

Impact of Smoking and Smoking Cessation on Arterial Stiffness and Aortic Wave Reflection in Hypertension

Noor A. Jatoi, Paula Jerrard-Dunne, John Feely, Azra Mahmud

Abstract—Cigarette smoking is an important modifiable cardiovascular risk factor and pathophysiological mechanisms may include a stiff vascular tree. Although smokers have stiffer arteries, whether smoking cessation is associated with reduced arterial stiffness is not known. We compared never-treated patients with essential hypertension (n=554) aged 18 to 80 years (56% females) classified as current smokers (n=150), ex-smokers (n=136), and nonsmokers (n=268). Ex-smokers were categorized into <1 year, >1 and <10 years, and >10 years of smoking cessation. Measurements included aortic stiffness, assessed as pulse wave velocity (Complior), wave reflection (augmentation index [AIx]), and transit time (T_R) (Sphygmocor). Current and ex-smokers had significantly higher pulse wave velocity and AIx compared with nonsmokers (pulse wave velocity for current smokers: 10.7 ± 0.2 ; ex-smokers: 10.6 ± 0.2 ; nonsmokers: 9.9 ± 0.1 m/s; $P < 0.001$; AIx for current smokers: 31 ± 1 ; ex-smokers: 30 ± 1 ; nonsmokers: $27 \pm 0.8\%$; $P < 0.05$), whereas T_R was lower in current and ex-smokers compared with nonsmokers (T_R for current smokers: 131 ± 1.0 ; ex-smokers: 135 ± 1 ; nonsmokers: 137 ± 0.8 m/s; $P < 0.0001$). There was a significant linear relationship between smoking status and pulse wave velocity ($P < 0.001$), AIx ($P < 0.001$), and T_R ($P < 0.001$), even after adjusting for age, sex, mean arterial pressure, heart rate, and body mass index. In ex-smokers, duration of smoking cessation had a significant linear relationship with improvement in pulse wave velocity ($P < 0.001$), AIx ($P < 0.001$), and T_R ($P < 0.001$), with arterial stiffness parameters returning to nonsignificant levels after a decade of smoking cessation. (*Hypertension*. 2007;49:981-985.)

Key Words: smoking ■ arterial stiffness ■ pulse wave velocity ■ augmentation index ■ hypertension ■ smoking cessation

Cigarette smoking is one of the most important avoidable causes of cardiovascular diseases worldwide,¹ and arterial stiffness may be one of the underlying pathophysiological mechanisms. Chronic cigarette smoking has been shown to be associated with increased arterial stiffness^{2,3} and increases immediately after smoking 1 cigarette.³ The standard measurement of arterial stiffness, aortic pulse wave velocity (PWV) in conjunction with augmentation index (AIx), an estimate of aortic wave reflection, provide a comprehensive assessment of arterial stiffness.⁴ There is evidence that both PWV^{5,6} and AIx⁷ are independent predictors of cardiovascular events.

Smoking cessation is an important lifestyle measure for the prevention of cardiovascular disease, and patients with myocardial infarction may experience as much as a 50% reduction in risk of reinfarction, sudden cardiac death, and total mortality if they quit smoking.⁸ However, the speed and magnitude of risk reduction when a smoker quits is debatable, with studies quoting 3 to 20 years of smoking cessation associated with significant risk reductions in coronary artery disease.⁸

Whether long-term smoking cessation is associated with a reduction in arterial stiffness compared with chronic smokers

is not known. Therefore, we compared differences in arterial stiffness among nonsmokers, ex-smokers, and current smokers in a cross-sectional study.

Methods

Subjects

A total of 554 untreated subjects aged 18 to 80 years (47.8 ± 0.6 years, mean \pm SEM), 56% female, 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 had smoked >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. Ex-smokers were categorized into 3 subgroups according to smoking cessation duration: those who quit cigarette smoking for <1 year, between 1 and 10 years, and >10 years.

Body weight, height, waist, and hip measurements were recorded in 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.

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Blood Pressure Measurements

Subjects rested in a supine position for 5 minutes 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 left arm with an automated digital oscillometric sphygmomanometer (Omron, Model HEM 705-CP, Omron Corporation). Three readings separated by 1-minute intervals were taken, and the mean was used for analysis. Peripheral pulse pressure was calculated as the difference between brachial systolic and diastolic BP.

