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Review

Arterial stiffness, the brain and cognition: A systematic review

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ABSTRACT

Background: Arterial stiffness is a known predictor of cardiovascular disease, and has also been associated with markers of cerebral small vessel disease as well as poor cognitive function and cognitive decline. The consistency of these associations and their relationship to each other are unclear.
Method: We conducted a systematic review of the evidence associating arterial stiffness with cognitive function and cognitive decline, and with makers of cerebral small vessel disease, specifically lacunar infarcts and white matter hyperintensities.
Results: Thirteen cross-sectional studies examining arterial stiffness and white matter hyperintensities or lacunar infarctions reported a positive association between increased arterial stiffness and radiological findings of cerebral small vessel disease. Two longitudinal studies examining the relationship between arterial stiffness and white matter hyperintensities found increased pulse wave velocity to be an independent predictor of white matter hyperintensity volume. Fifteen cross-sectional and seven longitudinal studies examining arterial stiffness and cognition were identified. Fourteen of the fifteen cross-sectional studies associated increased arterial stiffness with lower cognitive function, and six of the seven longitudinal studies found arterial stiffness to be predictive of cognitive decline.
Conclusion: Arterial stiffness is associated with cerebral small vessel disease and decreased cognitive function. However methodological limitations such as differing covariates between studies and an over-reliance on the MMSE to measure cognition are a concern across much of the literature.
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1. Introduction

Cognitive impairment is becoming increasingly common with the aging of societies, and causes an immense social, economic and emotional burden (Gorelick et al., 2011a). A greater understanding of the mechanisms leading to cognitive impairment with aging may hold the potential to prevent cognitive decline and to maintain cognitive ability into old age. Cardiovascular factors have long been recognized as playing a prime role in the vascular pathogenesis of cognitive decline, and recent studies have found several cardiovascular risk factors such as hypertension, diabetes mellitus and increased left ventricular mass to be linked with cognitive decline (Gorelick et al., 2011b; Hanon et al., 2005; Henskens et al., 2007). Arterial stiffening, a hallmark of vascular aging caused by structural and cellular change within vessel walls, is one such pathophysiological process that has also been associated with cognitive decline (Benetos et al., 2012; Gorelick et al., 2011b; Waldstein and Elias, 2001; Waldstein et al., 2007; Watson et al., 2011), but it remains a less well-studied risk factor of cognitive impairment in late life.

The understanding of the pathophysiology of arterial stiffness has improved in recent years. During aging, increased stiffening in the central elastic arteries due to haemodynamically induced fragmentation of elastin increases the speed of arterial wave propagation, leading reflected waves to arrive at the aorta prematurely during systole rather than diastole (O'Rourke et al., 2010). This results in increased systolic pressure, high resting flow and higher flow pulsations down the vascular tree (O'Rourke and Hashimoto, 2007; Zieman et al., 2005). Arterial stiffness is predictive of total and cardiovascular mortality and end-stage renal disease (Lacolley et al., 2008).

Additionally, arterial stiffness has been found to be associated with structural change in the brain, primarily white matter hyperintensities, cerebral lacunar infarction and cortical brain atrophy (Bateman et al., 2008; Henry Feugeas et al., 2005; Henry-Feugeas et al., 2009; Nichols et al., 2011). Microvascular brain lesions, and in particular white matter hyperintensities have independently been associated with cognitive decline (Bozzali et al., 2011; Eckerstrom et al., 2011; Gili et al., 2011; Grambaite et al., 2011) and as such may be part of the mechanism through which arterial stiffness is propagated into cognitive decline. In the literature it has been hypothesized that increased flow pulsations through the carotid and vertebral arteries extend deep into the microvasculature of the brain leading to vascular rupture and subsequent micro-hemorrhages, endothelial denudation and thrombotic obstruction (Henskens et al., 2008; O'Rourke, 2007). White matter hyperintensities in tracts supplied by the anterior and middle cerebral arteries have been related to functional impairments, indicating a relationship between cerebrovascular pathology, white matter change and cognitive ability (Wang et al., 2011).

