRS) Study Group. Silent embolic infarcts on computed tomography brain scans and risk of ipsilateral hemispheric events in patients with asymptomatic internal carotid artery stenosis. *J Vasc Surg*. 2009; 49: 902-909.
 - 39 Muller HFG, Viacoz A, Fisch L, et al. 18FDG-PET-CT. An imaging biomarker of high-risk carotid plaques, correlation to symptoms and microembolic signals. *Stroke*. 2014; 45: 3561-3566.
 - 40 Gupta A, Chazen JL, Hartman M, et al. Cerebrovascular reserve and stroke risk in patients with carotid stenosis or occlusion. A systematic review and meta-analysis. *Stroke*. 2012; 43: 2884-2891.
 - 41 Howard DP, van Lammeren GW, Rothwell PM, et al. Symptomatic carotid atherosclerotic disease. Correlations between plaque composition and ipsilateral stroke risk. *Stroke*. 2015; 46: 182-189.
 - 42 LeFevre ML, on behalf of the U.S. Preventive Services Task Force. Screening for asymptomatic carotid artery stenosis: U.S. Preventive Services Task Force Recommendation Statement. *Ann Intern Med*. 2014; 161: 356-362.
 - 43 den Hartog AG, Bovens SM, Koning W, et al. Current status of clinical magnetic resonance imaging for plaque characteristics in patients with carotid artery stenosis. *Euro J Vasc Endovasc Surg*. 2013; 45: 7-21.
 - 44 Naylor AR, Schroeder TV, Sillesen H. Clinical and imaging features associated with an increased risk of late stroke in patients with asymptomatic carotid disease. *Eur J Vasc Endovasc Surg*. 2014; 48: 633-640.
 - 45 European Stroke Organisation (ESO) Executive Committee and the ESO Writing Committee. Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. *Cerebrovasc Dis*. 2008; 25: 457-507.
 - 46 Karolinska Stroke Update Consensus Statements 2012. 1. Carotid endarterectomy versus angioplasty and stenting. www.strokeupdate.org/Cons_Carotid_session_2012.aspx. Accessed April 15, 2015.
 - 47 Tendera M, Aboyans V, Bartelink M-L, et al. ESC Guidelines on the diagnosis and treatment of peripheral artery diseases. *Eur Heart J*. 2011; 32: 2851-2906.
 - 48 NICE interventional procedure guidance 388. Carotid artery stent placement for asymptomatic extracranial carotid stenosis. Guidance 388 (Issued April 2011). www.nice.org.uk/ipg388. Accessed April 15, 2015.
 - 49 Meschia JF, Bushnell C, Boden-Albala B, et al. Guidelines for the primary prevention of stroke. A statement for healthcare professionals from the American Heart Association / American Stroke Association. *Stroke*. 2014; 45: 3754-3832.
 - 50 Coutts SB, Wein TH, Lindsay MP, et al. on behalf of the Heart and Stroke Foundation Canada Canadian Stroke Best Practices Advisory Committee. Canadian Stroke Best Practice Recommendations: Secondary prevention of stroke guidelines, update 2014. *Int J Stroke*. DOI 10.1111/ijis.12439.
 - 51 Ricotta JJ, AbuRahma A, Ascher E, et al. Updated Society of Vascular Surgery guidelines of management of extracranial carotid disease. Executive summary. *J Vasc Surg*. 2011; 54: 832-836.
 - 52 Shaneyfelt T. In guidelines we cannot trust. *Arch Intern Med*. 2012; 172: 1633-1634.
 - 53 Hart RG, Bailey RD. An assessment of guidelines for prevention of ischemic stroke. *Neurology*. 2002; 59: 977-982.
 - 54 Heldenberg E, Bass A. Is there a role for asymptomatic carotid artery stenosis screening? *Harefuah*. 2014; 153: 475-477.
 - 55 Goldstein LB, Bushnell CD, Adams RJ, et al. American Heart Association Stroke Council Guidelines for the primary prevention of stroke: a guideline for healthcare professionals from the American Heart Association / American Stroke Association. *Stroke*. 2011; 42: 517-584.
 - 56 Weyer GW, Davis AM. Screening for asymptomatic carotid artery stenosis. *JAMA*. 2015; 313: 192-193.
 - 57 Kolos I, Loukianov M, Dupik N, et al. Optimal medical treatment versus carotid endarterectomy: the rationale and design of the Aggressive Medical Treatment Evaluation for Asymptomatic Carotid Artery Stenosis (AMTEC) study. *Int J Stroke*. 2015; 10: 269-274.
 - 58 Rudarakanchana N, Dialynas M, Halliday A. Asymptomatic Carotid Surgery Trial – 2 (ACST-2): Rationale for a randomized clinical trial comparing carotid endarterectomy with carotid artery stenting in patients with asymptomatic carotid artery stenosis. *Eur J Vasc Endovasc Surg*. 2009; 38: 239-242.
 - 59 Woo K, Garg J, Hye RJ, Dille RB. Contemporary results of carotid endarterectomy for asymptomatic carotid stenosis. *Stroke*. 2010; 41: 975-978.
 - 60 Naylor AR. Identifying the high-risk carotid plaque. *Journal of Cardiovascular Surgery*. 2014; 55:11-20.
 - 61 Ng KH, Oczkowski W. Treatment options for asymptomatic carotid artery stenosis were compared. *ACP Journal Club*. 2013; 159: 6.
 - 62 Hirt L, Halliday A. Controversies in neurology: asymptomatic carotid stenosis – intervention or just stick to medical therapy. The argument for carotid endarterectomy. *J Neural Trans*. 2011; 118: 631-636.

