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Review article

Association of Physical, Psychological and Psychosocial Attributes With Arterial Stiffness in Cardiovascular Disorders: A Systematic Literature Review

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The prevalence of cardiovascular diseases is increasing exponentially in the industrialized world. According to the World Health Organization, 17.8 million people died in 2019 as a result of cardiovascular diseases (CVD), accounting for 31.0% of all fatalities worldwide. Even though CVD is more common in low and middle-income countries, it is responsible for three-quarters of all cardiovascular-related deaths worldwide. The most common attributes for the occurrence of CVD are the physical, psychological, and psychosocial factors. Arterial stiffness, which is a precursor of CVD, is most commonly affected by said factors and serves as a predictor for CVD diagnosis, treatment, and prevention. The purpose of this article is to learn more about the relationship between arterial stiffness and the physical, psychological, and psychosocial characteristics of cardiovascular diseases. In addition to proposed ways to lower the co-morbidities following CVD. PubMed, Medline, and Web of Science were used for the present review. Only articles published between 1988 and 2022 that discussed physical, psychological, and psychosocial characteristics were considered. A narrative discussion is used to extract and review the information from the selected articles. Several factors related to arterial stiffness and cardiovascular illness have been reviewed, and data has been compiled. This review proposed recommendations and a list of linked factors for prevention and to lower morbidity of cardiovascular illness.

Keywords: Behavioral Psychology, Cardiovascular disease, Obesity, Physical activity, Sedentary behavior

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INTRODUCTION

Cardiovascular disorders (CVD) likewise heart diseases and stroke are paramount for annual deaths across the world with also a higher incidence in India [1]. In 2019, World Health Organization (WHO) reported 17.8 million people died with CVD accounting for 31.0% of all deaths worldwide [2]. Thus, CVD was responsible for 38.0% of the 17.0 million premature fatalities. The burden of CVD is higher in poor and middle income nations accounting for three-fourth of all CVD fatalities worldwide. WHO estimates by 2030 India will have a CVD death rate of more than 35.0% [1] as CVD strikes 6-10 years early in Indian population and with proceed higher mortality rate in comparison to western population [1]. In the last four decades, the incidence of Coronary Artery Disease has surged by 300.0% and further rising at an alarming rate of 5%-6% per year in the Indian population [1]. Prevalence of CVD in the rural and urban India population has surged from 1.6% to 7.4% and 1.0% to 13.2% respectively [2]. The exponential economy explosion and urbanization emerging as substantial lifestyle transformer is a incorporating sink in physical activity and energy disbursement; shoot-up in the pro-atherogenic diet and tobacco use [3]. In addition, hazardous lifestyle danger among Indian population with 15.0% population smoking tobacco, 4.3 liters of pure alcohol consumed per capita, more than one fifth (21.0%) population suffering from hypertensive disorder, cardinal determinant of heart attack/failure, kidney disease and/ or stroke make the condition worse [4].

PHYSICAL FACTORS FOR ARTERIAL STIFFNESS

Sedentary behavior/Physical inactivity are recognized element for CVD costing $53.8 billion in 2013 worldwide with an estimated 13.4 million disability-adjusted life year lost [5]. Low levels of physical exercise and sedentary behaviour have both been linked to arterial stiffness. Studies suggest arterial stiffness to increase with age in both sedentary as well as physically active people. But, in physically active people the rate of change is much slower than sedentary individuals. Fernberg et al. [6], 2021 and Horta et al. [7], 2015 conducted self-report studies on younger populations in Sweden and Brazil, finding reduced arterial stiffness to be inversely related to moderate and vigorous physical activity while sedentary behaviour to be positively related. Ahmadi-Abhari et al. [8], 2017 found avoiding sedentary lifestyle and engaging in high levels of moderate to vigorous physical activity substantially link to a slowing of the age-related progression of aortic stiffness. Funck et al. [9], 2016 documented insufficient physical activity to be associated with increased arterial stiffness in both type II diabetes and healthy patients. Stamatelopoulos et al. [10], 2020 discovered mean Pulse Wave Velocity (PWV) values to drop linearly with increasing intensity of physical exercise in normal weight postmenopausal women. In addition, physical exercise was also linked to lower Body Mass Index (BMI), Low Density Lipoproteins (LDL), cholesterol, and triglyceride levels and insulin resistance. Nosova et al. [11], 2014 found inactivity to cause vascular "deconditioning" state defined by decreased endothelial function leading to arterial stiffness and increased arterial tone.

PSYCHOLOGOCAL FACTORS FOR ARTERIAL STIFFNESS

Smoking has long been recognized as a significant risk factor in development and progression of CVD. The pathophysiological process of vascular injuries following smoking is anticipated to be changes in hemostatic components of endothelial function and blood lipid profile. Even minor changes in the dynamic/elastic vessel-wall characteristics act as a powerful mechanism [12]. Studies in the healthy smokers show smoking to cause changes in functional variables such arterial dispensability and blood pressure. Smoking in hypertensive patient increases risk of CVD with acute increases in arterial stiffness, blood pressure, and heart rate in response to smoking may increase the strain on the arterial and left-ventricular walls as well as alter the distribution of circumferential and tensile stress [13]. Smoking causes plaque rupture and an acute ischemic event by causing an increase in the load on the plaque.

Studies also confirm the link between arterial stiffness and smoking habits in both normotensive and hypertensive young and old people (Fig. 1). Rehill et al. [14], 2006 found long-term tobacco smoking to be linked to endothelial dysfunction and an elevated Augmented Index (Alx). Furthermore after 4 weeks of quitting smoking there may be partial reversibility of the Alx in long-term smokers. The effect on endothelial dysfunction in long-term smokers, particularly young smokers has also been supported by Binder et al. [15], 2008. According to Vlachopoulos et al. [16], 2004, smoking cigar increases acute stiffness of major arteries and wave reflection and thus, is not a safe alternative to cigarette. Furthermore, in 2007, Rhee et al. [17], confirmed long-term smoking among male smokers with hypertension to acquire increased aortic stiffness and blood pressure significantly. In comparison to smokers with no history of hypertension, these effects last longer. In the same year, Jatoi et al. [18], discovered significant linear link between smoking status and arterial stiffness utilising Pulse wave velocity (PWV) and enhanced index with age, sex, BMI, heart rate, and mean

