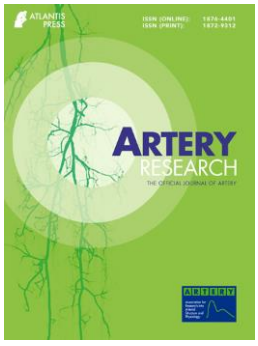

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Associations between smoking and alcohol use and arterial elasticity in patients with newly diagnosed essential hypertension: A cross-sectional study

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KEYWORDS
Smoking;
Alcohol consumption;
Essential hypertension;
Arterial stiffness

Abstract *Objective:* To assess the relationship between smoking and alcohol use (separately and combined) on arterial stiffness in patients with essential hypertension.
Materials and Methods: We assessed never-treated newly diagnosed patients with essential hypertension (n = 446) aged 18–80 years (52% males). Measurements included aortic stiffness, assessed as pulse wave velocity (PWV) (Complior), wave reflection (augmentation index [AIx]), and transit time [T_R] SphygmoCor).
Results: The 446 patients were categorized as: non-smokers (n = 204), ex-smokers (n = 121), current smokers (n = 121), non-drinkers (n = 59), moderate drinkers (n = 281), heavy drinkers (n = 106). Both AIx and PWV were significantly higher in current smokers than in ex-smokers and non-smokers [($p < 0.02$), ($p < 0.01$), respectively]. AIx and PWV were higher in non-drinkers followed by heavy drinkers and moderate drinkers [($p < 0.003$), ($p < 0.008$), respectively]. Among current and ex-smokers there was no significant difference in PWV or AIx by alcohol consumption (p = NS), while in non-smokers AIx was significantly ($p < 0.001$) higher in the non-drinkers followed by heavy drinkers and those with moderate alcohol consumption and PWV was significantly ($p < 0.001$) higher in heavy drinkers followed by non-drinkers and those with moderate alcohol consumption.

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Conclusion: Moderate alcohol consumption exerts a favorable effect on arterial stiffness in essential hypertensive patients. However, the combined effect of smoking cancels this favorable effect.

Introduction

Elastic arterial stiffening can be accelerated in the presence of essential hypertension, while that can produce arterial stiffness through functional and structural mechanisms.[1,2] Whether arterial stiffness is the result of arterial hypertension or its cause remains a controversy. However, it is commonly accepted that the determinants of large artery stiffness and hypertension may represent known risk factors, all of which may lead to vascular changes and affect both the heart and the kidneys.

Smoking is a major modifiable cardiovascular risk factor leading to alterations in endothelial function, lipid profile and dynamic properties of the arterial wall.[3] It has been reported that the compliance of both large and medium sized arteries decreases immediately after smoking one cigarette.[4,5] Similarly, Vlachopoulos et al.[6] have shown a significant undesirable effect of smoking and caffeine on arterial stiffness. Furthermore, smoking leads to a direct increase in both peripheral and central blood pressure.[7] Smoking is known to cause severe accumulation of exogenous active oxygen and free radicals resulting in a chronic increase in oxidative stress thus leading to endothelial dysfunction, vasoconstriction and vascular inflammation, three mechanisms that are known to induce arterial stiffness.[8,9]

The effect of alcohol consumption on the cardiovascular system demonstrates a J-shaped association.[10] Specifically, non-drinkers and heavy drinkers are at higher risk, while moderate alcohol consumption exerts a protective effect. It appears that moderate alcohol intake prevents atherosclerosis through beneficial effects on lipoprotein metabolism, hemostasis and microvascular inflammation, mechanisms which in turn are directly associated with arterial stiffness.[11–13]

The purpose of the present study was to assess the effect of smoking and alcohol separately, on a cohort of patients with newly diagnosed essential hypertension, and to further evaluate their combined effect on large artery elastic properties.

Materials and methods

A total of 446 untreated subjects aged 18–80 years (47.8 ± 0.6 years, mean $\pm$ SD), 52% male, undergoing assessment for hypertension, were studied. None of the patients had secondary hypertension, coronary artery disease, valvular heart disorders, dysrhythmias, diabetes, heart failure, or renal impairment, and none were taking any vasoactive drugs. Current smokers were defined as those who reported smoking >1 cigarette per day for >1 year, nonsmokers as those who had never smoked, and former or ex-smokers as those who had stopped smoking >1 month before examination. Alcohol consumption was evaluated from patients' self-reported average weekly alcohol intake. From this estimate, the ethanol equivalent (g/week) was then calculated, and subjects were categorized as: non-drinkers, moderate drinkers [≤14 units/week (female) and ≤21 units/week (male)]; and heavy drinkers [≥15 units/week (female) and ≥22 units/week (male)].

