Pressor effect of coffee and cigarette smoking in hypertensive patients

S. FREESTONE AND L. E. RAMSAY
University Department of Therapeutics, Royal Hallamshire Hospital, Sheffield, U.K.

Summary
1. Patients with mild hypertension who habitually smoked cigarettes and consumed caffeine were examined after abstaining from caffeine and cigarettes overnight. Their mean blood pressure (147/89 mmHg) was substantially lower than values recorded in the clinic (164/102 mmHg) and remained so when they continued to abstain (149/94 mmHg at 2 h).
2. Smoking two cigarettes (3.4 mg of nicotine) elevated blood pressure by 10/8 mmHg but for only 15 min.
3. Drinking coffee (200 mg of caffeine) elevated blood pressure by up to 10/7 mmHg between 1 and 2 h.
4. Combined coffee and cigarette smoking caused a sustained rise in blood pressure from 5 to 120 min to levels similar to those measured in the clinic (162/102 mmHg at 2 h). The interaction of coffee and cigarettes on blood pressure, but not on pulse rate, was significant.
5. Caffeine ingestion with or without smoking may be important in the genesis of mild hypertension.

Key words: β-adrenoceptor antagonist, coffee, diuretic, hypertension, smoking.

Introduction
Caffeine and nicotine are each known to increase blood pressure in acute experiments. Cigarette smoking elevates blood pressure by on average 10/8 mmHg but only for about 15 min [1–5]. Caffeine (250 mg) caused a rise in blood pressure of up to 14/10 mmHg, lasting for 2½ h [6]. Since the use of coffee and cigarettes is correlated [7, 8] and as both stimuli apparently increase blood pressure by adrenergic stimulation [4, 6] we have examined the effect of coffee and smoking in hypertensive subjects. We have also explored the practical importance of changes in blood pressure induced by cigarettes and coffee. It has been suggested that the use of caffeine may increase the prevalence of hypertension [6] but this is at variance with epidemiological studies [7, 9].

Methods
Sixteen hypertensive patients (eight untreated, eight taking a thiazide diuretic) consented to the study, which was approved by the hospital ethics committee. All were habitual cigarette smokers (mean 18/day) and drank tea and/or coffee regularly.

Each patient attended on four mornings at 09.00 hours, having abstained from cigarettes and caffeine-containing drinks from midnight. They were recumbent in the same room for the duration of each study period (150 min). After 30 min rest one of four stimuli was administered over 15 min. A different stimulus was administered each day with the order balanced between patients. The stimuli were:
(a) placebo (500 ml of warm orange juice, no caffeine, no cigarettes);
(b) coffee (500 ml = 200 mg of caffeine);
(c) smoking (two tipped cigarettes = 3.4 mg of nicotine, 500 ml of warm orange juice, no caffeine);
(d) coffee plus smoking (200 mg of caffeine and two cigarettes).

Recumbent pulse and blood pressure were recorded over 2 h with a Dinamap recorder [10]. The study could not be made blind but patients were not led to expect any particular outcome. It was predetermined that only those readings
obtained at 0, 5, 15, 30, 60 and 120 min would be analysed. This was done by a ‘repeated measures’ analysis of variance.

Results
Changes in pulse and blood pressure with each stimulus were similar in untreated and thiazide-treated patients, with no significant difference between the two groups, and the results were combined to examine the effects of individual stimuli. Cigarette smoking increased systolic and diastolic blood pressure at 5 and 15 min (Table 1) but thereafter these were no higher than pressures after placebo. Coffee alone significantly increased systolic pressure at 120 min and diastolic pressure at 15, 60 and 120 min. Coffee and smoking together increased systolic pressure and diastolic pressure highly significantly at all times from 5 to 120 min. The effect of the combined stimulus on blood pressure was larger than that of either coffee or smoking alone; at 60 min both systolic and diastolic pressure were significantly higher than after smoking or coffee alone. The blood pressure after coffee plus smoking averaged 11/8 mmHg higher than placebo values throughout the 2 h period. The mean (±SEM) peak blood pressure after coffee plus smoking in the untreated patients was 171 ± 4.4/111 ± 2.4 mmHg, whereas after orange juice it was 158 ± 6.0/97 ± 2.4 mmHg (P < 0.01). Equivalent readings for thiazide-treated patients were 162 ± 7.5/99 ± 4.1 mmHg after coffee plus smoking and 146 ± 8.2/88 ± 4.7 after placebo (P < 0.01).

In the hypertension clinic the untreated patients had a mean blood pressure of 164 ± 6.5/102 ± 2.8 mmHg on the last attendance before the study. At 09.30 hours on ‘placebo day’, having abstained from caffeine and cigarettes overnight, the mean blood pressure was 147 ± 6.8/89 ± 3.2 mmHg, the diastolic pressure being significantly lower than the clinic reading (P < 0.01). Over the next 2 h their blood pressure changed little (147 ± 5.7/90 ± 3.2 mmHg at 1 h, 149 ± 6.2/94 ± 2.6 mmHg at 2 h). In contrast, after coffee plus smoking the blood pressure rose from 143 ± 5.9/88 ± 3.6 mmHg at 09.30 hours to 158 ± 4.2/100 ± 3.2 mmHg at 1 h and 162 ± 5.0/102 ± 2.6 mmHg at 2 h, the last value closely resembling clinic readings. A similar pattern was observed in the thiazide-treated patients. Their clinic blood pressures averaged 147 ± 5.0/90 ± 3.9 mmHg. At 09.30 hours on ‘placebo day’, having abstained from caffeine and nicotine from midnight, their blood pressure was appreciably lower, at 134 ± 8.5/82 ± 4.7 mmHg (P < 0.05 for diastolic). After placebo it remained lower, being 134 ± 7.6/81 ± 4.9 mmHg at 2 h. However, after coffee plus smoking the average blood pressure rose from 133 ± 6.9/83 ± 4.5 mmHg at 09.30 hours to 151 ± 6.3/91 ± 3.9 mmHg at 1 h and 145 ± 6.5/90 ± 3.9 mmHg at 2 h, readings again similar to those obtained in the clinic.