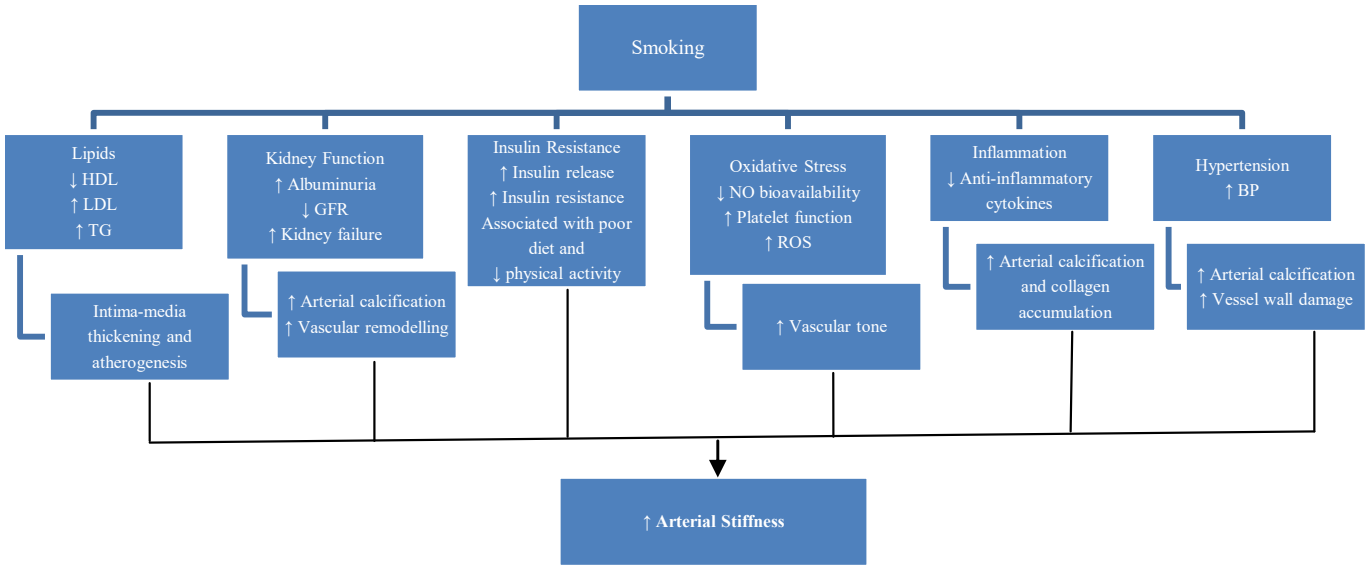

Fig. 1. Process of smoking-induced escalation in arterial stiffness.
TG: Triglycerides, LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein, GFR: Glomerular Filtration Rate, ROS: Reactive Oxygen Species, NO: Nitric Oxide, BP: Blood Pressure.

arterial pressure. According to Kim et al. [19], 2005, cigarette smoking raises systolic and diastolic blood pressure, heart rate and Brachial ankle PWV significantly at 5 minutes in both chronic smokers and non smokers before returning to normal at 15 and 30 minutes respectively. Furthermore, this study also found that chronic smokers reflect higher systolic blood pressure and brachial ankle PWV than non-smokers.

In addition to smoking alcohol consumption has well established link with hypertension, the most common cause of cardiovascular illness. Physiological activation of the endothelium to discharge endothelin or suppression of endothelium-dependent nitric oxide generation are two prominent mechanisms that raise blood pressure with consumption of alcohol [20]. Observational studies support light to moderate alcohol having a cardiac preventive impact in adults over the age of 40 years and predict J-shaped association between alcohol and arterial stiffness lowering the risk of CVD. In contrast, a study found moderate alcohol consumption to lower risk of CVD and ischemic stroke but increased the risk of subarachnoid haemorrhage in middle-aged women [21]. But, van den Elzen et al. [22], 2005 found moderate alcohol consumption to affect vascular stiffness particularly in younger women. Similarly, Oda et al. [23], 2017 found that even moderate alcohol consumption (140 gram per week) impaired endothelial and vascular functions in men. But Oda et al. [24], 2020 documented that little amount of alcohol had no deadly effect on women but excessive alcohol use impaired the endothelium function in women.

On the flipside, heavy and long-term alcohol consumption not only negates the benefits, but also puts the aortic stiffness at risk. van Trijp [25] study on younger males with average age of 28 years found heavy drinkers to pose significantly higher Alx, a precursor of arterial stiffness. Charakida et al. [26], supported the same with their study in 2019 which found that drinking methods having a significant impact on arterial stiffness with excessive intensity in contrast to frequency of drinking, resulting in negative effects on PWV. Furthermore, Tanaka [27], 2016 found Japanese men consuming more than 46 grams of alcohol per day were well associated with a lower mean value of percentage of endothelium dependent flow mediated vasodilation compared to those who had never drunk. Furthermore there was no discernible link between the beneficial effects of light alcohol consumption on endothelial function in this study.

In today's world obesity has emerged as an independent risk factor for CVD, chronic kidney disease and cognitive decline. It is caused by a diet high in saturated fat, highly processed carbohydrates and incorporation of a sedentary lifestyle [28]. The loss of cushioning effect is inextricably connected to an increase in female susceptibility to vascular stiffness [29]. In obese adults, endothelium and vascular smooth muscle cell hardening, immune cell dysfunction, perivascular adipose tissue inflammation, and extracellular matrix remodelling enhance the arterial rigidity [30]. This has been backed up by studies. Logan et al. [31], 2020 found a significant link between waist hip ratio and augmented index and elasticity of smaller arteries in adults below the age of 55 years. Furthermore, researchers discovered no link between BMI and AIx, as well as the elasticity of smaller arteries. Zebekakis et al. [32], 2005 discovered flexibility of muscle arteries decreases and diameter increases in

both genders over a wide range of age groups with a higher BMI. Safar et al. [33], 2006 discovered metabolic syndrome including BMI, dyslipidemia, hyperglycemia and hypertension to be is closely linked to increased aortic rigidity as people get older in age. Strasser et al. [34], 2015 found central obesity and visceral fat index to be strongly linked to PWV and arterial stiffness in the middle-aged population. To substantiate the preceding correlation, in a meta-analysis, Li et al. [35], 2017 discovered arterial stiffness to be recognised as a cardiovascular danger which increases in patients with obesity who have no symptoms of heart disease. Dyslipidemia such as elevated LDL cholesterol and triglycerides or low levels of High-Density Lipoprotein (HDL) (cholesterol, and/or total cholesterol are an established risk factor which play a crucial role in the development and progression of vascular disease such as CVD and ischemic stroke. Even among animals, hypercholesterolaemia induced by a cholesterol-rich diet leads to an initial reduction in arterial stiffness followed by a progressive increase over time which can be reversed by lowering serum cholesterol [36]. Studies have noted that large quantity of small dense low-density lipoprotein-cholestrol-C and disproportion of triglycerides and HDL-C may be an instigating factor for the progression of arterial stiffness in healthy population. Further it has been documented that cholesterol particularly oxidised LDL cholesterol has a range of non-atheromatous direct actions on the artery wall that can cause arterial stiffness. Peroxynitrite production and a generalised condition of exaggerated oxidative stress are also caused by oxidised LDL cholesterol, both of which can damage elastin directly [37].

