Acute and Chronic Effects of Smoking on the Arterial Wall Properties and the Hemodynamics in Smokers with Hypertension

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ABSTRACT

Background and Objectives: Smoking increases cardiovascular risk and it is known to acutely increase arterial stiffness in healthy smokers. We investigated the acute and chronic effects of smoking on the arterial mechanics in smokers with hypertension. Subjects and Methods: To evaluate the acute effects of smoking, the heart rate (HR), brachial blood pressure (BP), ankle systolic BP, heart-carotid pulse wave velocity (hcPWV), heart-femoral PWV (hfPWV), femoral-ankle PWV (faPWV), heart-brachial PWV (hbPWV), and the ankle-brachial index (ABI) were measured in male hypertensive smokers (n=22, 42±2 years, mean±SEM) and in normotensive smokers (n=30, 39±1 years) before and 5, 10 and 15 minutes after smoking 1 cigarette (nicotine content: 0.9 mg). To evaluate the chronic effects of smoking, we compared the HR, brachial BP, PWVs, ABI and carotid intima-media thickness (IMT) between the normotensive smokers and nonsmokers (n=32, 37±1 years), and also between the hypertensive smokers and nonsmokers (n=21, 48±2 years). Results: Smoking induced significant acute increases of the HR, brachial BP and hfPWV in the normotensive and hypertensive smokers (p<0.05), and smoking also induced significant acute increases in the faPWV and ankle systolic BP in the hypertensive smokers (p<0.05). The ABI, hcPWV and hfPWV in both groups, and the faPWV of the normotensive smokers were not changed after smoking 1 cigarette (p>0.05). The magnitude of the increase in the HR, brachial BP and hfPWV after smoking were not different between the hypertensive and normotensive smokers. There was no significant differences (p>0.05) between the chronic normotensive smokers and the nonsmokers, and also between the hypertensive smokers and the non-smokers. Conclusion: Smoking acutely increased the HR, brachial BP and aortic stiffness in hypertensive smokers and normotensive smokers, and it had a more significant impact on the femoral arterial stiffness in the hypertensive smokers than in the normotensive smokers. However there were no significant chronic effects of smoking on the hemodynamics, arterial stiffness and carotid IMT. Our findings suggest that the acute increase in aortic and femoral arterial stiffness after smoking may contribute to the increased incidence of adverse cardiovascular events in smokers with hypertension. (Korean Circulation J 2005;35:493–499)

KEY WORDS: Smoking; Hypertension; Arterial, stiffness; Intima-media thickness.

Introduction

Smoking is known as a major risk factor in the development and progression of cardiovascular disease. Despite of the extensive research, the pathophysiological mechanisms of smoking-related vascular damage have not been fully explored. Changes in the hemostatic factors, the endothelial function and the blood lipids have been proposed as mechanisms of vascular damage. Alteration of the dynamic vessel wall properties has also been considered as an important mechanism since some investigators have demonstrated acute changes of such functional variables as arterial stiffness immediately after smoking. However, the chronic effects of smoking are uncertain. Wollersheim et al. reported increased arterial stiffness of the popliteal artery and a tendency toward stiffening of the femoral and common carotid arteries in healthy habitual smokers. However, Kool et al. found no changes in the cross-sectional compliance and distensibility coefficients of the common carotid artery in healthy chronic smokers. Furthermore, the acute and chronic effects of smoking in hypertensive patients have not been evaluated.
Arterial stiffness is known to be an important cardiovascular risk factor and an independent predictor of all-cause and cardiovascular death. Arterial stiffness can be easily assessed by measuring the pulse wave velocity (PWV) with the recently developed automatic devices. The aim of this present study was to evaluate whether there are differences in the acute and chronic effects of smoking on the arterial hemodynamics and properties between hypertensive and normotensive smokers.