Body weight, height, waist, and hip measurements were recorded for each patient. Body mass index (BMI) was calculated as body weight (kilograms) divided by height (meters squared), and the waist: hip ratio was calculated. Fasting venous blood samples were drawn, and total cholesterol, high-density lipoprotein cholesterol, triglycerides, plasma glucose, and serum creatinine were measured by standard methods. The subjects gave informed consent, and the study had institutional ethics committee permission.

Blood pressure measurements

Subjects rested in a supine position for 5 min in a quiet room at 22 °C before the baseline hemodynamic measurements were obtained. Brachial blood pressure (BP) and heart rate were measured in the right arm with an automated digital oscillometric sphygmomanometer (Omron, Model HEM 705-CP, Omron Corporation, Shimogyo-ku, Kyoto, Japan). Three readings separated by 1-min intervals were taken, and the mean was used for analysis. Peripheral pulse pressure was calculated as the difference between brachial systolic and diastolic BP[14]

Measures of arterial stiffness and wave reflection

By using radial applanation tonometry, the aortic pressure waveform was derived by using a previously validated transfer function relating radial to aortic pressure waveform within the system software of the SphygmoCor (SphygmoCor, AtCor Medical, Version8.0) by an operator, and 2 measurements were performed in each subject. Ascending aortic pressures and the AIx were derived from the aortic pressure waveform, as described previously[15,16,18] Transit time (T_R) was measured from the foot of the wave to the inflection point on the aortic pressure wave form. Carotid–femoral PWV was measured with an automated system (Artech Medical) using the foot-to-foot method. The carotid and femoral waveforms were acquired simultaneously with 2 pressure-sensitive transducers, and the T_R of the pulse was calculated using the system software. The distance between the 2 arterial sites was measured on the body using a tape measure, and PWV

was calculated as the distance divided by time (meters per second). At least 12 successive readings were used for analysis to cover a complete respiratory cycle.

Statistical analysis

Data were analyzed using JMP (version 10). PWV, AIx, and T_R approximated normal distributions. Data are expressed as mean ± SD or 95% confidence intervals, with $P < 0.05$ considered significant. Mean differences in PWV, AIx, and TR between non-smokers, ex-smokers, and current smokers and the effects of alcohol consumption (non-drinkers, moderate drinkers and heavy drinkers) were assessed using ANOVA.

Results

The study population consisted of 446 patients with newly diagnosed essential hypertension, mean age 48.7 ± 0.6 years, 52.4% men, with mean SBP and DBP of 158 ± 10 and 94 ± 0.5 mmHg, respectively. There were 204 non-smokers, 121 ex-smokers, and 121 current smokers. When categorised according to their alcohol intake there were 59 non-drinkers, 281 moderate drinkers and 106 heavy drinkers. The study population characteristics are presented in Tables 1 and 2.

Arterial stiffness indices were compared among the three smoking categories. Univariate analysis revealed significantly higher levels of AIx in current smokers than in ex-smokers or non-smokers ($p < 0.02$). Similarly, PWV was higher among current smokers than in ex-smokers or non-smokers ($p < 0.018$). Finally, PP was significantly higher in current smokers than in ex-smokers and non-smokers ($p < 0.02$) while no significant difference was noted in HR, in SBP and DBP (p = NS). Multivariate analysis revealed that the observed differences in Aix and PWV remained significant after adjustment for HR, SBP, and DBP (Table 3).

Arterial stiffness indices were compared according to alcohol consumption. AIx was significantly higher in the group of non-drinkers followed by heavy drinkers and moderate drinking intake ($p < 0.003$) Similarly, PWV was higher in non-drinkers than in heavy drinkers and moderate drinkers ($p < 0.008$). PP was higher in non-drinkers than in heavy or moderate drinkers ($p < 0.05$). There was no difference in HR among the three groups. SBP was significantly higher in heavy drinkers followed by non-drinkers and moderate drinkers ($p < 0.02$), while DBP was higher in heavy drinkers than in moderate drinkers or non-drinkers ($p < 0.008$).