Measures of Arterial Stiffness and Wave Reflection

The aortic pressure waveform was derived using radial applanation tonometry by using a previously validated transfer function relating radial to aortic pressure waveform within the system software of the Sphygmocor (SphygmoCor, AtCor Medical, Version 8.0) by a single 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.³ Transit time (T_R) was measured from the foot of the wave to the inflection point on the aortic pressure waveform. 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 by 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. The interclass correlation coefficients between the first and second measurements on the 544

subjects were 0.94 for AIx, 0.89 for T_R , and 0.84 for PWV. The coefficients of variation for AIx, T_R , and PWV were <5%. The reproducibility of the measured variables was comparable to previous studies.^{9,10}

Statistical Analysis

Data were analyzed using SPSS (version 12.0). PWV, AIx, and T_R approximated normal distributions. Data are expressed as mean \pm SEM or 95% confidence intervals, with $P < 0.05$ considered significant. Mean differences in PWV, AIx, and T_R between never smokers, ex-smokers, and current smokers and the effects of duration of smoking cessation were assessed using ANOVA with posthoc Bonferroni corrections. To determine whether ex-smokers had arterial stiffness levels intermediate between never and current smokers, linear regression analysis was used to establish the presence of a linear trend. Three models were used: (1) adjusted for age and sex; (2) adjusted for age, sex, and mean arterial pressure; and (3) additional multivariate adjustment for the other major determinants of arterial stiffness, that is, heart rate and BMI.

Results

Patient Characteristics

The clinical characteristics of the study population are shown in Table 1. The ex-smokers were 5 years older than nonsmokers ($P < 0.01$) and 3 years older than current smokers, although this did not reach statistical significance ($P = 0.09$). Although there was no difference in sex distribution for smokers, significantly more males quit smoking than females

TABLE 1. Clinical Characteristics of the Hypertensive Patients Population Categorized According to Smoking Status (n=554, Mean \pm SEM)

Variables	Smoking Status			P
	Current (n=150)	Former (n=136)	Never (n=268)	
Age, y	48.39 \pm 1	50.66 \pm 1	45.99 \pm 0.8	0.003
Sex, male:female	78:72	89:47	126:142	0.001
Height, cm	169.13 \pm 0.84	170.62 \pm 0.88	168.52 \pm 0.63	0.32
Weight, kg	81.34 \pm 1.45	84.29 \pm 1.52	81.79 \pm 1.08	0.3
BMI, kg/m ²	28.16 \pm 0.4	28.98 \pm 0.42	28.73 \pm 0.3	0.34
Waist, cm	95.85 \pm 1.45	96.95 \pm 1.36	92.92 \pm 1.04	0.04
Hip, cm	105 \pm 1.2	105 \pm 1.1	106 \pm 0.8	0.83
Waist:hip ratio	0.91 \pm 0.01	0.92 \pm 0.01	0.89 \pm 0.01	0.01
Heart rate, bpm	71.5 \pm 1.0	70.2 \pm 1.0	70.4 \pm 0.7	0.55
Brachial systolic BP, mm Hg	158 \pm 1.83	160 \pm 1.93	154 \pm 1.38	0.05
Brachial diastolic BP, mm Hg	92.78 \pm 0.93	94.53 \pm 0.98	92.69 \pm 0.70	0.27
Brachial pulse pressure, mm Hg	65.4 \pm 1.4	65.5 \pm 1.5	61.7 \pm 1.1	0.05
Mean arterial pressure, mm Hg	112.03 \pm 1.28	113.48 \pm 1.15	110.34 \pm 0.8	0.09
Aortic systolic BP, mm Hg	146.27 \pm 1.82	148.87 \pm 1.91	142.86 \pm 1.37	0.02
Aortic diastolic BP, mm Hg	94.26 \pm 0.95	95.79 \pm 0.99	94.08 \pm 0.71	0.35
Aortic pulse pressure, mm Hg	52.13 \pm 1.41	53.08 \pm 1.41	48.78 \pm 0.97	0.02
AIx, %	30.60 \pm 0.1	30.07 \pm 1.02	27.33 \pm 0.84	0.03
PWV, m/s	10.72 \pm 1.93	10.6 \pm 0.2	9.95 \pm 0.12	0.001
T_R , ms	130.96 \pm 1.05	134.63 \pm 1.03	136.83 \pm 0.82	0.0001
Total cholesterol, mmol/L	5.36 \pm 0.08	5.19 \pm 0.08	5.07 \pm 0.06	0.02
High-density lipoprotein cholesterol, mmol/L	1.34 \pm 0.03	1.33 \pm 0.03	1.34 \pm 0.02	0.92
Triglycerides, mmol/L	1.92 \pm 0.11	1.97 \pm 0.11	1.43 \pm 0.08	0.001
Fasting glucose, mmol/L	5.3 \pm 0.06	5.31 \pm 0.06	5.23 \pm 0.04	0.44
Serum creatinine, μ mol/L	86.12 \pm 1.09	89.57 \pm 1.13	86.86 \pm 0.83	0.02