### Subject Methods

#### Subjects

We studied 30 apparently healthy male smokers (mean age: 39 ± 1 years) and 22 hypertensive male smokers (mean age: 42 ± 2 years). To compare the chronic effects of smoking in normotensive and hypertensive smokers with nonsmokers, 32 healthy male nonsmokers (mean age: 37 ± 1 years) and 21 hypertensive male nonsmokers (mean age: 48 ± 2 years) were studied as control subjects. Those subjects with diabetes, cardiovascular disease, cerebrovascular disease or those subjects who were taking any cardiovascular medications, including lipid lowering agents, were excluded from the study. To avoid the effects of antihypertensive drugs on arterial stiffness and carotid intima-media thickness (IMT), the patients suffering with hypertension and who have never been treated for this illness were included in the study. For smokers to be included in this study, they had to have smoked more than 10 cigarettes per day for longer than 6 years. All the subjects gave us their informed consent to participate in this study. The definition of hypertension was a systolic blood pressure (BP) of $\geq 140$ mmHg and/or a diastolic BP of $\geq 90$ mmHg. The level of the BP was confirmed by at least three repeated BP measurements over a period varying from days to weeks.

#### Study Protocol

All subjects were asked to refrain from caffeine, alcohol or smoking during the 12 hours prior to testing. The study was carried out between 8:00 AM and 9:00 AM in a quiet room. The subjects were first allowed to rest in a supine position for 15 minutes. The baseline brachial BP, ankle systolic BP, heart rate (HR), carotid IMT and PWV were measured after this rest period. To evaluate the acute effects of smoking, the hypertensive and normotensive smokers were asked to smoke 1 cigarette (nicotine content, 0.9 mg) within 5 minutes and to inhale the smoke. Their BP, HR and PWV were then measured at 5, 10 and 15 minutes after baseline. To evaluate the chronic effects of smoking, the baseline brachial BP, ankle systolic BP, HR, carotid IMT and PWV of the nonsmokers were measured under the same conditions as the smokers, and the data was then compared. Both the right and left brachial BP and the right and left ankle systolic BP were measured simultaneously, and mean values of both the brachial BPs and the ankle systolic BPs were used for analysis.

### Measurements of PWV

The PWV measurements were performed using an automated device (VP-2000, Colin Co. Ltd., Japan). This device records the PWV, brachial and ankle BP, ECG and the heart sounds simultaneously. One trained observer performed all the measurements. In brief, the ECG electrodes were placed on both wrists, and a heart sound microphone was placed on the left sternal border. Occlusion cuffs that were connected to both the plethysmographic and oscillometric sensors were wrapped around both upper arms and the ankles. Both the brachial and ankle BPs were measured simultaneously, and ankle-brachial pressure index (ABI) was calculated. The brachial and tibial arterial pulse waves were acquired using oscillometric sensors. The carotid and femoral arterial waves were acquired using multielement tonometric sensors that were placed at the left carotid artery and the left femoral artery. The heart-carotid pulse transit time was measured from the second heart sound to the notch of the carotid pulse waves. The heart-brachial pulse transit time was measured from the second heart sound to the notch of the brachial pulse waves. The heart-femoral (from the aortic orifice to the femoral artery) pulse transit time was calculated by the summation of the carotid-femoral pulse transit time and the heart-carotid pulse transit time. The femoral-ankle pulse transit time was measured by the foot-to-foot method, from the foot of the femoral arterial pulse waves to the tibial arterial pulse waves. The path lengths of the heart-carotid segment, the heart-brachial segment, the heart-femoral segment and the femoral-ankle segment were calculated automatically from the patient’s height. The heart-carotid (hcPWV), heart-femoral (hfPWV), femoral-ankle (faPWV) and heart-brachial (hbPWV) PWVs were calculated for each arterial segment by dividing the path lengths by the corresponding pulse transit times and this was expressed as m/sec. Both the right and left faPWVs were measured simultaneously, and mean of both faPWVs was used for analysis. Reproducibility of the PWV measurements was evaluated by repeating the measurements in 20 healthy subjects on two different occasions according to the Bland-Altman method. The intraobserver repeatability coefficient values were 850 cm/sec, 815 cm/sec, 870 cm/sec and 479 cm/sec for the hcPWV, hfPWV, faPWV and hbPWV, respectively. The intraobserver variation coefficients were 2.9%, 3.1%, 2.4% and 2.1% for the hcPWV, hfPWV, faPWV and hbPWV, respectively.
Measurements of carotid IMT
Ultrasound measurement of the IMT of the common carotid artery (CCA) was performed with 8 MHz linear array transducer (Sequoia 512, Acuson, USA) as previously described. All the measurements were performed by one trained sonographer with the subject in the supine position and the subject’s head was slightly turned contralateral to the side being examined. The right and left CCA were scanned first in a transverse plane and then these vessels were scanned longitudinally. Two images of the right CCA and 2 images of the left CCA were scanned and stored as digital images. Measurement of the IMT was conducted by using a computer system with automatic IMT measurement software (M’ATH®-STD®, Metris, France). The IMT was measured 2 cm proximal to the carotid bifurcation along at least 1 cm of axial length, and the IMT was measured as the distance between the lumen-intima interface and the media-adventitia interface with using an automated edge detection algorithm. Measurements with a quality index $\geq 0.5$ were used in the analysis. For analysis, the mean of the right and left CCA IMTs was used. Reproducibility of the IMT measurement was evaluated by repeating the measurements in 15 healthy subjects on two different occasions. The intraobserver repeatability coefficient value was 0.015 mm. The intraobserver variation coefficient was 0.6%.