Multivariate analysis was then performed and the model was adjusted for HR, SBP and DBP. Aix remained significantly higher among non-drinkers followed by heavy drinkers and moderate drinkers ($p < 0.005$), while higher levels of PWV were noted in non-drinkers followed by heavy drinkers and moderate drinkers ($p < 0.01$) see Table 4.

Finally, we created a model combining alcohol consumption and smoking habits to assess the association with arterial stiffness parameters . Among non-smokers AIx was significantly higher for non-drinkers followed by heavy drinkers and moderate drinkers ($p < 0.0001$), and PWV was higher in heavy drinkers followed by non-drinkers and moderate drinkers ($p < 0.02$). For current smokers no significant difference was noted by alcohol use for either AIx or PWV. Finally, among ex-smokers there was no significant difference between alcohol use groups for AIx however, PWV was higher in non-drinkers followed by heavy drinkers and moderate drinkers $p < 0.02$), see Table 5.

Multivariate analysis was then performed and the model was adjusted for HR, SBP and DBP. Among non-smokers AIx remained significantly ($p < 0.001$) higher in the non-drinkers followed by heavy-drinkers and those with moderate alcohol consumption. PWV remained significantly ($p < 0.001$) higher in heavy drinkers followed by non-drinkers and moderate drinkers. For current smokers no significant difference was noted in AIx and PWV according to alcohol consumption (p = NS). Finally, among ex-smokers no significant difference was observed according to alcohol consumption, while PWV remained significantly higher ($p < 0.02$) in non-drinkers compared to heavy drinkers and moderate drinkers.

Table 1 Clinical characteristics of study population (n = 446).

Variables	(Mean ± SD)
Age (Years)	48.7 ± 0.6
Gender (M:F)	52:48 (%)
Height (cm)	169 ± 0.46
Weight (kg)	82.1 ± 0.81
BMI (kg/m^2)	28.72 ± 0.23
Waist (cm)	95.13 ± 0.8
Hip (cm)	105 ± 0.65
Smoking & Alcohol Status	
Non-Smoker (%)	204 (45.8)
Ex-Smoker (%)	121 (27.1)
Current-Smoker (%)	121 (27.1)
Alcohol Nil	59 (13.6)
Alcohol Moderate	281 (63)
Alcohol Heavy	106 (23.8)
Hemodynamic status	
Heart Rate (bpm)	71.6 ± 0.5
Brachial SBP (mmHg)	158 ± 1
Brachial DBP (mmHg)	94 ± 0.5
Pulse Pressure (mmHg)	64 ± 1
Augmentation Index (%)	27.28 ± 0.55
Pulse Wave Velocity (m/s)	10.36 ± 0.11
Transit Time (m/s)	134 ± 0.6
Biochemistry	
Total cholesterol (mmol/L)	5.2 ± 0.05
HDL cholesterol (mmol/L)	1.35 ± 0.02
LDL cholesterol (mmol/L)	3.02 ± 0.05
VLDL cholesterol (mmol/L)	0.98 ± 0.03
Triglycerides (mmol/L)	1.73 ± 0.06
GGT	40.1 ± 0.7
AST	24.45 ± 0.57
Creatinine	87.07 ± 0.6
Glucose	5.28 ± 0.03
MCV	91.3 ± 0.03

Discussion

The present study aimed to assess the relationships between smoking and alcohol habits (separately and

Table 2 Clinical characteristics of study population categorized according to Alcohol status (n = 446).