Ferrier et al. [38], 2002 noticed a rigorous decline in cholesterol to be favorable in the treatment of ischemic cardiac disease population and normal lipid levels with depletion in arterial stiffness in larger arteries. To add to the evidence, a review published by Wilkinson and Cockcroft [39], 2007 found link between plasma cholesterol and the stiffness of the major arteries. To back this another study published by Li et al. [40], 2018 found ratio of small dense low density lipoprotein-cholesterol to triglycerides or high density lipoprotein-cholesterol to predict arterial stiffness progression in normotensive subjects and that a higher ratio was directly linked to a higher risk of arterial stiffness.

PSYCHOSOCIAL FACTORS FOR ARTERIAL STIFFNESS

Psychosocial factors have also been linked to CVD especially with individuals experiencing persistent work stress, social isolation and depression. In today's environment, mental stress is a novel risk factor for CVD that has been linked to left ventricular dysfunction, myocardial ischemia and infarction, and even sudden cardiac death in otherwise healthy people [41]. The specific nature of its pathways that mediate the link between psychosocial stress and cardiovascular risk is still unknown. Though several explanations have been proposed to explain the process that causes the same, acute psychological stress according to some theories induces transitory endothelial dysfunction which contributes to this risk [42]. In addition, stress-related inflammatory reactions too is likely to play a vital role. Low socioeconomic level has been linked to chronic inflammation and an increased risk of CVD in adults [43]. Vlachopoulos et al. [44], 2006 documented acute mental stress to cause sustained increase in aortic stiffness and wave reflections in nineteen healthy people. Similarly, Ellins et al. [45], 2008 noted inflammatory reactions to acute psychological stress are linked to structural alterations in the artery wall in otherwise healthy people. Looking into anxiety is a substantial and independent driver of arterial stiffness [46]. A brief period of mild to moderate stress not inducing lasting alterations in autonomic function reflect considerable negative effects on arterial stiffness in women [47]. In the year 2020, Kume et al. [48], found mental stress to increase arterial stiffness in a variety of segments in young male adults but this increase was not uniform across all segments. Lipman et al. [49], 2002 documented middle-aged and older individuals, presented with larger arterial pressure responses to mental stress are associated with increased carotid stiffness and poorer arterial baroreflex sensitivity.

CONCLUSION

Cardiovascular diseases are prevalent in all sections of the society across the globe (Table 1). With modification in lifestyle namely reducing psychosocial factors, cessation from smoking and alcohol consumption, reducing the prevalence of obesity and promotion of healthy eating and physical activity do control morbidity due to cardiovascular illness.

NOTES

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Table 1. Presentation of articles according to the objectives, methodology, results, conclusion and PEDro scoring

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Fernberg et al. (2021) [6]	To present data on Physical activity (PA) and time spent sedentary, in the population of Swedish, young adults. Secondly, to explore the association between PA and Arterial stiffness (AS).	Self-reported healthy, non-smoking, Swedish, young adults, between the ages of 18-25 yrs were included in the study. The cross-sectional study used Lifestyle, Biomarkers and Atherosclerosis. The daily PA was objectively measured with an accelerometer for 1 wk. The study measured the Pulse wave velocity (PWV) and Augmentation index (AIx) using applanation tonometry.	76% of the participants spent approximately at least 30 min per d in the recommended Moderate and vigorous physical activity (MVPA). Lower AS was associated with more MVPA and total PA in the total population.	The study highlighted the health-enhancing possibilities of time spent in PA on the vascular function, measured as PWV and AIx. In public health perspective it is highly relevant to expand preventive efforts apart the high-risk groups and encourage young adults to be physically active.	6
Horta et al. (2015) [7]	The study objectively measured PA and sedentary-time are associated with arterial stiffness in Brazilian young adults.	Cross-sectional analysis method was used on the participants of the 1982 Pelotas (Brazil) birth cohort and followed-up from birth to 30 yrs of age. PA was assessed as the average acceleration (mg), time spent in MVPA – min/d and sedentary time (min/d) were calculated from acceleration data. Portable ultrasound was used to assess the carotid-femoral pulse wave velocity (cf-PWV) (m/s). Systolic blood pressure (SBP) and Diastolic blood pressure (DBP), Waist circumference (WC) and body mass index (BMI) were analyzed as possible mediators. Analysis was done using multiple linear regression and g-computation formula.	PWV was significantly lower in the two highest quartiles of overall PA (0.26 m/s) compared with the lowest quartile. Participants in the highest quartile of sedentary time had 0.39 m/s higher PWV (95% CI: 0.20; 0.57) than those in the lowest quartile. Individuals achieving ≥ 30 min/d in MVPA had lower PWV (β = –0.35; 95% CI: –0.56; –0.14). Mutually adjusted analyses between MVPA and sedentary time and PWV changed the coefficients, although results from sedentary time remained more consistent. WC captured 44% of the association between MVPA and PWV. DBP explained 46% of the association between acceleration and PWV.	PA was inversely related to PWV in young adults, whereas sedentary time was directly correlated with the PWV.	5
Ahmadi-Abhari et al. (2017) [8]	Physical activity, sedentary behavior, and long-term changes in aortic stiffness: The whitehall II study.	Data was extracted from the Whitehall II cohort study. About 10,308 male and female aged 35 to 55 yrs, were recruited to the study in 1985 (response rate was 73%). Participants were followed up with questionnaire surveys and clinical examinations after every 4 to 5 yrs. For analysis baseline was first measured for PWV at the 2008-2009 clinical assessment. The study sample included 5,196 participants who had at least 1 measurement of PA, and PWV assessed at the 2008-2009 (n = 4,347) or the 2012-2013 (n = 4,485) clinics.	A smaller 5-yr increase in PWV was observed for each additional hr/wk spent in sports activity. The study result documented that walking, housework, gardening, or do-it-yourself activities were not strongly correlated with aortic stiffening. Each additional hr/wk spent in sitting was correlated with faster PWV progression in models adjusted for PA. Improving the level of PA over time was correlated with a smaller subsequent increase in PWV in comparison to no change in the level of activity.	The study concluded that higher levels of moderate-to-vigorous PA and avoiding sedentary behavior were each correlated with a decline in the age-related progression of aortic stiffness independent to the conventional vascular risk factors.	4