Despite much advancement in the understanding of arterial stiffness, the role of arterial stiffness in structural brain changes and as a clinical correlate or predictor of cognitive decline is still unclear. Clinically, arterial stiffness could be related to accelerated cognitive aging and cognitive impairment and decline due to small vessel disease and could be a risk factor for dementia. In order to address the possible aetiological and clinical role of arterial stiffness in cognitive aging and related structural brain changes, we conducted a systematic review combining both clinical and brain-imaging data. This systematic review has three aims: (1) to examine the associations between arterial stiffness and structural change in the brain, specifically lacunar infarction and white matter hyperintensities as markers of small vessel disease, (2) to examine the cross-sectional and longitudinal clinical associations between arterial stiffness and cognitive impairment and decline and (3) to summarize the reported relationship between arterial stiffness and dementia to have a full understanding of the possible role of arterial stiffness on the brain. The results of this systematic review are discussed taking into account the findings of previous reviews on arterial stiffness and dementia.

2. Methods

A search of Medline, PubMed and PsycInfo was conducted until February 2012 using the following terms: "arterial stiffness", "arterial stiffening", "arterial ageing", "pulse wave velocity", "augmentation index", "PWV", "pulse pressure" or "arterial compliance." For literature dealing with brain structure, the following terms were combined with arterial stiffness terms: "brain", "leukoaraiosis", "small vessel", "white matter hyperintensities", "white matter", "lacunar infarct", "microvascular", "MRI", "cerebral" or "infarction". For literature dealing with cognition, the previously listed terms were combined with one of the following: "cognition", "cognitive decline", "cognitive function", "brain", "mini mental state examination", or "memory". In this review we included articles in English that examined arterial stiffness and cognition with their primary aim to identify the relationship between these two constructs. Articles examining arterial stiffness with another condition such as diabetes mellitus, renal failure or hypertension were excluded. Similarly, studies that examined levels of arterial stiffness in patients who had clinical dementia were excluded, as a systematic review on this topic was recently published on this topic. Studies that examined subjects with subjective memory complaints were included due to the review's focus on cognitive decline. Longitudinal studies were only included if they had at least 2 measurements, taken at least 12 months apart. The following measures of arterial stiffness were considered acceptable: Carotid-femoral pulse wave velocity (cf-PWV), brachial-ankle pulse wave velocity (ba-PWV), aortic pulse wave velocity, aortic pulse pressure and augmentation index. Cf-PWV has been established as the gold-standard non-invasive measure of arterial stiffness (Laurent et al., 2006). The measurement is determined via applanation tonometry an estimation of the velocity of the propagation of the forward and backward pressure waves between the carotid and femoral arteries (Lacolley et al., 2008). While (cf-PWV) is the gold standard, PWV may also be measured at other points in the arterial tree such as brachial-ankle or at the aorta directly via ultrasound and both these measures were included due to observed correlations with cf-PWV of 0.75–0.89 (Sugawara et al., 2010) and 0.68 (Vappou et al., 2011) respectively. The indirect measurements of arterial stiffness of aortic pulse pressure and augmentation index were also included as they have been identified as predictive of cardiovascular mortality (Vlachopoulos et al., 2010). Pulse pressure is measured as systolic blood pressure minus diastolic blood pressure (Waldstein et al., 2007), and augmentation index is the augmentation pressure (a measure of the contribution of wave reflection to systolic arterial pressure) divided by pulse pressure as taken as a percentage (Janner et al., 2012). Papers examining small vessel disease were only included if they used magnetic-resonance imaging. The initial search identified fifty-one relevant studies, twenty-nine of which met the above inclusion criteria. The rest were excluded due to examining cohorts defined by the existence of pre-existing health problems such metabolic disease. However, the meta-analyses and systematic reviews already published examining the relationship between arterial stiffness and dementia are also summarized.

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