# Maximal wall shear stress in carotid stenoses and functionality of the circle of Wil

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Background˚: Mechanisms of embolic material release
Elevated wall shear stresses encountered in advanced occlusive lesions of internal ca play an important role in the mechanism of atherosclerotic plaques rupture and t combination with complex biological phenomena. In spite of an abundant literature a arterial stenoses and the importance of the underlying problem of public health – embolization is the major cause of strokes which are themselves one of the most important cause of death and disabili few authors dealt with the evaluation of MWSS in severe carotid stenoses. Moreover, the unique structure of cerebral macrocirculation, including an arterial network (loop), called the circle o afferent vessels - the internal carotid (ICaA) and the basilar (BA) arteries - and the efferent o cerebral arteries -, was never taken into account in the assessment of this MWSS, th the flow rate through a carotid stenosis is highly dependent not only on its radius re of the collateral circulatory pathways of the circle of Willis [1]. In the present wo this factor on the MWSS in carotid stenoses.

Methods:
We computed the flow rate through a carotid stenosis as a function of the degrees of ipsilateral stenosis and of the one on the opposite or contralateral side as well as of the diameters of anterior (ACoA) and posterior communicating (PCoA) arteries . We used a non-linear one-dimensional unsteady mathematica flow through the circle of Willis [1, 2], considered as a network of twenty elastic on Fig. 1.. The effects of the carotid stenoses are put into the network model by means of the two semi-empirica formulae of Young and Tsai [3], relating the pressure drop to the flow rate.

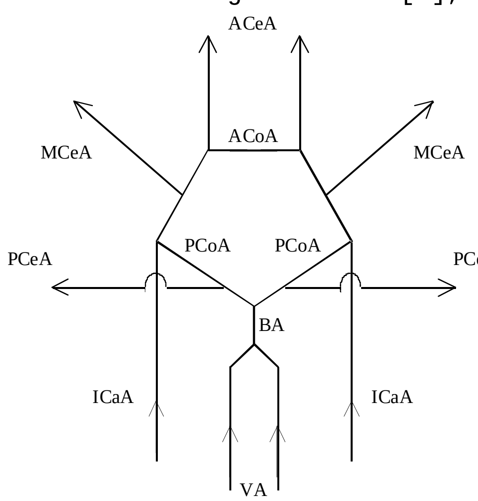


**Fig. 1.** : Diagram of the circle of Willis and its afferent and efferent arteries.

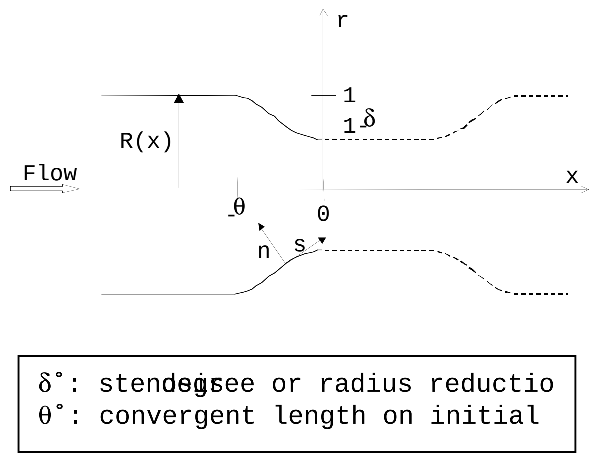


**Fig. 2.** : Geometry and nondimensional parameters of stenosis.

We calculated MWSS as a function of flow rate and geometry of stenosis, i.e., initial and radius reduction (Fig.2), by using the boundary layer theory and steady axisym flow after independence of MWSS on the entry velocity profile was demonstrated, the simple on the above parameters, which could be measured in clinical practice, was established

$$\mathrm{MWSS}\left(\delta,\mathrm{Re}_{eq}\right)=\frac{0{,}231}{(1-\delta)^{3.311}}\sqrt{\mathrm{Re}_{eq}}+\frac{0{,}718}{(1-\delta)^{2{,}982}},\quad \mathrm{Re}_{eq}=\frac{\mathrm{Re}_0}{\theta}$$

where $Re_0$ is the Reynolds number based on the initial radius, θ and δ are defined on Figure 2.