Statistical analysis
Statistical analysis was performed using Microsoft Excel 2002 and SPSS version 12.0KO. The data are reported as means $\pm$ SEM. A $p<0.05$ was considered sig-

| Demographic data of the study subjects | | | | |
| --- | --- | --- | --- |
| | Normotensives | | Hypertensives | | |
| | Smokers | Nonsmokers | Smokers | Nonsmokers | |
| Number | 30 | 32 | 22 | 21 | |
| Age (years) | $39 \pm 1$ | $37 \pm 1$ | $42 \pm 2$ | $48 \pm 2$ | |
| Height (cm) | 171.8 $\pm$ 1.0 | 169.9 $\pm$ 1.1 | 170.5 $\pm$ 1.3 | 169.6 $\pm$ 1.1 | |
| Weight (kg) | 71.6 $\pm$ 1.7 | 69.1 $\pm$ 1.7 | 74.6 $\pm$ 2.8 | 72.9 $\pm$ 1.7 | |
| Body mass index (kg/m$^2$) | 24.2 $\pm$ 0.4 | 23.9 $\pm$ 0.4 | 25.6 $\pm$ 0.7 | 25.3 $\pm$ 0.6 | |
| Total cholesterol (mg/dL) | 191.0 $\pm$ 5.4 | 196.2 $\pm$ 6.1 | 213.0 $\pm$ 7.3$^*$ | 210.7 $\pm$ 6.6 | |
| Fasting blood glucose (mg/dL) | 89.0 $\pm$ 2.1 | 88.4 $\pm$ 1.8 | 89.5 $\pm$ 2.5 | 94.7 $\pm$ 2.5 | |

Data are means $\pm$ SEM; $p$=not significant between the normotensive smokers and nonsmokers, and between the hypertensive smokers and nonsmokers. $^*$: $p=0.021$ vs. normotensive smokers

Fig. 1. Acute changes of HR (A), brachial systolic BP (B), diastolic BP (C), and ankle systolic BP (D) at 5, 10, and 15 minutes after smoking 1 cigarette for normotensive and hypertensive smokers. The HR and brachial BP in both the hypertensive and normotensive smokers, and the ankle systolic BP in the hypertensive smokers increased acutely at 5 minutes after smoking. Data are presented as means $\pm$ SEM. NS: not significant, HR: heart rate, BP: blood pressure.
significant. The acute changes of the hemodynamic parameters over time in the acute part of our study were analyzed by repeated-measures of ANOVA. The differences between the smokers and nonsmokers were analyzed by performing independent t-tests.