Table 1. Continued 1

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Funck et al. (2016) [9]	Low PA is associated with increased AS in patients recently diagnosed with type 2 diabetes.	A cross-sectional study included 100 patients with type 2 diabetes and 100 sex- and age-matched controls. AS (cf-PWV) was measured using the SphygmoCor device (AtCor Medical) and PA was assessed by an accelerometer (counts per minute [cpm), Actiheart [CamNtech]) worn by the participants for up to 6 d. High versus low levels of PA was defined according to the median level of activity.	Smokers (n=50) and non-smokers (n=50) were included in the final analysis. Participants with low PA had higher cf-PWV compared to participants with high PA: (i) Patients and controls combined: 9.3 ± 1.7 m/s vs. 7.8 ± 1.5 m/s, $p < 0.001$; (ii) Patients with diabetes: 9.5 ± 1.8 m/s vs. 8.3 ± 1.6 m/s, $p = 0.02$ and (iii) Controls: 9.0 ± 1.4 m/s vs. 7.7 ± 1.4 m/s, $p < 0.01$). The difference remained remarkable after adjustment for other outcome measures of cf-PWV including whole body fat percentage ($p < 0.01$). No remarkable interaction between diabetes and the effect of low activity was observed.	Low PA is related with increased AS in patients recently diagnosed with type 2 diabetes and in healthy control group.	7
Stamatelopoulos et al. (2020) [10]	To investigate the effect of different levels of intensity of PA on metabolic and vascular profile in healthy postmenopausal women.	This cross-sectional study had healthy postmenopausal women who were enrolled from the Menopause Clinic of Aretaieion Hospital. Women who visited the clinic for the first time between September 2015 and July 2019 were asked to participate in this study. A routine evaluation program which included breast mammography, gynecological examination, and Papanicolaou smear, as well as evaluation of renal/thyroid/liver function was done for the study participants. 625 healthy women were selected to participate in this study after following the exclusion criteria.	Mean values of PWV decreased linearly with increasing intensity of PA. In non-obese postmenopausal women, PWV values associated with: (a) the total number of metabolic equivalent tasks (METs) (b-coefficient = –0.261, $p = .002$) as well as with SBP; (b) or with the number of moderate METs (b-coefficient = 0.192, $p = 0.025$) as well as with age and SBP.	This study provided evidence that PA is related with a better cardiometabolic profile and lower AS in postmenopausal women. This correlation was more evident in lean postmenopausal women.	6
Nosova et al. (2014) [11]	Short-term exposure to inactivity would lead to endothelial dysfunction, AS, and escalate vascular inflammation.	5 healthy subjects underwent five d of bed rest (BR) to simulate inactivity. Vascular function, inflammation, and metabolism were measured before BR, daily during BR, and two d after BR recovery period. Isocaloric diet was followed throughout the period.	BR led to remarkable decline in brachial artery and femoral artery flow-mediated vasodilation (FMD). The central augmentation index aggravated with BR. DBP increased (58 ± 7 mmHg versus 62 ± 7 mmHg, $p = 0.02$), however no change in SBP and heart rate (HR) noticed. 15-Hydroxyeicosatetraenoic acid, an arachidonic acid metabolite, increased but the other inflammatory and metabolic biomarkers remained unchanged.	The study findings showed that acute exposure to sedentary behavior results in decline in endothelial function, AS, enhanced DBP, and an increase in 15-hydroxyeicosatetraenoic acid. Further, study speculated that inactivity promotes a vascular “deconditioning” state characterized by impaired endothelial function, leading to AS and increased arterial tone.	00

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Table 1. Continued 2

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Rehill et al. (2006) [14]	The investigate the effect of chronic cigarette smoking on arterial wave reflection (study 1) and smoking cessation on pulse wave analysis (study 2).	Smokers (n=50) and non-smokers in the study 1. Study 2 included 20 volunteer participants from the stop smoking clinic at the Royal Hallamshire Hospital. Systemic AIx and cf-PWV were measured using the SphygmoCor system. Brachial blood pressure (BP) (Omron 705-CP-E), AIx and PWV were recorded at a single visit in study 1. Measurements in study 2 were done for the variables on "quit d" and four weeks later.	In study 1, AIx was remarkably at the higher end in smokers in comparison to the nonsmokers. Multiple regression analysis showed a significant interaction among AIx and age, DBP, smoking status ($p < 0.001$), blood glucose ($p = 0.045$) and weight ($p = 0.049$). In study 2, AIx remarkably declined after 4 wks of abstinence in successful quitters ($n = 10$) when compared with relapsed smokers ($n = 4$). PWV did not reach significance in either study.	Chronic tobacco smoking is correlated with endothelial dysfunction and enhanced AIx in participants of a wide age range free from additional cardiovascular risk factors, partially reversible after 4 wks of smoking cessation.	5
Binder et al. (2008) [15]	The objective of the study was to investigate the effect of chronic smoking on AS at a peripheral site using pulse wave analysis.	The study included the 42 non-smokers having average age of 20.2 +/– 1.3 yr and 45 smokers having average age of 24.3 +/– 2.4 yr. 4 parameters including stiffness index (SI), reflection index (RI), crest time (CT) and interwave distance (IWD) were assessed by means of an adapted device based on pletysmographic principles that transform volume changes to voltage changes.	SI corresponding to PWV was 0.64 m/s higher in smokers than in non-smokers (7.25 +/– 0.53 m/s versus 7.89 +/– 0.73 m/s, $p < 0.001$). RI was remarkably high in smokers than in non-smokers. IWD for non-smokers was 8.01 +/– 0.13%, in smokers study documented an increase in 16% (9.21 +/– 0.83% [$p < 0.001$]). The study further detected a marginal incline in CT in smokers when compared to non-smokers.	Chronic tobacco smoking is correlated with endothelial dysfunction. All the assessed outcome measures were found to be elevated in smokers. The results of the study suggested that the deleterious effect of cigarette smoking on the vascular system can be found even in young smokers having history of smoking less than 10 yrs.	5
Vlachopoulos et al. (2004) [16]	The study investigated the acute effect of cigar smoking on aortic elastic properties and wave reflection.	This study studied the effect of smoking one cigar in twelve healthy subjects according to a randomized, sham procedure-controlled, cross-over design. Aortic stiffness was assessed with carotid–femoral PWV and wave reflection with AIx of the aortic pressure waveform.	Cigar smoking provoked remarkable increase in PWV (by 0.80 m/sec, $p = 0.001$) denoting a rise in aortic stiffness. AIx remarkably elevated (by 6.1%, $p < 0.05$) denoting a rise of wave reflections. These rise in AS indices were evident promptly after the initiation of cigar smoking and lasted throughout the duration of the study (2 hrs). Concurrently, both radial and aortic systolic, and pulse pressure elevated remarkably throughout the study.	The present study showed for the first time that cigar smoking escalates acutely stiffness of large arteries and wave reflection, thus revealing that it is not a safe alternative to cigarettes.	5