Variables	Non-drinkers n = 59	Moderate drinkers (F:<14U, M<21U) n = 281	Heavy drinkers (F:>14U, M>21U) n = 106	*P* Value
Age (Years)	55.3 ± 1.7	47.7 ± 0.8	47.6 ± 1.3	0.0002
Gender (M:F)	35:65	49:51	75:25	0.0476
Height (cm)	165.2 ± 1.3	168.8± 0.6	172 ± 1	<0.0001
Weight (kg)	80.2 ± 2.3	81.8 ± 1.1	86 ± 1.7	NS
BMI (kg/m2)	29.19 ± 0.64	28.52 ± 0.3	29 ± 0.48	NS
Waist (cm)	95 ± 2.8	94.2 ± 1	97.4 ± 1.6	NS
Hip (cm)	105.8 ± 1.8	104.4 ± 0.8	106 ± 1.3	NS
Smoking-status Non: Ex-Smoker: Current-Smoker (%)	59:24:17	48:26:26	33:31:36	0.0136
Alcohol-Status (Average Units/wk)	–	8 ± 0.65	39.22 ± 1.06	–
Heart rate (bpm)	71 ± 1	71 ± 1	70 ± 1	NS
Brachial SBP (mmHg)	160 ± 3	156 ± 1	162 ± 2	0.0275
Brachial DBP (mmHg)	92 ± 1	93 ± 1	97 ± 1	0.0087
Pulse pressure (mmHg)	68 ± 2	63 ± 1	65 ± 2	0.05
Augmentation Index (%)	31.9 ± 1.5	26.3 ± 0.7	27.3 ± 1.1	0.0034
Pulse Wave Velocity (m/s)	10.96 ± 0.29	10.1 ± 0.14	10.65 ± 0.21	0.0084
Transit Time (m/s)	131 ± 1.7	135 ± 0.8	135 ± 1.2	NS
Total cholesterol (mmol/L)	5.3 ± 0.1	5.1 ± 1	5.4 ± 0.1	0.05
HDL cholesterol (mmol/L)	1.39 ± 0.05	1.33 ± 0.02	1.37 ± 0.04	NS
LDL cholesterol (mmol/L)	3.3 ± 0.14	2.96 ± 0.06	3.05 ± 0.1	NS
VLDL cholesterol (mmol/L)	0.81 ± 0.1	0.96 ± 0.04	1.1 ± 0.07	0.038
Triglycerides (mmol/L)	1.48 ± 0.17	1.67 ± 0.08	2.1 ± 0.13	0.0042
GGT	30.9 ± 4.4	33 ± 2	63.8 ± 3.3	<0.0001
AST	22.2 ± 1.5	23.2 ± 0.7	29.1 ± 1.1	<0.0001
Creatinine	84.3 ± 1.7	87 ± 0.7	88.8 ± 1.2	NS
Glucose	5.21 ± 0.09	5.26 ± 0.04	5.39 ± 0.06	NS
Hemoglobin	14.06 ± 0.3	14.62 ± 0.11	14.98 ± 0.2	0.0184
MCV	88.25 ± 0.8	91 ± 0.35	93.87 ± 1.6	<0.0001

combined) on arterial stiffness in newly diagnosed patients with essential hypertension. We found that both Aix and PWV tended to be higher in current smokers than in ex-smokers or non-smokers independently of SBP, DBP and HR. We also found that both AIX and PWV tended to be higher values in non-drinkers followed by heavy drinkers and moderate drinkers. Finally, when the combined effect of alcohol and smoking was assessed, we found no difference in AIX or PWV non-smokers and current smokers irrespective of their alcohol consumption; while in ex-smokers PWV was higher in non-drinkers followed by heavy drinkers and moderate drinkers.

The chronic effects of smoking on arterial elastic properties have been so far controversial. A tendency for increased stiffness in the common femoral and carotid arteries has been previously reported in habitual smokers.[17] Also, an invasive study showed abnormalities of the brachial artery pressure waveform in smokers, while another study[18] demonstrated increase Aix and PWV in healthy smokers compared to non-smokers, but others[4,19] found no difference in regional arterial compliance between smokers and nonsmokers.

Various studies have also shown that smoking plays a major role in the occurrence of arteriosclerosis and arteriosclerotic disease.[20–22] Smoking leads to arteriosclerosis by inducing nitric oxide damage to vascular endothelial cells, contributing to high BP or diabetes mellitus, and stimulating coagulation and fibrinolysis pathways.[23] Recently it was reported that carotid-ankle vascular index (CAVI), a new marker of arterial stiffness, is higher in current smokers compared to ex-smokers and non smokers,[24] while in another study when comparing Ankle-brachial index (ABI) and PWV among smokers, ex-smokers and non-smokers the former presented with higher values of arterial stiffness indices and smoking cessation for a period of 12 months was associated with an improvement in arterial stiffness.[25] In the present study, when the relationship between smoking and arterial stiffness was assessed separately, the findings were similar to the previous mentioned studies: thus arterial stiffness indices were higher in current smokers followed by ex-smokers and non-smokers regardless of the peripheral hemodynamics.