Zapobieganie udarom mózgu u chorych z bezobjawową miażdżycą tętnic szyjnych: rewaskularyzacja nie jest rozwiązaniem

Robert G. Hart¹, Kuan H. Ng²

1 Stroke Program, Department of Medicine (Neurology), Population Health Research Institute, McMaster University, Hamilton Health Sciences, Hamilton, Ontario, Kanada

2 Stroke Program, Department of Medicine (Neurology), McMaster University, Hamilton Health Sciences, Hamilton, Ontario, Kanada

SŁOWA KLUCZOWE

bezobjawowe
zwężenie tętnic
szyjnych,
endarterektomia
tętnic szyjnych,
miażdżycza tętnic
szyjnych,
rewaskularyzacja
tętnic szyjnych

STRESZCZENIE

Bezobjawowe zwężenie tętnic szyjnych (*asymptomatic carotid stenosis* – ACS) >50% występuje u ok. 2% osób w wieku 60 lat i u jeszcze większego odsetka starszych chorych. Głównymi niezależnymi czynnikami ryzyka rozwoju choroby są zaawansowany wiek, płeć męska, palenie papierosów oraz choroby układu naczyniowego w wywiadzie. Najlepsze dostępne dowody nie przemawiają za wykonywaniem populacyjnych badań przesiewowych w kierunku ACS ani za rutynową rewaskularyzacją w przypadku rozpoznania ACS. Istnieje pilna potrzeba wyselekcjonowania chorych z ACS oraz wysokim ryzykiem ipsilateralnego udaru mózgu (pomimo nowoczesnego leczenia zachowawczego), uzasadniającym wdrożenie leczenia zabiegowego. Podstawą leczenia zachowawczego ACS są leki przeciwplatekcyjne (najczęściej kwas acetylosalicylowy w niewielkiej dawce), statyny w wysokich dawkach, kontrola ciśnienia tętniczego oraz zaprzestanie palenia papierosów. Chorzy z ACS powinni być regularnie informowani o wymagających kontaktu z lekarzem objawach przemijającego ataku niedokrwiennego oraz udaru mózgu. Aktualne wytyczne różnią się od siebie w zakresie rekomendacji dotyczących wykonywania rewaskularyzacji (tj. endarterektomii lub angioplastyki wewnątrznaczyniowej z wszczepieniem stentu). U chorych z ACS dotychczas leczonych zachowawczo korzyści z rewaskularyzacji pozostają niepewne.

Adres do korespondencji:

Robert G. Hart, MD, Population
Health Research Institute DBCVSR
C4-105, 237 Barton Street East,
Hamilton, Ontario L8L 2X2, Kanada,
tel.: +1-905-521-2100, fax:
+1-905-577-1427, e-mail:
robert.hart@phri.ca

Praca wpłynęła: 05.04.2015.

Przyjęta do druku: 13.04.2015.

Publikacja online: 17.04.2015.

Nie zgłoszono sprzeczności
interesów.

Pol Arch Med Wewn. 2015;

125 (5): 363-369

Copyright by Medycyna Praktyczna,

Kraków 2015