Note that such a relationship correlates accurately and extends the results found in
resolution of Navier-Stokes equation for particular geometrical characteristics.
Introducing in this formula the average flow rate computed by means of the network
stenosed arteries can thus be analysed in the pathophysiological context of these les
their severity and the anatomy of the arterial network.

Results Huge variability of MWSS
Variations of MWSS as a function of stenotic pattern (varying the stenotic degree by steps of
different arrangements of anterior and posterior communicating arteries diameters (fr
ones to fully-functional large ones) emphasize the huge variability of this parameter (

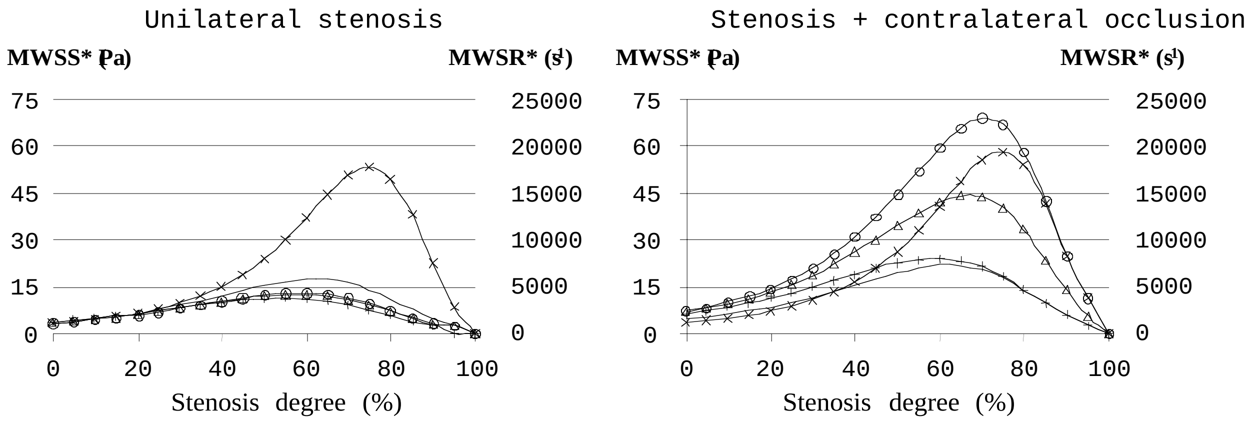


**Fig. 3.** : Dimensional maximal wall shear stress (MWSS*) and rate (MWSR*) as a function of stenosis degree, for five arrangements of anterior and posterior communicating arteries diameters ; × : AcoA=0.4 mm / PCoA=0.4 mm ; o : ACoA=0.4 mm / PCoA=1.6 mm ; o : ACoA=1.6 mm / PCoA=0.4 mm ; Δ : ACoA=1.6 mm / PCoA=1 mm ; + : ACoA=1.6 mm / PCoA=1.6 mm.

Whatever controlateral stenosis degree, MWSS is always maximal inside the ipsilateral for stenosis between 6
80%- the value and position of this maximum depending on collateral circulatory path
zero for occlusions.
For a given degree of unilateral stenosis (Fig 3, left), MWSS is maximal when collat
are not efficient ( both thin anterior and posterior communicating arteries).
For a given degree of stenosis associated to an occlusion (Fig. 3, right), MWSS is m
communicating artery is broad and the posterior communicating arteries are narrow. Th
this artery results in a favourable supply to the controlateral occluded side and an unfavourable increa
and MWSS through the ipsilateral stenosis, leading to very high MWSS values (> 55 Pa) in stenoses
(60%). This last result was obtained because we have not only considered an isolated
included it in the whole network. It could explain the uncertainty about the percenta
carotid obstructive lesions must be considered severe for the risk of stroke.

Conclusion:
Even so further investigations of the mechanical properties of thrombi and plaques
understanding of the role of MWSS in the embolic mechanisms, our results suggest th
release could be maximum between 60 and 80% stenosis, where MWSS is maximal.

References:

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