While the effect of alcohol consumption on cardiovascular risk profile has been previously assessed,[10–12] pointing to a favorable impact of moderate consumption comparised with heavy or no consumption, the relative effect on arterial stiffness remains controversial. In a cross-sectional study among Japanese-American men and women the risk for high aortic PWV was lower among current drinkers and ex-drinkers than among non-drinkers,[26] while in another study including middle-aged Japanese men the incidence of aortic stiffness was not related to alcohol intake.[27] Moreover, the same group of investigators reported that alcohol is an important risk factor for the development of aortic

Table 3 Impact of smoking status on arterial stiffness in hypertensive patients (n = 446).

Variables	Non-Smokers (n = 204)	Current- Smokers (n = 121)	Ex-Smokers (n = 121)	*P* Value ANOVA
Age (Years)	46.7.±0.9	49.6 ± 1.2	51.4 ± 1.2	0.0049
Gender (M:F)	**30:59**	**139:139**	**79:27**	**< 0.01**
Height (cm)	168.2 ± 0.7	169.3 ± 0.9	170.5 ± 0.9	NS
Weight (kg)	81.7 ± 1.25	81.9 ± 1.6	84.8 ± 1.6	NS
BMI (kg/m2)	28.78 ± 0.35	28.35 ± 0.45	29 ± 0.45	NS
Waist (cm)	93.03 ± 1.17	97.05 ± 1.6	96.8 ± 1.44	0.05
Hip (cm)	104.5 ± 1	105.6 ± 1.3	105.2 ± 1.2	NS
Hemodynamic Measurements				
Heart rate (bpm)	70.4 ± 0.8	71.4 ± 1	70.3 ± 1	NS
Brachial SBP (mmHg)	155.1 ± 1.6	159.5 ± 2.1	160.4 ± 2.1	NS
Brachial DBP (mmHg)	93.5 ± 0.8	93.5 ± 1	94.6 ± 1	NS
Pulse Pressure (mmHg)	61.6 ± 1.2	66.6 ± 1.6	65.8 ± 1.6	0.0254
Augmentation Index (%)	25.8 ± 0.8	29.4 ± 1.1	27.7 ± 1	0.0246
Pulse Wave Velocity (m/s)	10.03 ± 0.16	10.71 ± 0.22	10.58 ± 0.2	0.0188
Transit Time (m/s)	136 ± 0.9	130.7 ± 1.2	134.7 ± 1.2	0.0012
Biochemistry				
Total cholesterol (mmol/L)	5.07 ± 0.07	5.4 ± 0.09	5.2 ± 0.09	0.0194
Triglycerides (mmol/L)	1.42 ± 0.09	1.99 ± 0.12	2.01 ± 0.11	<0.0001
LDL cholesterol (mmol/L)	3.12 ± 0.11	2.97 ± 0.06	3.05 ± 0.1	0.47
Creatinine	85.95 ± 0.87	86.09 ± 1.12	90.07 ± 1.15	0.0103
Glucose	5.22 ± 0.05	5.36 ± 0.06	5.31 ± 0.06	NS
Alcohol status (Units/wk)	11.17 ± 1.22	19.48 ± 1.6	14.64 ± 1.6	0.0002
MCV	91.14 ± 0.61	93.31 ± 0.54	90.25 ± 0.41	<0.0001

stiffness at an intake of more than 16 glasses of an alcoholic beverage per week.[28] On the other hand in a cross-sectional study among postmenopausal women an inverse association between alcohol consumption and aortic PWV was reported,[29] while the same investigators published another study demonstrating that among men aged 40–80 years there is a J-shaped association between alcohol consumption and PWV.[30] The Rotterdam Study provided further evidence of the favorable effect of moderate alcohol consumption on arterial stiffness.[31] The authors reported that moderate alcohol consumption in women was associated with lower arterial stiffness independently of cardiovascular risk factors and atherosclerosis. In the present study, when the effect of alcohol alone was addressed, the J-shape association was demonstrated. It appears that moderate alcohol consumption among newly diagnosed essential hypertensive patients is associated with a favorable AIx and PWV profile independent of the peripheral hemodynamics. The underlying mechanisms remain unknown; however, possible beneficial effects of moderate alcohol consumption on lipoprotein metabolism, hemostasis and microvascular inflammation along with a reduced risk for type 2 diabetes mellitus and increased insulin sensitivity may be factors.

