

Western University

Scholarship@Western

Department of Medicine Publications

Medicine Department

1-1-2020

Asymptomatic Carotid Stenosis: High Risk With Suboptimal Treatment: Re: Five Year Outcomes in Men Screened for Carotid Artery Stenosis at 65 Years of Age: A Population Based Cohort Study

J. David Spence

Robarts Research Institute, jdspence@uwo.ca

Follow this and additional works at: <https://ir.lib.uwo.ca/medpub>

Citation of this paper:

Spence, J. David, "Asymptomatic Carotid Stenosis: High Risk With Suboptimal Treatment: Re: Five Year Outcomes in Men Screened for Carotid Artery Stenosis at 65 Years of Age: A Population Based Cohort Study" (2020). *Department of Medicine Publications*. 180.

<https://ir.lib.uwo.ca/medpub/180>

CORRESPONDENCE

Asymptomatic Carotid Stenosis: High Risk With Suboptimal Treatment

Re: Five Year Outcomes in Men Screened for Carotid Artery Stenosis at 65 Years of Age: A Population Based Cohort Study

A recent paper in this journal¹ reported that among 3 057 Swedish men screened for carotid stenosis at age 65 in 2007–2009, the risk of events over five years among patients with 80–99% stenosis was surprisingly high: 42% developed symptoms. The authors say that this occurred “despite optimum medical treatment.” But the treatment was hardly optimal. At the five year follow up, only 22% received antiplatelet agents and 29% statins.

Patients with asymptomatic carotid stenosis have such a high cardiovascular risk (if only of coronary events) that they should *all* receive intensive medical therapy, including lifestyle modification. In recent years, overlapping the time in which this study was conducted, the risk of stroke in asymptomatic stenosis has declined elsewhere to ~0.5%.² It is surprising, given the reputation of Swedish healthcare, that these patients were not treated more intensively.

In 2010 we reported³ that among 438 patients with asymptomatic carotid stenosis, the two year risk of stroke declined from 8.8% to 1%, and the two year risk of myocardial

Table 1. Elements of more intensive therapy based on “treating arteries”

Measure	Intervention
<i>Lifestyle modification</i>	
All	Show patients images of their plaque, ⁴ compare the patient's plaque burden with that of healthy persons of the same age and sex, describe the risks associated with that degree of plaque burden and progression, and the possibility and benefits of plaque regression
Smoking cessation	Counselling, liberal nicotine replacement, varenicline or bupropion (depending on history of depression)
Mediterranean diet	Counselling, provision of a booklet summarising advice and providing recipes and links to internet sites; repeated at follow up visits as necessary
Obesity	Counselling on caloric restriction, referral to dietician, bariatric surgery in refractory patients with severe obesity and diabetes or insulin resistance
Exercise	Recommendations for moderate exercise at least 30 min a day, with advice tailored to the patient's disabilities if any
Blood pressure	Advice on how to reduce salt intake, limit alcohol intake, avoid liquorice, decongestants
<i>Medical therapy</i>	
Blood pressure control	Physiologically individualised therapy for resistant hypertension based on renin/aldosterone profile; switch NDAIDs to sulindac

Table 1-continued

Measure	Intervention
Lipid lowering	Statins increasing according to plaque progression to the highest dose tolerated (with use of CoQ10 to minimise myopathic symptoms); then addition of ezetimibe, and as needed for low HDL-C/high triglycerides, addition of fibrates; PCSK9 inhibitors if feasible.
Antiplatelet agents	Low dose aspirin, with addition of clopidogrel in patients with severe stenosis or other indicators of high risk plaque
Anticoagulation	In patients with atrial fibrillation or other probable cardiac source of emboli ^{5,6}
Insulin resistance	Pioglitazone; reinforcement of lifestyle issues
Diabetes	Reinforcement of lifestyle changes; referral to diabetes clinic

The objective of therapy is not to simply achieve target levels of blood pressure, low density lipoprotein cholesterol and other risk factors, but to halt progression or achieve regression of plaque burden. CoQ10 = coenzyme Q10; HDL-C = high density lipoprotein cholesterol; NSAID = non-steroidal anti-inflammatory drugs.

Reproduced by permission of the American Medical Association from Yang C, Bogiatzi C, Spence JD. Risk of Stroke at the Time of Carotid Occlusion. JAMA Neurol 2015; 72(11):1261–7.

infarction declined from 7.6% to 1%, after implementing intensive medical therapy based on “treating arteries”. The elements of “treating arteries” are shown in Table 1. Treating arteries without measuring plaque would be like treating hypertension without measuring blood pressure.

REFERENCES

- Hogberg D, Björck M, Mani K, Svensjö S, Wanhainen A. Five year Outcomes in men screened for carotid artery stenosis at 65 Years of age: a population based Cohort study. *Eur J Vasc Endovasc Surg* 2019;57:759–66.
- Naylor AR. Time to rethink management strategies in asymptomatic carotid artery disease. *Nat Rev Cardiol* 2012;9:116–24.
- Spence JD, Coates V, Li H, Tamayo A, Munoz C, Hackam DG, et al. Effects of Intensive medical therapy on microemboli and cardiovascular risk in asymptomatic carotid stenosis. *Arch Neurol* 2010;67:180–6.

J.David Spence
Stroke Prevention & Atherosclerosis Research Centre,
Robarts Research Institute, Western University, 1400
Western Road, London, N6G 2V4 ON Canada
Email-address: dspence@robarts.ca

Available online 7 September 2019

© 2019 European Society for Vascular Surgery. Published by Elsevier B.V. All rights reserved.

<https://doi.org/10.1016/j.ejvs.2019.07.035>
DOI of original article: <https://doi.org/10.1016/j.ejvs.2019.07.036>