TABLE 2. PWV, AIx, and T_R in Never, Ex-, and Current Smokers (Mean, 95% CI)

Model	Never Smoker (n=150)	Ex-Smoker (n=136)	Current Smoker (n=268)	P ANOVA	P Linear Trend
PWV, m/s					
Model 1*	10.3 (10.0 to 10.5)	10.5 (10.1 to 10.8)	10.9 (10.5 to 11.2)	0.02	0.006
Model 2†	10.3 (10.1 to 10.5)	10.4 (10.1 to 10.7)	10.9 (10.5 to 11.2)	0.02	0.008
Model 3‡	10.3 (10.1 to 10.6)	10.4 (10.0 to 10.7)	10.9 (10.5 to 11.2)	0.03	0.017
AIx, %					
Model 1*	28 (26 to 29)	30 (28 to 32)	30 (29 to 32)	0.04	0.015
Model 2†	28 (27 to 29)	30 (28 to 31)	30 (29 to 32)	0.05	0.016
Model 3‡	28 (27 to 29)	29 (28 to 31)	31 (29 to 32)	0.01	0.001
T _R , ms					
Model 1*	137 (135 to 138)	135 (133 to 137)	131 (129 to 133)	0.001	0.001
Model 2†	137 (135 to 138)	135 (133 to 137)	131 (129 to 133)	0.001	0.001
Model 3‡	137 (135 to 138)	135 (133 to 137)	132 (130 to 133)	0.001	0.001

The 3 models were used *(1) adjusting for age and sex; †(2) adjusting for age, sex, and mean arterial pressure; and ‡(3) with additional multivariate adjustment for mean heart rate and BMI.

(*P*<0.001). There was no significant difference in BMI among the 3 groups, although there was a trend for higher waist circumference and waist:hip ratio in ex-smokers. Total cholesterol was raised in smokers (*P*<0.05), and plasma creatinine (*P*<0.05) was higher in ex-smokers.

Smoking Status and Arterial Stiffness

Brachial and aortic systolic BP and pulse pressure were significantly lower in nonsmokers compared with both smokers and ex-smokers (*P*<0.05). For PWV, AIx, and T_R there was a direct linear relationship between smoking status and arterial stiffness, with ex-smokers having levels intermediate between current smokers and nonsmokers (Table 1). To study whether there was an independent relationship between arterial stiffness and smoking status, we constructed linear regression models for PWV, AIx, and T_R. We took PWV as the dependent variable and the independent variables included in the first model were age and sex; age, sex, and mean arterial pressure in the second model; and age, sex, mean arterial pressure, heart rate, and BMI in the third model. There was a significant linear relationship between PWV and smoking status (Table 2). Similar models with AIx and T_R as the dependent variables showed significant linear relationships with smoking status (Table 2).

Smoking Cessation Status and Arterial Stiffness

Data on duration of smoking cessation were available for 122 ex-smokers, and the demographics categorized according to smoking cessation status are given in Table 3. Ex-smokers of <1-year duration had arterial stiffness similar to current smokers, ex-smokers of 1 to 10 years in duration had intermediate levels, and arterial stiffness in ex-smokers of >10 years duration was not significantly different to that of never smokers (Figure). Linear regression models showed a significant direct relationship between the duration of smoking cessation and PWV (*P*<0.001), AIx (*P*<0.001), and T_R (*P*< 0.001) in ex-smokers, after adjusting for age, sex, BMI, and mean arterial pressure.