Table 1. Continued 3

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Rhee et al. (2007) [17]	The study investigated the acute effects of cigarette smoking on arterial stiffness and BP in hypertensive male smokers.	HR, brachial and ankle BP, and PWV were measured in twenty- two hypertensive male smokers (HTs) and in thirty normotensive male smokers (NTs) before and 5, 10, and 15 min after smoking one cigarette (nicotine content, 0.9 mg).	The result of the study showed that smoking induced acute rise of HR, brachial BP, and heart-femoral pulse wave velocity (hfPWV) in NTs and HTs ($p < 0.05$). Ankle systolic BP and femoral-ankle PWV were acutely raised in HTs ($p < 0.05$), but not in NTs. In HTs, brachial SBP and hfPWV at 15 mins were higher than at baseline ($p < 0.05$). An acute rise in hfPWV in the HTs was remarkable ($p = 0.025$) after adjustment for total cholesterol, time-dependent HR, and brachial mean arterial pressure (MAP, but acute changes of other PWVs lost statistical significance.	Cigarette smoking acutely causes rise in aortic stiffness and BP in male smokers with hypertension, and the effects persist longer than in male smokers without hypertension.	7
Jatoi et al. (2007) [18]	The objective of the study to evaluate the Impact of Smoking and Smoking Cessation on AS and Aortic Wave Reflection in Hypertension.	The study compared non-treated patients with essential hypertension (n = 554) between the age of 18 to 80 yrs classified as current smokers (n = 150), ex-smokers (n = 136), and nonsmokers (n = 268). Ex-smokers were further divided into < 1 yr, > 1 and < 10 yrs, and >10 yrs of smoking cessation. Measurements included aortic stiffness, recorded as PWV, wave reflection (AIx), and transit time (TR).	Current and ex-smokers had remarkably higher PWV and AIx in comparison to non-smokers, whereas TR was found to be less both in current and ex-smokers in comparison to nonsmokers. The study found a remarkable linear relationship between smoking status and PWV ($p < 0.001$), AIx ($p < 0.001$), and TR ($p < 0.001$), even after adjusting for age, sex, MAP, HR, and BMI. In ex-smokers, duration of smoking cessation had a significant linear relationship with improvement in PWV ($p < 0.001$), AIx ($p < 0.001$), and TR ($p < 0.001$), with AS parameters returning to non-significant levels after a decade of smoking cessation.	The study concluded that non-treated essential hypertensive population with history of chronic cigarette smoking is correlated with raised aortic stiffness and wave reflection, which are reversible with smoking cessation, although it may take more than a decade to see levels of nonsmokers. The one of the underlying mechanism behind the rise in the cardiovascular events in hypertensive patients may be chronic smoking.	6
Kim et al. (2005) [19]	The study objective is to examine the acute and chronic effects of smoking on AS by measuring brachial-ankle pulse wave velocity (baPWV) using an oscillometric method.	All healthy male subjects (chronic smokers, n = 40, 30.3 yrs old vs. non-smokers, n = 40, 28.3 yrs old). Smoked two cigarettes (nicotine 1.5 mg) within 10 mins and measured BP, HR and baPWV at baseline, 5, 15, 30, 45 and 60 mins and compared with control group (n = 20, 29.3 yrs old).	SBP was found to be high in chronic smokers than non-smokers or controls. Smoking elevates the SBP and DBP and HR remarkably at 5 mins in both chronic smokers and non-smokers as compared with baseline levels or control group and returned to baseline level after 15 mins. Pulse pressure did not elevate significantly. baPWV increased significantly in both chronic smokers and non-smokers at 5 mins and remained at higher end for 30 mins compared with control group. Smoking elevated baPWV to a greater extent in chronic smokers than in non-smokers.	Acutely, cigarette smoking elevated the BP, HR and baPWV in chronic smokers and non-smokers. These effects were more pronounced in chronic smokers in comparison to the non-smokers. The study findings suggested that cigarette smoking have adverse effects on cardiovascular system by stiffening arteries.	5

Table 1. Continued 4

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
van den Elzen et al. (2005) [22]	The present study examined whether a relation between alcohol intake and aortic stiffness is already present at a younger age.	The cohort study analyzed the cross-sectional data with men and women aged 28 yrs were stratified by gender (240 men and 283 women). A questionnaire was used to derive the alcohol intake and aortic stiffness was analyzed by PWV.	In women an alcoholic beverage intake of > / = 1 glass/d is correlated with a 0.36 m/s (95% CI, –0.58 to –0.14) lower PWV when compared with non-drinkers. In men alcohol intake is inversely proportional to PWV, but this was not significant. These findings were independent of age, BP and HR.	The concluded that intake of moderate quantity of alcohol may affect vascular stiffness at an early age, notably in women. These findings may be viewed as compatible with a vascular protective effect of alcohol that expresses well before the occurrence of symptomatic cardiovascular disease.	6
Oda et al. (2017) [23]	The objective of this study was to estimate the effects of dose-dependent alcohol consumption on endothelial function.	The study measured FMD in 2,734 men aged 21-81 yrs who provided information on alcohol intake at three general hospitals. The subjects were divided into five groups; non-drinkers (0 g/wk), light drinkers (> 0 to 140 g/wk), moderate drinkers (> 140 to 280 g/wk), heavy drinkers (> 280 to 420 g/wk), and excessive heavy drinkers (> 420 g/wk).	FMD showed a gradual decline in accordance with alcohol consumption in the entire study population (non-drinkers, 6.6% ± 3.4%; light drinkers, 6.2% ± 3.0%; moderate drinkers, 6.0% ± 3.0%; heavy drinkers, 5.5% ± 2.9%; excessive heavy drinkers, 5.3% ± 3.0%; $p < 0.001$). There was a remarkable difference in FMD between the light alcohol drinker group and the non-drinker group ($p = 0.015$). After adjustment for other risk factors, the odds of having FMD in the lowest quartile was found to be significantly increased in the 4 drinker groups than in the non-drinker group: light (OR, 1.38; 95% CI, 1.10 to 1.75), moderate (OR, 1.36; 95% CI, 1.01 to 1.82), heavy (OR, 2.05; 95% CI, 1.46 to 2.87), excessive (OR, 2.04; 95% CI, 1.43 to 2.89).	The study suggests that FMD is impaired in relation to alcohol consumption and that FMD is remarkably lower even in light alcohol drinkers than in non-drinkers. Alcohol intake per se may be harmful for vascular function.	7
Oda et al. (2020) [24]	Endothelial function is preserved in light to moderate alcohol drinkers but is impaired in heavy drinkers in women: flow-mediated dilation Japan study.	This study measured FMD in 702 women aged 17-86 yrs who provided information on alcohol consumption. The subjects were divided into four groups: non-drinkers (0 g/wk), light drinkers (> 0 g to 140 g/wk), moderate drinkers (> 140 g to 280 g/wk) and heavy drinkers (> 280 g/wk). There was no significant difference in FMD among the four groups.	The study compared fifty moderate drinkers and fifty non-drinkers matched for age and medical histories and twenty two heavy drinkers and twenty two non-drinkers in matched pair analysis. There was no remarkable difference in FMD between moderate drinkers and non-drinkers (8.2% ± 4.3% vs. 8.1 ± 3.5, $p = 0.91$), while FMD in heavy drinkers were found to be remarkably lower in comparison to non-drinkers (5.9% ± 2.5% vs. 8.9% ± 3.5%, $p = 0.002$).	These findings suggest that heavy alcohol consumption is correlated with endothelial dysfunction but that light to moderate alcohol consumption is not correlated with endothelial dysfunction in women.	7