Table 4 Spearman rank correlations of variables against alcohol consumption (n = 446).

Variables	Correlation (r)	*p*-value
Age (Years)	−0.09869	0.0374
Gender (M:F)		
Height (cm)	0.206429	<0.0001
Weight (kg)	0.140279	0.003
BMI (kg/m2)	0.037035	0.4374
Waist (cm)	0.141262	0.0110
Hip (cm)	0.048372	0.3877
Hemodynamic measurements		
Heart rate (bpm)	0.0188587	0.6954
Brachial SBP (mmHg)	0.116072	0.0143
Brachial DBP (mmHg)	0.133053	0.0049
Pulse Pressure (mmHg)	0.061222	0.1969
Augmentation Index (%)	−0.08713	0.0663
Pulse Wave Velocity (m/s)	0.042126	0.4408
Transient Time (m/s)	0.030235	0.5242
Biochemistry		
Total cholesterol (mmol/L)	0.107038	0.0292
Triglycerides cholesterol (mmol/L)	0.195164	<0.0001
Creatinine	0.089356	0.0687
Glucose	0.134585	0.006
MCV	0.30534	<0.0001

While the separate effects of smoking and alcohol consumption on arterial stiffness revealed associations in line with previous studies, the combined effects gave different findings. We observed no beneficial effect of moderate alcohol consumption on arterial stiffness among current and ex-smokers., which reflects the possible stronger effect of smoking on large artery elasticity. It appears that the detrimental effect of smoking outweighs the possible beneficial effect of other cofactors, including alcohol. The fact that in the non-smoker group the J-shaped association of alcohol and both Aix and PWV appeared again strengthens the above observation.

Table 5 Clinical characteristics of patients according to smoking and alcohol status (n = 446).

