

ORAL PRESENTATIONS

Physics linkages between arterial stiffness, cerebrovascular flow and cognitive impairment

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Background

Reduced cerebrovascular flow and localized hypoperfusion have been widely associated with various forms of cognitive impairment. Vascular cognitive impairment has also been associated with arterial stiffening and arteriosclerotic development. The linkages between cerebrovascular arteriosclerosis and localized cerebral ischemia, as dictated by the physics of fluid flow, while relatively unaddressed, are vascular contributions to cognitive impairment.

Methods

The physics of fluid dynamics, particularly that of wave propagation in compliant vessels, was applied to the analysis of blood flow in a complex cerebrovascular tree, consisting of hundreds of thousands of arterial junctions. Each of these arterial junctions or bifurcations may partially reflect antegrade pulsatile flow into retrograde flow, resulting in reduced peripheral cerebral pulsatile flow.

Results

The physics of fluid dynamics predicts¹

that, at each bifurcation in the complex cerebrovascular maze, the percentage of antegrade flow which is reflected in retrograde flow is determined by the quotient of the arterial stiffnesses ratio and cross-sectional areas ratio on each side of each bifurcation. Physics predicts that, if the arterial stiffness/area ratios quotient is equal to unity then, ideally, there is no pulsatile retrograde flow. A ten percent pulsatile reflection would result from an arterial mismatch (in the quotation of trunk to branch artery stiffnesses to areas ratios) of about twenty percent. In other words, if a trunk artery at a bifurcation stiffens with aging by twenty percent relative to the bifurcations branch arteries, and the dimensions are unchanged, then the pulsatile antegrade flow reduces by about ten percent. In progressing from the extracranial arteries into the cerebral capillaries this pulsatile flow reduction process may be repeated at each bifurcation tier, causing an accumulating reduction in pulsatile flow into the capillaries. If the amplitude of the pulsatile retrograde flow is sufficiently large, the collision of the retrograde and antegrade pulsatile flows may result in transient flow reversal at select locations in the vascular bed. Such flow reversals may occur where a maximum in the retrograde flow pattern coincides with a minimum in the antegrade flow pattern, resulting in localized diastolic flow reversal. Persistent blood cell flow reversal, with its "scrubbing effect" on endothelial cells, combined with the flow's associated pulse pressure amplification, may cause lifting of endothelial cells, thereby exposing the underlying intima and media to deposition of foreign materials, such as lipids and calcium. Hence, localized retrograde flow may induce arteriosclerotic development, which in turn, may increase bifurcation arterial stiffness ratios, which may further increase

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pulsatile flow reversals, increase arteriosclerotic development, decrease peripheral flow and increase arteriole ischemia at select cerebral locations.

Conclusions

The physics of fluid dynamics predicts quantifiable cross-linkages between macro and microcirculation, as shown in Figure 1, which include age-related increases of localized arterial stiffness, cerebral ischemia and ultimately, parenchymal hypoxia.^{2,3} The specific cerebral locations of such hypoxia would be dependent on localized vascular and physiological conditions. The manifestation and nature of any cognitive impairment associated with localized hypoxia, would, therefore, also be dependent on localized vascular and physiological conditions.

References

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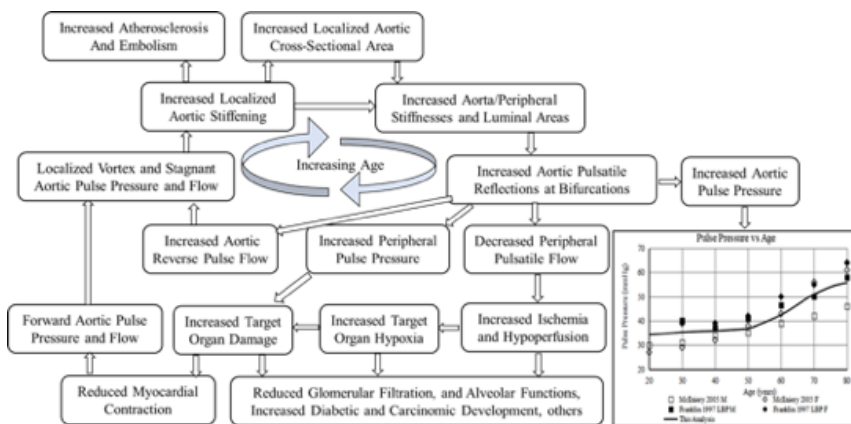


Figure 1. Representation of the cross-linkages between micro and macrocirculation, including influences of arterial bifurcations on both pulsatile pressure and blood flow.