**Results**

**Acute effects of smoking in the normotensive and hypertensive smokers**

There was no difference in the baseline characteristics between the normotensive smokers and the hypertensive smokers except for the total cholesterol level (Table 1). Smoking caused a significantly acute increase of the HR and the brachial BP in both the hypertensive and normotensive smokers, and smoking caused a significantly acute increase in the ankle systolic BP in the hypertensive smokers (Fig. 1). In the normotensive smokers, the HR increased acutely at 5 minutes after smoking (from $60.6 \pm 1.6$ to $73.4 \pm 2.0$ bpm, $p < 0.001$). Similar to the normotensive smokers, the HR increased at 5 minutes after smoking in the hypertensive smokers (from $66.7 \pm 1.7$ to $79.6 \pm 2.2$ bpm, $p < 0.001$). The magnitude of the HR increase in the normotensive smokers was similar to that noted in the hypertensive smokers (21% vs. 20%, $p > 0.05$). The HR did not return to baseline until 15 minutes after smoking in both the normotensive smokers ($60.6 \pm 1.6$ vs. $67.5 \pm 1.8$ bpm, $p < 0.001$) and the hypertensive smokers ($66.7 \pm 1.7$ vs. $72.3 \pm 2.0$ bpm, $p < 0.005$). In the normotensive smokers, smoking caused significant acute increase of the brachial systolic BP (from $121.2 \pm 1.8$ to $126.2 \pm 1.6$ mmHg, $p < 0.001$) and the brachial diastolic BP (from $74.5 \pm 1.3$ to $80.8 \pm 1.2$ mmHg, $p < 0.001$) at 5 minutes after smoking. The brachial systolic BP in the hypertensive smokers also increased (from $153.0 \pm 3.6$ to $159.1 \pm 3.3$ mmHg, $p = 0.001$) as did the brachial diastolic BP (from $97.4 \pm 2.1$ to $104.5 \pm 2.3$ mmHg, $p < 0.001$) at 5 minutes after smoking. In the normotensive subjects, the brachial systolic BP returned to the baseline at 15 minutes after smoking ($121.2 \pm 1.8$ vs. $122.6 \pm 1.8$ mmHg, $p > 0.05$), but the brachial diastolic BP did not return to the baseline ($74.5 \pm 1.3$ vs. $78.1 \pm 1.1$ mmHg, $p < 0.001$). However, in the hypertensive smokers, the brachial systolic and diastolic BP did not return to the baseline at 15 minutes after smoking (systolic BP $153.0 \pm 3.6$ vs. $156.6 \pm 3.7$ mmHg, $p < 0.05$; diastolic BP $97.4 \pm 2.1$ vs. $100.8 \pm 2.3$ mmHg, $p = 0.001$). The magnitude of the increases of the ankle systolic BP and the diastolic BP in the normotensive smokers were not different from the hypertensive smokers (systolic BP $4.2$ vs. $4.3\%$, $p > 0.05$; diastolic BP $7.5$ vs. $8.8\%$, $p > 0.05$, respectively). The ankle systolic BP increased significantly in the hypertensive smokers at 5 minutes after smoking (from $178.2 \pm 18.2$ to $184.7 \pm 20.2$ mmHg, $p < 0.05$), but this did not happen in the normotensive smokers.