Table 1. Continued 5

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
van Trijp et al. (2005) [25]	The objective of the study was to study the effect of alcohol consumption on the AIx, a measure of arterial wave reflection in a population of healthy young men.	329 men (mean age 28 yrs) from the Atherosclerosis Risk in Young Adults-study were studied. The level of alcohol consumption and risk factors for cardiovascular (CV) disease were determined. The AIx was estimated by radial applanation tonometry using a Sphygmocor device. The correlation between alcohol intake level and AIx was determined using linear regression models.	The result of the study documented a positive graded relation between alcohol intake and AIx. Subjects who did not drink, who drank 1 to 2 glasses/d, or who drank ≥ 3 glasses of alcohol/d had, respectively, a –0.6% (95% CI –4.2, 3.0), 0.2% (95% CI –2.6, 2.9), and 3.4% (95% CI 0.2, 6.7) difference in AIx compared with very light drinkers (< 1 glass/d). After adjustment for current smoking, BMI and HDL-cholesterol, those consuming > 3 glasses/d had a 3.29% (95% CI 0.01, 6.7) higher AIx compared with those consuming < 1 glass/d.	In a population of healthy young men, the heaviest drinkers had a remarkably higher AIx. This finding supports the evidence that elevating the amount of alcohol consumption is related to vascular damage at young age.	6
Charakida et al. (2019) [26]	To determine the impact of smoking and alcohol exposure during adolescence on AS at 17 yrs.	This study used the questionnaires method to assess the smoking and alcohol use at 13, 15, and 17 yrs in 1,266 participants from the Avon Longitudinal Study of Parents and Children study. Smoking status (smokers and non-smoker) and intensity ("high" ≥ 100, "moderate" 20-99, and "low or never" < 20 cigarettes in lifetime) were ascertained. Alcohol consumption is classified on the basis of frequency (low or high) and intensity of drinking (light intensity [LI] < 2), medium (medium intensity [MI] 3-9), and heavy (heavy intensity [HI] > 10 drinks on a typical drinking d).	The study result proved that exposure to higher smoking was correlated with higher PWV in comparison to non-smokers [5.81 ± 0.725 vs. 5.71 ± 0.677 m/s, mean adjusted difference 0.211 (0.087-0.334) m/s, p = 0.001]. Participants who quit smoking had similar PWV to never smokers (p = 0.160). PWV had escalated in high-intensity drinkers [HI 5.85 ± 0.8 vs. LI 5.67 ± 0.604 m/s, mean adjusted difference 0.266 (0.055-0.476) m/s, p = 0.013]. There was an additive effect of smoking intensity and alcohol intensity, so that 'high' smokers who were also HI drinkers had higher PWV in comparison to never-smokers and LI drinkers [mean adjusted increase 0.603 (0.229-0.978) m/s, p = 0.002].	The study concluded that smoking exposure even at low levels and intensity of alcohol use was correlated individually and together with raised AS. Public health strategies need to prevent adoption of these habits in adolescence to preserve or restore arterial health.	5
Tanaka et al. (2016) [27]	To determine the potential effect of alcohol consumption on endothelial function.	This study included four hundred four men aged 30-79 yrs, were recruited from residents in two communities under the Circulatory Risk in Communities study in 2013 and 2014. The individuals were asked about the frequency and volume of alcohol beverages and converted the data into grams of ethanol per d. Endothelial function was assessed by brachial artery FMD measurements during reactive hyperemia.	Individuals who drank ≥ 46 gm/d ethanol had a lower age-adjusted mean %FMD than non-drinkers (p < 0.01). Compared with non-drinkers, the age-adjusted ORs (95% CI) of low %FMD (< 5.3%) for former, light (< 23.0 gm/d ethanol), moderate (23.0-45.9 gm/d ethanol), and heavy (≥ 46.0 gm/d ethanol) drinkers were 1.61 (0.67-3.89), 0.84 (0.43-1.66), 1.09 (0.52-2.25), and 2.99 (1.56-5.70), respectively. The corresponding multivariable-adjusted ORs were 1.76 (0.69-4.50), 0.86 (0.42-1.76), 0.98 (0.45-2.12), and 2.39 (1.15-4.95), respectively.	The study concluded that heavy alcohol consumption may be an independent risk factor of endothelial dysfunction in Japanese men.	6

