



Preventing carotid stroke

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Two special series focusing on issues regarding the prevention and treatment of stroke in people with carotid stenosis were published in this journal in 2020 and 2021 (1,2). Among the articles: Del Brutto asks “Why are we still debating criteria for carotid artery stenosis?” (1); Lavenson advocates a “quick carotid (ultrasound) scan” to screen for carotid stenosis that might result in the prevention of more than 150,000 strokes each year saving more than \$15,000,000,000 in treatment and rehabilitation costs with an expenditure of nearly half that amount (2); Mechtouff explores the hemodynamics of the cerebroarterial circulation (2); Viticchi relates hypoperfusion to cognition (2). All of the articles describe in detail the empirical observations from large numbers of patients studied in attempts to discern which people have “significant” carotid stenosis (1,2).

The continuing debate uses hemodynamic and other laboratory measures to predict properties of anatomic stenosis and then uses the properties of anatomic stenosis to predict stroke during subsequent life. Here, instead, I assert that extreme hemodynamic forces lead to chronic impaired cognition and to occasional catastrophic predictable stroke events: both can be prevented.

Nearly 10% of adults have a congenital disconnected circle of Willis (coW) leaving one internal carotid artery (ICA) as the sole supply of the ipsilateral middle cerebral artery (MCA) (3): 2% supply both anterior cerebral arteries (ACA); 4% supply the ipsilateral posterior cerebral artery (PCA). Although in 3.7% of cases, the isolated ICA supplies only the MCA with 3.3 cc/s in systole, in nearly 1% of cases, the isolated ICA in addition supplies both ACAs plus the ipsilateral PCA requiring a systolic ICA flow of 8.3 cc/s (*Figure 1*). After age 50, nearly 10% of people develop a stenosis of the proximal ICA due to the growth of a medial

monoclonal atheroma (4). When that atheroma thickens beyond 1.5 mm (5) vasa vasorum from the adventitia proliferate to invade the atheroma providing essential nutrition for the expanding atheroma. As the atheroma grows to embrace the lumen, compensatory adventitial dilation is arrested; further atheroma expansion impinges on the lumen causing stenosis. So long as the flow does not diminish, progressive reduction of stenotic diameter requires increased intrastenotic velocity (6). The Spencer/Reid curve shows that when the stenosis becomes severe, in the absence of collateral flow via the coW, the high ICA resistance to flow results in regional cerebral hypotension (rCh). Spencer/Reid assume that the ICA supplies only the ipsilateral MCA and ACA. The hazardous stenoses are those that are the sole supply to the MCA and adjacent cerebral arteries (7). In that case, an intrastenotic velocity of 500 cm/s creates a 100 mmHg intrastenotic Venturi pressure depression promoting neovascular inflation and hemorrhage in the atheroma (8). With the onset of turbulence in the distal expansion of the stenosis marked by carotid bruit, the pressure difference across the stenosis can approach 100 mmHg (modified Bernoulli equation) leaving dependent regions of the brain with critical 40 mmHg hypotension in a patient with 140 mmHg systemic systolic hypertension. This pressure difference across the stenosis can cause rupture of the atheroma releasing atheroembolic debris toward the hypotensive dependent brain regions. In addition to bruit, these cases have ophthalmic artery (OA) hypotension with ocular pulse delay; if untreated, 13.5% of these patients have stroke in 2 years (9). In addition to the MCA, the skin above the eyebrow (supraorbital angiosome = SOA) is supplied by the ipsilateral ICA so when distal ICA pressure is low, the temperature of the SOA is more