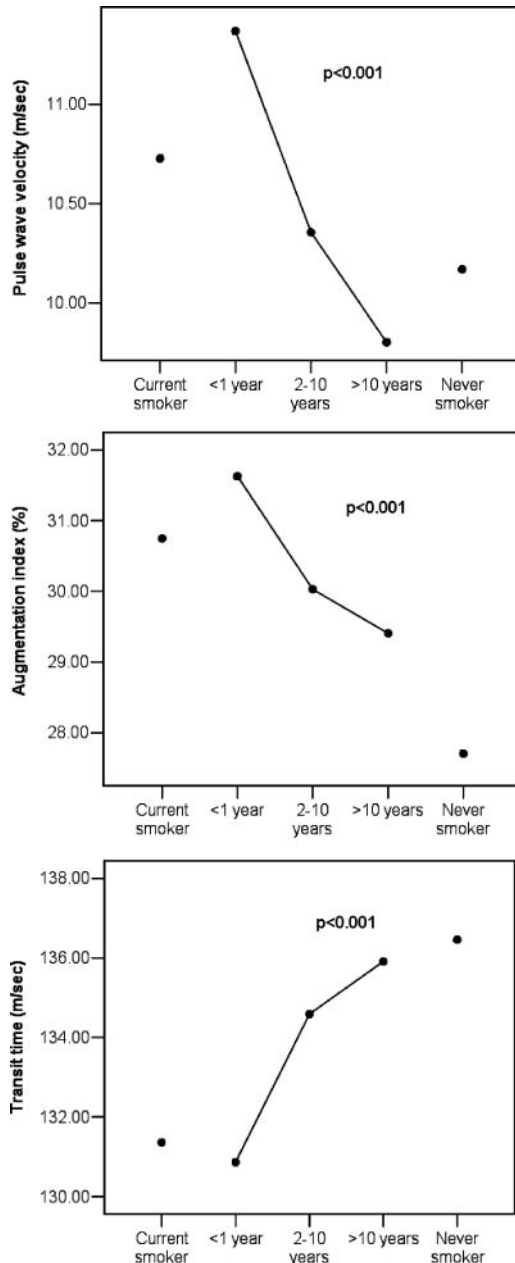
Discussion

This study confirms in a large, untreated homogenous population that there is a significant linear relationship between smoking status and PWV, AIx, and T_R, even when adjusted for covariates including age, sex, mean arterial pressure, and BMI (Table 2). Further analysis that included duration of smoking cessation showed that reversal of the deleterious effects of smoking on arterial stiffness is likely to take >10 years to achieve levels of stiffness similar to that of never smokers (Figure).

Because arterial stiffness is an independent predictor of events in hypertensive patients,⁵⁻⁷ it may be one of the mechanisms for smoking-related vascular disease in such patients. Although chronic smoking has been shown to be associated with increased stiffness in healthy subjects,^{2,3} data on the effects of smoking on arterial stiffness are lacking in an untreated hypertensive population. To the best of our knowledge, this is the first study to show that, in untreated hypertensive patients, a population characterized by already stiff vessels, chronic smoking further increases arterial stiff-

TABLE 3. Clinical Characteristics of Ex-Smokers Categorized According to Smoking Cessation Status (Mean±SEM, n=122)

Parameters	<1 y (n=22)	>1 and <10 y (n=40)	>10 y (n=60)	P
Age, y	47±3	49±2	55±1	0.01
Sex, male/female	9/13	16/24	15/45	0.19
BMI, kg/m ²	28±0.8	28±0.6	30±0.5	0.12
Waist:hip ratio	0.90±0.02	0.90±0.02	0.94±0.01	0.14
Systolic BP, mm Hg	156±5	157±3	164±3	0.15
Diastolic BP, mm Hg	92±2	94±2	96±1	0.23
Total cholesterol, mmol/L	5±0.2	5.2±0.2	5.3±0.1	0.84
Triglycerides, mmol/L	2±0.4	1.9±0.2	2±0.2	0.96
High-density lipoprotein cholesterol, mmol/L	1.40±0.07	1.3±0.05	1.3±0.04	0.50
Glucose, mmol/L	5.3±0.09	5.2±0.07	5.4±0.07	0.16



Aortic stiffness (PWV), wave reflection (AIx), and T_R against duration of smoking cessation (estimated marginal means after adjustment for age, sex, mean arterial pressure, mean heart rate, and BMI). Values for current and never smokers included for comparison.

ness. We also think that, as the increased aortic stiffness is independent of BP, the effects of hypertension and smoking on the vascular wall may be cumulative.

