# Discussion of a novel adjunctive therapy for patients with type 2 diabetes and mild cognitive impairment or Alzheimer's disease: A test for the hypothesis that vascular factors are modifiable risks for cognitive impairment

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All major dementias share an underlying vascular component. In Alzheimer's disease (AD), nearly 80% of autopsy-confirmed cases show evidence of cerebrovascular disease, doubling the likelihood that neurodegenerative pathology will manifest as dementia or mild cognitive impairment (MCI) earlier in life. Often, vascular pathologies precede neurodegeneration associated with AD. Type 2 diabetes (T2D) involves a complex cascade of cardiovascular malfunction that impairs glucose delivery to neurons and doubles the risk of developing AD. Cerebral hypoperfusion and endothelial dysfunction in T2D may underlie this risk relationship and contribute to neuronal apoptosis. Following the WHO proclamation linking global brain and cardiovascular health efforts, 3 important considerations are necessary to translate clinical evidence into effective interventions for patients with AD: 1) recognizing that vascular components of brain health provide new target opportunities for effective AD prevention; 2) realizing that ongoing efforts to repurpose and reindicate regulatory-approved vascular interventions for AD treatment can offer an innovative, efficient approach to provide safe and quickly effective interventions for dementia; and 3) appreciating that there are under-represented, vulnerable populations with increased burden and more rapid progression of dementia, due to increased risk for cardiovascular comorbidities, higher incidence of T2D, and less access to quality healthcare. Addressing these 3 factors should increase the likelihood of available effective dementia interventions. Specifically, external counterpulsation (ECP) technology offers one novel approach to introduce both an FDA-approved product and a CMS-eligible medical therapy that may be reindicated for the AD and comorbid T2D population. Presently, this therapy is indicated for refractory angina to improve coronary and peripheral hemodynamics. ECP sequentially compresses and decompresses the vasculature of extremities in synchrony with the cardiac cycle to increase blood flow, instigate angiogenesis, and improve endothelial function, all of which have been shown to influence neuronal function and brain health. In light of recent approval of aducanumab, the need for therapies to ameliorate a vital clinical feature of the disease (ie, cognitive decline), and thereby reduce duration of disability, is especially clear.

## Speaker Biography

Patrick M Moriarty, MD, is professor of medicine at University of Kansas Medical Center in Kansas City. He is director of Clinical Pharmacology and the Atherosclerosis & Lipid-Apheresis Center. He earned his Doctorate in Medicine and Surgery at University of Rome School of Medicine and Surgery, Italy, and completed his residency in internal medicine and training in clinical pharmacology at University of Kansas Medical Center. He is a fellow of the American College of Physicians, American College of Cardiology, European Society of Cardiology, and National Lipid Association; he was 2014-2015 president of the International Society for Apheresis. He is a manuscript reviewer for multiple journals, and associate editor for *Journal of Clinical Apheresis* and *Journal of Clinical Lipidology*. Dr. Moriarty has published over 100 peer-reviewed articles/chapters. His research activities include atherosclerosis, familial hypercholesterolemia, Lp(a), lipoprotein-apheresis, vascular inflammation, dementia, and hemorheology. He has participated in numerous trials related to dyslipidemia, Lp(a), obesity, hypertension, dementia, and diabetes. He is a member of the scientific advisory board for the FH Foundation. His clinical activities involve the treatment and diagnosis of atherosclerosis and cardiovascular disease. University of Kansas Lipoprotein-Apheresis Center is the largest in North America for treatment of familial hypercholesterolemia and elevated Lp(a)..

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