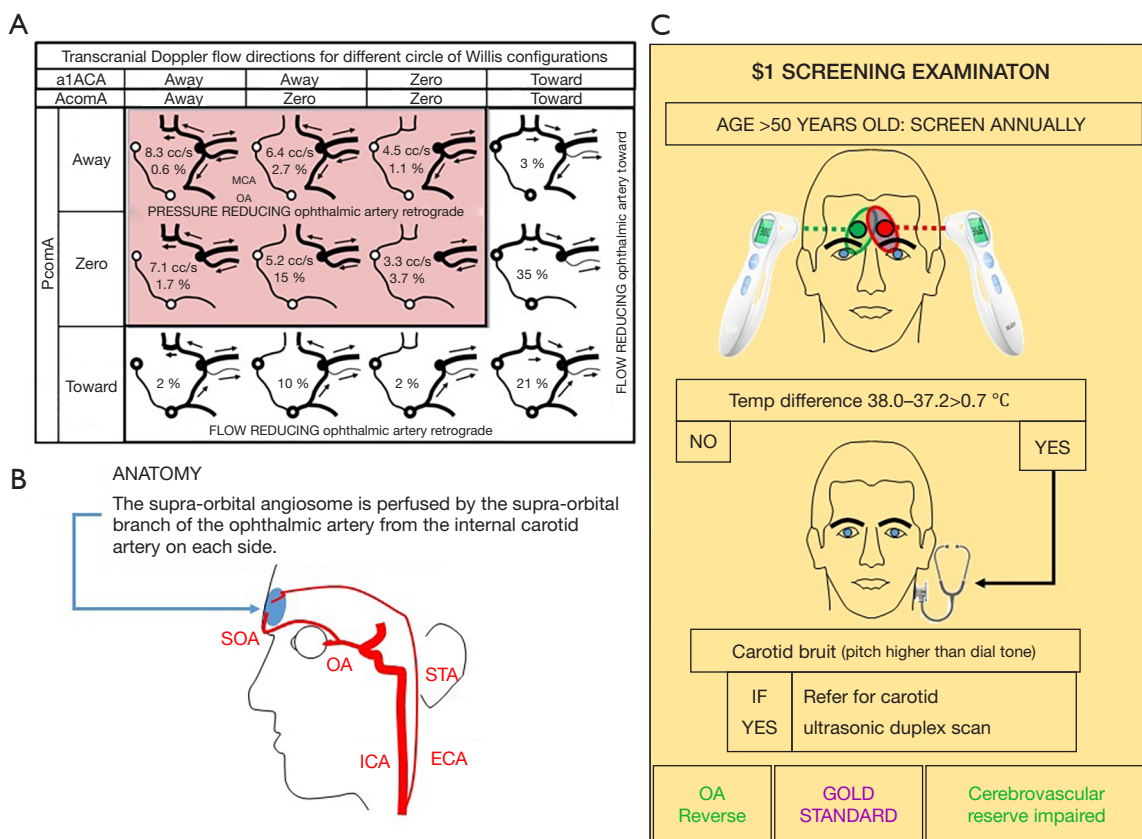


Figure 1 Essentials of a \$1.00 screening examination for pressure reducing carotid stenosis showing (A) congenital circle of Willis configurations (coW) with prevalence (%) and flow (cc/s), (B) distribution of ICA flow to the SOA and (C) screening procedure. ACA, anterior cerebral arteries; SOA, supraorbital angiosome; OA, ophthalmic artery; STA, superficial temporal artery; ICA, internal carotid artery; ECA, external carotid artery.

than 0.7 °C below the contralateral normal skin, with a 99% specificity for carotid stenosis (10). Patients with severe ICA stenosis combined with an isolated ICA have a 43.7% chance of stroke in 2 years on medical treatment, and a 12.3% chance of stroke in 2 years with anatomic revascularization (11) making the number needed to treat (NNT =3) to prevent one stroke.

A screening program (Figure 1) in primary care might identify people who can benefit from anatomic carotid revascularization by endarterectomy or stent (CEA/CAS). All people over age 50 screened at annual physical exam with a non-contact infrared thermometer with a left/right SOA temperature difference greater than 0.7 °C are evaluated with neck auscultation for ipsilateral carotid bruit to identify the cases most likely to benefit from CEA/CAS. Expect 1/100 positive tests (1,000,000/100,000,000 in the USA). Those cases are referred for advanced

Doppler ultrasound studies including: bilateral systolic arm BP measurement, bilateral ultrasound spectral Doppler velocimetry with waveforms of the carotid arteries, directional OA waveforms, and, if “temporal windows” are present, MCAs with measurement of acceleration time plus 30 s breath-hold cerebrovascular reserve assessment. At least 1/10 cases will test positive for ICA peak systolic velocity >300 cm/s, OA reversal, prolonged MCA acceleration time (tardus parvus) and impaired cerebrovascular reserve. These 100,000 cases are most likely to benefit from CEA/CAS. At estimated costs of \$1 for supraorbital thermography, \$300 for carotid ultrasound examination, \$10,000 for carotid endarterectomy, and \$100,000 for stroke treatment/rehabilitation, the cost of preventing 30,000 strokes would be \$1.5 billion, saving of 1.5 billion compared to the \$3 billion to treat their recovery and manage their disability.

Such a system can be implemented by individual

geriatric practices using equipment on hand, tabulating the number of referrals with positive and negative vascular exams. With a reported specificity of 99% (10), the false positive rate should be low. Before CEA/CAS, a trial of aggressive antiatherosclerotic medical therapy monitoring for decreasing bruit frequency and duration plus increasing SOA temperature followed by vascular exam measurements is recommended. Before and after treatment, cognition measures should improve creating immediate diagnostic confidence within each geriatric practice bypassing the need for prolonged/expensive formal multicenter clinical trials. Although the sensitivity of the screening program might be near 50% (10), this limitation does not detract from the value of the program.

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Footnote

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Ethical Statement: The author is accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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