**Fig. 2.** Acute changes of hcPWV (A), hhPWV (B), hfPWV (C), and faPWV (D) at 5, 10, and 15 minutes after smoking 1 cigarette for the normotensive and hypertensive smokers. Smoking induced a significant acute increase of the hfPWV at 5 minutes after smoking in both the hypertensive and normotensive smokers. In the hypertensive smokers, the faPWV increased significantly at 5 minutes after smoking, but this was not noted in the normotensive smokers. Data are presented as means±SEM. NS: not significant, hcPWV: heart-carotid pulse wave velocity, hfPWV: heart-femoral pulse wave velocity.
The baseline regional arterial stiffness of the hypertensive smokers, which was measured by PWV, was higher than for the normotensive smokers (hcPWV 906.0 ± 51.1 vs. 692.7 ± 27.4 cm/sec, p = 0.001; hfPWV 648.7 ± 17.8 vs. 570.6 ± 10.1, p < 0.001; faPWV 974.6 ± 30.0 vs. 766.9 ± 14.2, p < 0.001: faPWV 1200.2 ± 29.1 vs. 1077.9 ± 35.4, p = 0.015, hypertensive vs. normotensive respectively).

The acute effect of smoking on the regional arterial stiffness was different between the normotensive and hypertensive subjects (Fig. 2). Smoking induced a significant acute increase of the hfPWV at 5 minutes after smoking in both the hypertensive (from 974.6 ± 30.0 to 1066 ± 33.9 cm/sec, p < 0.001) and normotensive smokers (from 766.9 ± 14.2 to 818.3 ± 17.0 cm/sec, p = 0.001). In the hypertensive smokers, the faPWV increased significantly from the baseline of 1200.2 ± 29.1 to 1320.1 ± 31.2 cm/sec at 5 minutes after smoking (p < 0.05). However, in the normotensive smokers, the faPWV did not change significantly (from 1077.9 ± 35.4 to 1041.8 ± 15.7 cm/sec, p > 0.05). Smoking did not induce significant changes of the hcPWV and hfPWV in both the hypertensive and normotensive smokers. The magnitude of the increase in the hfPWV at 5 minutes after smoking was not different between the normotensive and hypertensive smokers (7.0 vs. 10.0%, p < 0.05, for normotensive and hypertensive, respectively). In the hypertensive smokers, the effect of smoking on the hfPWV persisted until 15 minutes after smoking (974.6 ± 30.0 vs. 1019.7 ± 34.5 cm/sec, p < 0.05, for the baseline and 15 minutes, respectively). However, the hfPWV returned to the baseline at 15 minutes after smoking (766.9 ± 14.2 vs. 786.0 ± 15.1 cm/sec, p > 0.05, for the baseline and 15 minutes, respectively) in the normotensive smokers.

Chronic effects of smoking in the normotensive and hypertensive smokers

There was no difference in the baseline characteristics between the normotensive smokers and nonsmokers, and between the hypertensive smokers and nonsmokers (Table 1). In the normotensive and hypertensive subjects, there were no significant differences for the HR, the systolic and diastolic BP and the ABI between the smokers and nonsmokers. The regional PWVs and carotid IMTs were also not different (Table 2).

Discussion

The acute effects of smoking

The present study shows that the acute detrimental effects of cigarette smoking on the regional arterial stiffness are different between normotensive healthy smokers and hypertensive smokers. Smoking caused acute increases of the BP, HR and aortic stiffness in both the normotensive and hypertensive smokers. The femoral arterial stiffness and the ankle systolic BP increased acutely after smoking in the hypertensive smokers, but not in the normotensive smokers. Although the increased HR, systolic BP and PWV returned to their baseline values at 15 minutes after smoking in the normotensive smokers, these measurements did not return to the baseline values in the hypertensive smokers. This result indicates that cigarette smoking may have more acute detrimental effects on the aortic and femoral arterial properties in the hypertensive smokers than in the normotensive smokers.

Several previous studies have demonstrated acute decreases in the brachial and radial arterial distensibility in healthy smokers, but most of these studies didn’t evaluate the acute changes of femoral arterial stiffness after smoking. In those studies, the acute effect of smoking on the carotid arterial distensibility was not significant between the normotensive smokers and nonsmokers, and between the hypertensive smokers and nonsmokers.