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Table 1. Continued 6

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Logan et al. (2020) [31]	The purpose of this study was to investigate the relationship between AS and obesity.	AS was defined as high AIx and low elasticity (C1, large artery elasticity; C2, small artery elasticity) in participants enrolled in the Multi-Ethnic Study of Atherosclerosis at baseline. The study compared the AIx, C1, and C2 by BMI (< 25, 25-29.9, 30-39.9, ≥ 40 kg/m^2) and waist-hip ratio (WHR) (< 0.85, 0.85-0.99, ≥ 1). The obesity–AS correlation was tested across 10-yr age intervals among 6,177 participants (62 ± 10 years old, 52% female).	The analysed the AIx of 5,815 participants, whose complete data was collected. The average AIx was 14.88% ± 2.2%, C1 was 13.36 ± 5.6 mL/mmHg × 10, and C2 was 4.48 ± 2.8 mL/mmHg × 100.	This study demonstrated that the participants with higher BMI and WHR were more likely to have less AS defined as low AIx and high C1 and C2, independent of common cardiovascular disease (CVD) risk factors, alluding to the obesity paradox. The illustrated the complexity of the relationship between body habitus and AS. Considering the prevalence of obesity and the prognostic value of AS in the future development of CVD, explicating the elusive correlation between obesity and AS will have remarkable implications for the early detection and prevention of CVD.	7
Zebekakis et al. (2005) [32]	To investigate whether the relationship between AS and BMI was consistent across an age range from 10 to 86 yrs.	The study used the cross-sectional population-based design, randomly enrolled 1,306 individuals (median age 43.9 yrs; 50.5% women). The carotid, femoral and brachial arteries and cf-PWV were measured with wall-tracking ultrasound system.	Before and after adjustment, arterial diameter increased with BMI in all territories, with an opposite trend for arterial distensibility. In men and women, the relationships of brachial and femoral properties with BMI were consistent across the whole age range. In men and women, carotid distensibility declined more with BMI at younger age than older age. In middle-aged and older women, but not in men of any age, PWV elevated with higher BMI.	Across a wide age range, the diameter and stiffness of muscular arteries accelerated with higher BMI. In elastic arteries, the relationship between AS and BMI was more complex and varied with sex and age.	8
Safar et al. (2006) [33]	The purpose of the study was to evaluate whether a clustering of metabolic risk factors might accelerate the progression of AS with age in subjects with metabolic syndrome (MS).	476 subjects were classified at baseline according to their number of CV risk factors (from zero to three and more), after adjustment for smoking habits. The CV risk factors were: hypertension, body mass index, dyslipidemia, hypertriglyceridemia, and hyperglycemia, classified according to traditional criterions. Subjects were followed for six yrs and had, at the beginning and end of the survey, determinations of BP, HR, and aortic PWV.	At baseline, BP, HR, plasma creatinine, and PWV were significantly higher ($p < 0.001$) in the group with three and more CV risk factors than in groups with zero to two risk factors. During the follow-up, the increase in PWV, but not in pulse pressure, was remarkably higher ($p < 0.01$) in the group with three and more risk factors (i.e., metabolic syndrome) than in other groups. Results were unmodified after adjustments for age, gender, baseline values, drug treatment, smoking habits, and mean arterial pressure.	The study concluded that MS is correlated with an increased progression of aortic stiffness with age, supporting premature senescence in these patients.	6

Table 1. Continued 7

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Strasser et al. (2015) [34]	To determine whether abdominal and visceral adipose tissue may be a better predictor of AS than general obesity in middle-aged adults.	A total of one hundred forty six participants (76 men, 70 women; 50 years) were recruited. The automatic vascular screening device (Omron VP-1000 plus) was used to measure BP simultaneously in the arms and ankles and to determine AS by PWV.	The result of the study documented that both carotid-femoral PWV and brachial-ankle PWV were remarkably associated with BMI (both $p < 0.05$) but not with body fat percentage. Measures of abdominal obesity, including WC and visceral fat mass (via DXA), were strongly correlated with PWV and remained positively associated with AS after adjustment for age and gender.	Abdominal obesity and visceral fat are linked with large artery stiffness. These findings support the importance of adiposity measures as a risk factor for arterial stiffening in middle-aged adults.	6
Li et al. (2017) [35]	The study performed a meta-analysis evaluating the impact of obesity/ overweightness on AS in healthy subjects.	The meta-analysis study conducted literature search using databases (eg, MEDLINE, EMBASE) and citations cross-referenced. Studies evaluating the relationship between obesity/ overweightness and cf-PWV, baPWV, and AIx were systematically searched. A total of 10 studies (1,124 obese/ overweight subjects, 1,884 controls) were included.	The meta-analysis found that compared to controls, obese/overweight subjects had remarkably higher cf-PWV (standardized mean difference [SMD] 0.50 m/s; 95%CI 0.15, 0.86; $p = 0.005$), baPWV (SMD 0.41 m/s; 95% CI 0.08, 0.74; $p = 0.014$), and AIx (SMD 1.02; 95%CI 0.16, 1.87; $p < 0.0001$). When analyzing 'high quality' studies, the difference in AS among obese/ overweight subjects and controls remain (SMD 0.73 m/s; 95%CI 0.16, 1.30; $p = 0.013$).	AS, a recognized marker of CV risk, is raised in obese/overweight subjects without overt cardiovascular diseases.	6
Ferrier et al. (2002) [38]	To investigate the effects of intensive cholesterol reduction on large AS and BP in normolipidemic patients with isolated systolic hypertension (ISH).	In a randomized, double-blinded, cross-over study design, twenty-two patients with stage I ISH received 3 mos of atorvastatin therapy (80 mg/d) and three mos of placebo treatment. Systemic arterial compliance was measured non-invasively using carotid applanation tonometry and Doppler velocimetry of the ascending aorta.	The result of the study found that Atorvastatin intervention declined the level of total and low-density lipoprotein cholesterol and triglyceride levels by 36% +/– 2% ($p < 0.001$), 48 +/- 3% ($p < 0.001$) and 23% +/– 5% ($p = 0.003$), respectively, and raised high density lipoprotein cholesterol by 7% +/– 3% ($p = 0.03$). Systemic arterial compliance was more after treatment (placebo vs. atorvastatin: 0.36 +/– 0.03 vs. 0.43 +/– 0.05 mL/mmHg, $p = 0.03$). Brachial systolic BP was lower after atorvastatin treatment (154 +/– 3 vs. 148 +/– 2 mmHg, $p = 0.03$), as were mean (111 +/– 2 vs. 107 +/– 2 mmHg, $p = 0.04$) and diastolic BP (83 +/– 1 vs. 81 +/– 2 mmHg, $p = 0.04$). There was a trend toward a depletion in pulse pressure (71 +/– 3 vs. 67 +/– 2 mmHg, $p = 0.08$).	The study concluded that intensive cholesterol depletion may be beneficial in the treatment of patients with ISH and normal lipid levels, through a reduction in large AS.	7

