

Intracranial Hemodynamics in Alzheimer's Disease

Meeting: Annual Meeting, American Geriatric Society Annual Meeting, May 2004

Meeting Date: May 2004, Las Vegas, Nevada

Knebl, Janice¹; Orr, David¹; McConathy, Walter¹; Lam, Peter¹; Crutchfield, Kevin².

1. Internal Medicine, Univ. of North Texas Health Sciences Center, Ft Worth, TX

2. Cerebrovascular Medicine, New Health Sciences, Inc., Rockville, MD

Objective: To determine the hemodynamic characteristics of Alzheimer's Disease through the utilization of Dynamic Vascular Assessment (DVA) methodology, Background: Dementia of the Alzheimer's Type is associated with the deposition of beta-amyloid protein in the vascular endothelium and parenchyma of the brain. Global low cerebral blood flow and hypometabolism have been described, however, hemodynamics associated with these changes have not been elucidated. Utilization of transcranial Doppler (TCD) ultrasound along with DVA methodology to evaluate intracranial hemodynamics has not been studied in Alzheimer's Disease.

Methods: Patients (N=56) with a diagnosis of probable dementia of the Alzheimer's type, and age-matched controls (n=31), voluntarily underwent full TCD evaluation with Dynamic Vascular Assessment (DVA) to characterize the hemodynamics of the intracranial vasculature.

Results: The Dynamic Flow index [DFI, mean flow velocity (mfv)/pulsatility index], a surrogate marker for velocity-limiting small vessel disease was significantly diminished in TCD locations representing Intracranial cerebral blood flow. These values also correlated with the Folstein minimal status scores. Pulsatility Index was significantly increased in these same TCD locations. TCD sites representing extracranial blood flow, such as the ophthalmic artery, did not differ between Alzheimer's subjects and controls. In addition, the systolic upstroke acceleration did not differ between the two groups, suggesting that the global low flow is not secondary to diminished proximal flow. In fact, the Dynamic Work Index [DWI, systolic acceleration/mfv] was increased in the Alzheimer's subjects suggesting increased kinetic expenditure to maintain flow and/or vessel stiffening.

Conclusion: Alzheimer's Type Dementia appears to have characteristic cerebral blood flow hemodynamics. The increased impedance dramatically alters forward force and ultimately cerebral blood flow. Based on this data it is possible that low cerebral blood flow in Alzheimer's disease is secondary to increased impedance in the small capacitance vessels, the arterioles. This may be due to increased amyloid deposition with alteration of vascular architecture, increased intracranial pressure (explaining the unaltered extracranial vessel hemodynamics), or a combination of both.