Table 2. Chronic effects of smoking on the arterial hemodynamic variables and arterial stiffness

<table>
<thead>
<tr>
<th></th>
<th>Normotensives</th>
<th>Nonsmokers</th>
<th>Hypertensives</th>
<th>Nonsmokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>60.6 ± 1.6</td>
<td>64.1 ± 1.8</td>
<td>66.7 ± 1.7</td>
<td>73.1 ± 1.9</td>
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<tr>
<td>Brachial SBP (mmHg)</td>
<td>121.2 ± 1.8</td>
<td>120.1 ± 1.6</td>
<td>153.0 ± 3.6</td>
<td>149.8 ± 2.5</td>
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<tr>
<td>Brachial DBP (mmHg)</td>
<td>74.5 ± 1.3</td>
<td>73.6 ± 1.2</td>
<td>97.4 ± 2.1</td>
<td>95.5 ± 2.0</td>
</tr>
<tr>
<td>Ankle SBP (mmHg)</td>
<td>137.0 ± 2.4</td>
<td>135.1 ± 2.1</td>
<td>178.2 ± 3.9</td>
<td>175.6 ± 3.6</td>
</tr>
<tr>
<td>ABI</td>
<td>1.12 ± 0.01</td>
<td>1.12 ± 0.01</td>
<td>1.16 ± 0.01</td>
<td>1.16 ± 0.01</td>
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<tr>
<td>hcPWV (cm/sec)</td>
<td>692.7 ± 27.4</td>
<td>661.3 ± 16.6</td>
<td>906.0 ± 51.1</td>
<td>951.4 ± 66.4</td>
</tr>
<tr>
<td>hbPWV (cm/sec)</td>
<td>570.6 ± 10.1</td>
<td>554.5 ± 7.9</td>
<td>648.7 ± 17.8</td>
<td>657.6 ± 27.9</td>
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<tr>
<td>hfPWV (cm/sec)</td>
<td>766.9 ± 14.2</td>
<td>765.8 ± 10.8</td>
<td>974.6 ± 30.1</td>
<td>992.3 ± 57.8</td>
</tr>
<tr>
<td>faPWV (cm/sec)</td>
<td>1077.9 ± 35.4</td>
<td>1007.9 ± 16.0</td>
<td>1200.2 ± 29.1</td>
<td>1221.1 ± 39.2</td>
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<tr>
<td>Carotid IMT (mm)</td>
<td>0.54 ± 0.02</td>
<td>0.52 ± 0.11</td>
<td>0.60 ± 0.03</td>
<td>0.60 ± 0.02</td>
</tr>
</tbody>
</table>

Data are means ± SEM. HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, ABI: ankle brachial index, hcPWV: heart-carotid pulse wave velocity, hbPWV: heart-brachial pulse wave velocity, hfPWV: heart-femoral pulse wave velocity, faPWV: femoral-ankle pulse wave velocity, IMT: intima-media thickness, p = not significant between the normotensive smokers and nonsmokers, and between the hypertensive smokers and nonsmokers.
In the present study, the stiffness of both the carotid and brachial arteries was not changed in both the groups. The different results from the various studies for the acute effects of smoking on the carotid and brachial artery may be due to the different measurement methods. Arterial distensibility is measured by calculating with systolic and diastolic diameter of the artery, and the BP. Thus, the arterial distensibility can reflect only the focal arterial properties, but it does not reflect the properties of a regional segment (i.e. the heart to the brachial segment or the heart to the carotid segment). The PWV reflects the arterial stiffness of the regional segments. The other possibility is that results were skewed by the relatively small number of subjects that we studied. The present study was performed on a small number of subjects, and they showed a trend for the acute increase of the carotid and brachial arterial stiffness after smoking, although this was not significant. Thus, to confirm the acute effect of smoking on the carotid and brachial arterial stiffness, further evaluation with a large number of subjects is needed.