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Table 1. Continued 8

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Wilkinson et al. (2007) [39]	The aim of this review is to focus on the relationship between cholesterol, lipids and AS.	This review covered the three main areas: epidemiological relationships, likely mechanisms and the potential benefits of lipid-lowering.	The review documented that most of the available data suggested that there is probably a positive correlation between AS and cholesterol. However, the current studies are far from conclusive and interpretation is hampered by the use of multiple different techniques, indices and small sample sizes.	The review concluded that the majority of the available evidence suggested a positive correlation between plasma cholesterol and the stiffness of the large arteries. Lipid-lowering agents appeared to remarkably reduce AS.	4
Li et al. (2018) [40]	The study designed to identify feasible indicators for predicting AS progression.	The study followed up 816 normotensive participants without diabetes or CVD for nearly 5 yrs. Cholesterol parameters, ratios and other clinical and laboratory data were collected at baseline. cf-PWV were measured at baseline and the end of follow-up.	PWV progression subjects had higher values of PWV parameters, small dense low-density lipoprotein- cholesterol (sdLDL-C) and triglycerides [TG]/high-density lipoprotein [HDL]-C ratio. sdLDL-C and TG/HDL-C were remarkably correlated with all PWV parameters. Multiple regression models showed that sdLDL-C was closely correlated with follow-up PWV (β = 0.222, $p < 0.001$) and ΔPWV (β = 0.275, $p < 0.001$). TG/HDL-C was only one cholesterol ratios that correlated with all PWV parameters. sdLDL-C (OR = 2.070, 95% CI: 1.162 to 3.688, p = 0.014) and TG/HDL-C (OR = 1.355, 95% CI: 1.136 to 1.617, p = 0.001) could remarkably determine the progression of PWV after correction for covariates. High small dense low-density lipoprotein-cholesterol quantiles subjects were more likely to develop AS progression than low quantiles (Tertiles 3 vs. Tertiles 1, RR = 2.867, 95%CI: 1.106 to 7.434, p = 0.03).	The study documented that sdLDL-C and TG/HDL-C ratio can independently predict AS progression in normotensive subjects, and high level sdLDL-C and TG/HDL-C ratio were associated with a higher risk of AS.	6

Table 1. Continued 9

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Vlachopoulos et al. (2006) [44]	The purpose of this study was to assess the effect of acute mental stress on aortic stiffness and wave reflections.	A randomized, sham-procedure-controlled, crossover design studies the effect of a mental arithmetic test for the assessment in nineteen healthy individuals. cf-PWV and AIx were measured as indices of aortic stiffness and wave reflections, respectively.	The result of the study proved that mental stress induced a sustained rise in central systolic and pulse pressure throughout the whole study (systolic: by 7.5 mmHg, $p < 0.05$; pulse: by 5.7 mmHg, $p < 0.01$). The rise in peripheral systolic and pulse pressure was not significant throughout the study, but only when their peak values were compared with baseline (systolic: by 6.2 mmHg, peak at 0 mins; pulse: by 6.6 mmHg, peak at 5 mins, $p < 0.05$ for both). There was a sustained rise in PWV (by 0.57 m/s, $p < 0.005$) throughout the study denoting a sustained rise in aortic stiffness. Similarly, AIx showed a sustained rise with mental stress (by 6.16%, $p < 0.05$) denoting raised wave reflections from the periphery.	The study concluded that acute mental stress results in a prolonged rise in aortic stiffness and wave reflections. Given the important pathophysiologic and prognostic role of these parameters, the results of this study provide important mechanistic links between acute mental stress and increased CV risk.	6
Ellins et al. (2008) [45]	The study assessed the relationship between carotid AS and inflammatory responses to acute psychophysiologic stress.	The study collected the data from the participants in the Whitehall II epidemiological cohort who took part in the psychobiology substudy. The psychobiology substudy involved two hundred twenty-eight volunteers (123 men, 105 women) who underwent psychophysiological testing in 1999-2000.	The result of the study effect was remarkable in the analyses of systolic and diastolic BP ($F(3432) = 249.9$ and 265.6 respectively.	The study concluded that inflammatory responses to acute psychological stress are correlated with structural changes in the arterial wall in apparently healthy subjects.	5
Kume et al. (2020) [48]	The study aimed to examine the impact of acute mental stress on segmental AS.	In the main experiment, seventeen young male subjects (mean age, 20.1 ± 0.7 yrs) performed a 5-min MS and control task in a random order. PWV from the heart to brachium pulse wave velocity (hbPWV) and the heart-ankle pulse wave velocity (haPWV), PWV between the brachial artery and the baPWV, and the cardio-ankle vascular index (CAVI) were simultaneously measured at baseline and 5, 15, and 30 mins after the task.	Compared to baseline values, hbPWV, baPWV, haPWV, and CAVI significantly increased until 30 mins after the MS task, whereas these variables did not significantly change following the control task. At 5 and 30 mins after the MS task, %age changes from baseline were remarkably higher in hbPWV (+5.2 ± 4.4 and 6.6% ± 4.9%) than in baPWV (+2.2 ± 2.1 and 2.2% ± 2.0%) or haPWV (+3.6 ± 2.6 and 4.3% ± 2.9%) and were also significantly lower in brachial artery and ankle PWV than in heart to ankle PWV.	These findings suggest that acute MS elicits a rise in AS in different segments and this AS is not uniform among the segments.	6

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Table 1. Continued 10

Authors (yr)	Objective	Intervention	Results	Conclusion	PEDro Score
Lipman et al. (2002) [49]	This study examined the relationship of pressor responses during MS to AS and baroreflex sensitivity.	The study screened the subjects and informed of possible risks. Subjects (n = 24), aged 51 to 86 yrs (63.0 ± 8.0), were negative for the following: current coronary artery disease, hypertension (BP > 140/90), diabetes, or BMI > 30. In addition, each subject's response to a Bruce graded exercise test to volitional fatigue was within normal limits, and carotid ultrasound examination demonstrated no evidence of disease.	Both the speech and math tasks induced remarkable rise in BP and HR (MAP and HR vs. baseline, $p \leq 0.004$). Although the responses to the speech and math tasks were qualitatively similar within individuals, as previously reported the responses were generally greatest during speech. The average rise in MAP during speech (25.31 ± 2.81) was substantial than that of math (15.65 ± 2.01; $p \leq 0.004$), and the average rise in HR during the speech task (13.42 ± 1.77) was significantly higher than that observed during the math task (9.32 ± 1.26; $p \leq 0.004$). Carotid stiffness ranged from 7.5 to 30.2 and baroreflex sensitivity ranged from 1.2 to 11.1 ms/mmHg. Although these two parameters demonstrated a strong inverse relation ($r = -0.55$; $p = 0.006$), neither parameter correlated with subject age. The baseline MAP and HR were significantly related to baroreflex sensitivity.	The study concluded that substantial rise in arterial pressure responses to mental stress modestly relate to greater carotid stiffness and lower arterial baroreflex sensitivity in middle-aged and older individuals. Considering the strong association between carotid stiffness and baroreflex sensitivity, an augmented pressor response may be one manifestation of diminished baroreflex function caused by vascular stiffness.	6

mg: Milligrams, m/s: Meter per second, CI: Confidence interval, min.: Minute, mm: millimeter, mL: millilitre, mmHg: millimetre(s) of mercury, SI: International System of Units, RR: relative risk, Kg: kilogram, vs.: versus, HDL: high-density lipoprotein, Ors: odds ratios, g: gram, MS: mental stress, DXA: dual-energy x-ray absorptiometry.
ΔPWV Change from baseline PWV to follow-up PWV.

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