Variables	Non-smokers n = 204 (45.74%)				Current smokers n = 121 (27.13%)				Ex-Smokers n = 121 (27.13%)			
	Alcohol (Nil)	Alcohol (Moderate)	Alcohol (Heavy)	*P* Value	Alcohol (Nil)	Alcohol (Moderate)	Alcohol (Heavy)	*P* Value	Alcohol (Nil)	Alcohol (Moderate)	Alcohol (Heavy)	*P* Value
Age (Years)	52.5 ± 2.2	44.8 ± 1.1	47.9 ± 2.1	0.0061	61.8 ± 4.1	50.1 ± 1.5	45.5 ± 2.1	0.0021	57.5 ± 3.4	51 ± 1.5	49.7 ± 2.2	NS
Gender (M:F%)	17:83	43:57	44:56	NS	60:40	47:52	71:29	0.0338	64:36	62:38	79:21	0.02
Height (cm)	162.3 ± 1.7	168.1 ± 0.9	174.4 ± 1.7	<0.0001	166.6 ± 3	168.9 ± 1.1	170.7 ± 1.6	NS	171.5 ± 2.7	170 ± 1.2	171.2 ± 1.7	NS
Weight (kg)	73.1 ± 2.9	82.1 ± 1.5	88.8 ± 2.9	<0.0008	89.2 ± 6.1	79 ± 2.3	85.4 ± 3.1	NS	91.4 ± 4.3	83.9 ± 1.9	83.8 ± 2.8	NS
BMI (kg/m2)	27.6 ± 0.9	29 ± 0.4	29.2 ± 0.9	NS	32.1 ± 1.2	27.4 ± 0.6	29.2 ± 0.9	0.0195	31.1 ± 1.1	28.8 ± 0.5	28.5 ± 0.7	NS
Waist (cm)	87.7 ± 2.9	92.3 ± 1.4	100.1 ± 2.6	0.0052	98.9 ± 5.2	96.9 ± 2.3	96.8 ± 2.8	NS	106.3 ± 4	95.5 ± 1.8	95.4 ± 2.5	0.043
Hip (cm)	102.2 ± 2.3	103.9 ± 1.1	108.5 ± 2.2	NS	109.4 ± 5	105.3 ± 2.2	105 ± 2.6	NS	110.4 ± 2.8	104.6 ± 1.3	104 ± 1.8	NS
Average units/ week (n)	0	6.8 ± 0.8	39.1 ± 1.6	<0.0001	0	9.5 ± 1.7	43.7 ± 2.3	<0.0001	0	8.7 ± 1	34.21 ± 1.45	<0.0001
No Cigarettes	-	-	-	-	19 ± 3.3	17.5 ± 1.3	19.3 ± 2.1	NS	—	-	-	-
Hemodynamic measurements												
Heart rate (bpm)	71 ± 2	71 ± 1	69 ± 2	NS	74 ± 4	71 ± 1	71 ± 2	NS	69 ± 3	71 ± 1	70 ± 2	NS
Brachial SBP (mmHg)	153 ± 4	155 ± 2	158 ± 4	NS	175 ± 8	155 ± 3	163 ± 4	0.0454	167.1 ± 5.7	157.1 ± 2.5	165 ± 3.7	NS
Brachial DBP (mmHg)	91 ± 1	93 ± 1	98 ± 2	0.0209	93 ± 4	93 ± 2	95 ± 2	NS	93.8 ± 2.7	93.6 ± 1.2	97.2 ± 1.8	NS
Pulse Pressure (mmHg)	62 ± 3	62 ± 1	60 ± 3	NS	83 ± 6	64 ± 2	68 ± 3	0.0166	73.3 ± 4.5	63.5 ± 2	67.8 ± 3	NS
Augmentation Index (%)	32.4 ± 2	24.1 ± 1	25.8 ± 2	0.0013	31.7 ± 3.5	30.1 ± 1.3	27.5 ± 1.8	NS	30.9 ± 3	26.7 ± 1.3	28.5 ± 1.9	NS
Pulse Wave Velocity (m/s)	10.1 ± 0.4	9.8 ± 0.2	10.8 ± 0.3	0.0239	12.2 ± 0.8	10.7 ± 0.3	10.3 ± 0.4	NS	11.9 ± 0.6	10.2 ± 0.3	10.8 ± 0.4	0.0205
Transit Time (m/s)	129.5 ± 2.1	137.1 ± 1.1	138.4 ± 2.1	0.0033	129 ± 4	130 ± 1.5	133 ± 2.1	NS	137.7 ± 3.3	135.2 ± 1.4	132.4 ± 2.2	NS
Biochemistry												
Total Cholesterol (mmol/L)	5.1 ± 0.2	5 ± 0.1	5.3 ± 0.2	NS	5.8 ± 0.3	5.3 ± 0.1	5.5 ± 0.2	NS	5.3 ± ± 0.3	5.1 ± 0.1	5.3 ± 0.2	NS
Triglycerides (mmol/L)	1.27 ± 0.1	1.4 ± 0.1	1.7 ± 0.1	NS	1.71 ± 0.43	1.84 ± 0.17	2.38 ± 0.23	NS	1.79 ± 0.42	1.96 ± 0.19	2.21 ± 0.28	NS
Creatinine	82 ± 2	85.8 ± 1	90.5 ± 2	0.0113	85.7 ± 4.2	85.8 ± 1.5	86.7 ± 2.1	NS	90.9 ± 3.9	90.2 ± 1.5	89.6 ± 2.3	NS
Glucose	5.2 ± 0.1	5.18 ± 0.1	5.41 ± 0.1	NS	5.39 ± 0.27	5.34 ± 0.1	5.38 ± 0.14	NS	5.11 ± 0.16	5.31 ± 0.06	5.38 ± 0.1	NS
MCV	88.7 ± 0.9	90.2 ± 0.4	92 ± 1.1	NS	87.3 ± 1.7	93.1 ± 0.8	96 ± 1	0.0002	87.5 ± 2.7	90.8 ± 0.8	92.6 ± 1.1	NS

In conclusion, the present study found that smoking was associated with poorer arterial stiffness indices in patients with essential hypertension while moderate alcohol consumption was associated with less stiffness, as indicated by lower levels of both PWV and Aix. However, the combined effect of smoking and alcohol consumption was associated with a greater effect on PWV. Further studies need to be conducted in order to clarify the possible broad spectrum of the underlying mechanisms involved.

Declaration of conflicting interests

Conflict of interest: None.

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