Hypertension and smoking are powerful independent risk factors for cardiovascular morbidity and mortality, and concomitant smoking in hypertensive patients may increase the risk of coronary heart disease. The mechanisms that are involved in this process are not fully understood. The present study suggests that the acute increase in arterial stiffness may be a mechanism that is responsible for smoking-related vascular damage. In addition to several other responsible mechanisms, alteration of the dynamic arterial wall properties is considered to play an important role for smoking related vascular damage. Increased arterial stiffness has been recognized as an important marker for the increased risk of cardiovascular disease and it is an independent predictor of all-cause and cardiovascular death. The acute increase in arterial stiffness, blood pressure and heart rate after smoking may enhance the load on the arterial walls and the left ventricular wall, and this can induce alteration in the distribution of the circumferential stress and tensile stress. An acute increase of the load at the plaques caused by smoking may induce plaque rupture and this can lead to acute ischemic events. Although we did not measure the ankle diastolic BP and the pulse pressure, smoking induced an acute increase in brachial BP in both the hypertensive and normotensive smokers, and it also induced an acute increase of the ankle systolic BP in the hypertensive smokers after smoking. In hypertensive smokers, the acute increase of the femoral arterial stiffness and the ankle systolic BP after smoking may play an important role in the development and progression of lower extremity arterial disease, along with the syner-
gistic effect of hypertension.

With regard to the mechanism for the acute increase in arterial stiffness after smoking, arterial stiffening may be partly due to the increase in blood pressure. However, in the present study, the acute increase in blood pressure cannot fully explain the arterial stiffening because the brachial and carotid arterial PWVs after smoking were not changed in spite of the increase in blood pressure. The other proposed mechanism for the acute effect of smoking is nicotine mediated endothelial dysfunction.

Age, hypertension, diabetes and renal disease are known to increase arterial stiffness. Among the baseline characteristics, BP and the total cholesterol level were different between the normotensive smokers and the hypertensive smokers. The higher baseline BP in the hypertensive smokers may have influenced the acute effect of smoking on the arterial stiffness. However, as indicated above, the possibility of this is low. The effect of hypercholesterolemia on arterial stiffness is controversial, and the present study was designed to evaluate the acute changes of arterial stiffness after smoking. Thus, the result of the present study may not have been affected by the difference in the level of total cholesterol.

The chronic effects of smoking

In the present study, we could not demonstrate the chronic effects of smoking on the arterial wall dynamics. The chronic effects of smoking on the arterial wall dynamics have been controversial. Smoking is a major risk factor for cardiovascular disease; on the other hand, there is no evidence that smoking is the cause of hypertension. Several previous studies have reported on increased plaque arterial stiffness or abnormalities of the brachial artery pressure waveform in habitual smokers. However, many other studies, including present study, found no difference in the regional arterial compliance between the chronic smokers and nonsmokers. Liang et al. have reported on increased carotid IMT in both normotensive and hypertensive smokers as compared to nonsmokers. The ARIC study also demonstrated the significant effects of smoking on the progression of carotid atherosclerosis. The different results of the present study may have come about because of the study subjects. First, arterial stiffness is not dependent on structural changes alone, but it is significantly influenced by endothelium-dependent contraction. Arterial stiffness can increase without there being structural changes of the arterial wall. In the present study, none of the subjects had evidence of advanced atherosclerosis such as carotid plaque, and the regional arterial stiffness was not dif-
different from the nonsmokers. Also, the studies of Liang et al. and the ARIC study were performed on large study populations.

Conclusion
The present study demonstrated that smoking causes acute increases of aortic stiffness, brachial BP and heart rate in both the hypertensive and normotensive smokers, and smoking caused acute increases in femoral arterial stiffness and ankle systolic BP for the hypertensive smokers after smoking. The acute detrimental effects of smoking on the arterial hemodynamics may be a mechanism responsible for smoking-related vascular damage. In the hypertensive smokers, smoking caused a more acute detrimental effect on the femoral arterial stiffness and ankle BP, and this may contribute to the progression of atherosclerosis. Although the present study cannot demonstrate the chronic effect of smoking on arterial stiffness, further studies with a large population are needed to define the chronic effect of smoking on both the arterial wall properties and the hemodynamics.

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