Although effects of smoking cessation on BP are well documented,¹¹ those on arterial stiffness parameters are variable. A comparative longitudinal study over 2 years¹² did not show any effect of smoking cessation on carotid parameters, including intimal thickness, stiffness, or distensibility in people who stopped smoking compared with smokers and nonsmokers. This was a relatively small ($n=33$) study, and the nonsmoking group included ex-smokers. In a recently published longitudinal study of 4 weeks of smoking cessa-

tion, AIx was reduced but not to the level of the nonsmokers,¹³ whereas PWV was unchanged in this short-term study. However, our findings are in agreement with the recent study by Li et al,² where, in healthy younger normotensive subjects, again in a somewhat older group, smoking cessation was associated with a significant reduction in arterial stiffness and systemic vascular resistance. Also, in agreement, they showed a reduction in arterial stiffness in former smokers to the level of nonsmokers after 10 years of smoking cessation. However, using radial pressure waveform analysis, they did not find any difference in large artery stiffness. It is possible that the measure, PWV, used in our study is more specific for a large artery (aorta), or perhaps the presence of hypertension in an older population amplified the effects of smoking on large artery stiffness.² The mechanisms involved in amelioration in arterial stiffness with smoking cessation may include lipid-soluble smoke particles,¹⁴ endothelial dysfunction,¹⁵ or vascular inflammation,¹⁶ because smoking cessation leads to reduction in levels of inflammatory markers.¹⁷ BP does not appear to be involved, because ex-smokers have higher BP levels than current smokers in our study, as seen previously,¹¹ and when adjusted for both age and BP, the association with arterial stiffness remained significant in our study (Table 2). Although it may take more than a decade to reverse these vascular changes, and the effect is relatively small, smoking cessation may help reduce cardiovascular events through amelioration in arterial stiffening even in long-term hypertensive smokers. Our results are in agreement with published data reporting cardiovascular risk reduction with smoking cessation for periods ranging from 3 to 20 years.⁸

There are certain caveats in our study. Using a cross-sectional design, we can only observe an association between vascular parameters and smoking status and cannot establish a causal relationship. Although we have adjusted for all of the major confounders in the analysis, the presence of unknown confounders cannot be ignored. For example, people who successfully quit smoking may be different from those who continue in different ways, including age, sex, psychosocial characteristics, and other factors. We have adjusted for age and sex in our analysis, because ex-smokers were older and predominantly male. A further limitation may be the misclassification of smoking status, because patients who continued to smoke may claim to have quit smoking, as we do not have data on biochemical markers of smoking in this study. However, self-reporting has been shown to be quite accurate when compared with biochemical evidence of tobacco inhalation.¹⁸ Despite these caveats, we believe that our study shows not only that smoking-induced arterial stiffness is increased in hypertensive smokers compared with nonsmokers but suggests also that these vascular changes are reversible, although it may take more than a decade for values to revert to that of never smokers. This highlights the importance of avoidance of smoking and the great need to promote smoking cessation in hypertensive patients who continue to smoke, as reduction in arterial stiffness may still be possible. However, these results need to be confirmed in a prospective longitudinal study.

Perspectives

We have shown in a large, untreated hypertensive population that chronic cigarette smoking is associated with increased 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 high aortic stiffness and wave reflection seen with chronic smoking may be 1 of the underlying mechanisms for the increased cardiovascular events observed in hypertensive patients. Therefore, considering the independent prognostic usefulness of arterial stiffness in the hypertensive population,^{5-7,19} its assessment may not only identify hypertensive patients at higher cardiovascular risk but may also be used to monitor arterial health in those who quit smoking.

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Disclosures

None.

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Correction

In the *Hypertension* article by Jatoi et al (Jatoi NA, Jerrard-Dunne P, Feely J, Mahmud A. Impact of smoking and smoking cessation on arterial stiffness and aortic wave reflection in hypertension. *Hypertension*. 2007;49:981–985), the corresponding author should be Azra Mahmud, Department of Pharmacology and Therapeutics, Trinity Centre for Health Sciences, St James's Hospital, Dublin 8, Ireland. E-mail mahmuda@tcd.ie The authors regret the error.