Discussion
We have confirmed a pressor effect of smoking [1–5] and coffee [11] in our patients with mild hypertension. A new finding is that the combination of smoking and coffee had a larger effect on blood pressure than either stimulus alone with the effect sustained throughout the period of study from 5 to 120 min. This effect showed no sign of waning at 2 h, in keeping with the half-life of caffeine of up to 10 h [12].

We have also shown that patients who were

<table>
<thead>
<tr>
<th>Table 1. Influence of placebo, smoking two cigarettes, drinking coffee, or taking cigarettes plus coffee, on recumbent pulse and blood pressure of 16 hypertensive patients.</th>
<th>Time (min) . . .</th>
<th>0</th>
<th>5</th>
<th>15</th>
<th>30</th>
<th>60</th>
<th>120</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse rate (min⁻¹)</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Placebo</td>
<td>69</td>
<td>71</td>
<td>67</td>
<td>65</td>
<td>67</td>
<td>66</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>70</td>
<td>82***</td>
<td>81***</td>
<td>76***</td>
<td>71**</td>
<td>71*</td>
<td></td>
</tr>
<tr>
<td>Coffee</td>
<td>68</td>
<td>73</td>
<td>71</td>
<td>66</td>
<td>65</td>
<td>66</td>
<td></td>
</tr>
<tr>
<td>Coffee + smoking</td>
<td>67</td>
<td>80***</td>
<td>81***</td>
<td>75***</td>
<td>70*</td>
<td>71**</td>
<td></td>
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<tr>
<td>Blood pressure (systolic/diastolic) (mmHg)</td>
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<td></td>
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</tr>
<tr>
<td>Placebo</td>
<td>141/86</td>
<td>142/86</td>
<td>142/84</td>
<td>144/88</td>
<td>143/86</td>
<td>142/88</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>139/84</td>
<td>152**/95***</td>
<td>153***/91***</td>
<td>149/90</td>
<td>145/88</td>
<td>148/91</td>
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<tr>
<td>Coffee</td>
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<td>145/89</td>
<td>145/91*</td>
<td>144/87</td>
<td>147/91*</td>
<td>152***/94**</td>
<td></td>
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<tr>
<td>Coffee + smoking</td>
<td>138/86</td>
<td>152**/95***</td>
<td>153***/94***</td>
<td>153**/91*</td>
<td>155***/96***</td>
<td>154***/94***</td>
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</tbody>
</table>
mildly hypertensive in the clinic had a mean blood pressure within the normal range after abstaining from caffeine and cigarettes overnight. Although this alone does not imply a causal role for these factors the changes observed over the next 2 h do. When these patients remained free of caffeine and nicotine their blood pressure changed little and remained well below clinic readings. When they consumed coffee and smoked two cigarettes there was a prompt and sustained rise in blood pressure to values very similar to those observed in the clinic. The conditions of our study were relevant to the everyday habits of our patients and the doses of caffeine and nicotine used were not exceptional for them.

These results and those of Robertson et al. [6] conflict with epidemiological findings, which have shown no positive relationship between blood pressure and smoking habits [13–16] or the amount of coffee consumed [7, 9].

The possibility that epidemiological studies have failed to detect an important effect of caffeine and nicotine on blood pressure must be considered. The short-lived effect of smoking alone would be difficult to detect unless the blood pressure was measured within minutes of smoking. It is less easy to understand how the prolonged effects of caffeine, with or without smoking, have failed to emerge. One possible explanation is that subjects have been examined fasting [9], when they have low levels of caffeine in the body. Moreover, caffeine intake is difficult to estimate because it is obtained from several sources and there is an inverse correlation between consumption of tea and coffee [8], two major sources of caffeine. Even when a single source is considered estimates of caffeine consumption are likely to be very inaccurate as a cup of coffee prepared at home may contain anything from 14 to 335 mg of caffeine [17]. One study in which subjects who drank tea or coffee were compared with total abstainers found the blood pressure of the caffeine-users to be about 10/5 mmHg higher [18]. Our data suggest that the consumption of caffeine, with or without cigarettes, may be important in the genesis of mild hypertension. The magnitude of the effect for the combined stimulus averaging 11/8 mmHg over at least 2 h is large enough to have a major impact on the prevalence of hypertension.

In the present study there was no evidence that treatment with a thiazide diuretic altered the pressor action of coffee and cigarettes. In further studies the pressor effect has persisted despite treatment with the non-selective β-adrenoceptor antagonists propranolol and oxprenolol. However, the β1-selective blocker atenolol attenuated the pressor effect of coffee and smoking.

Acknowledgments

We are grateful to Stuart Pharmaceuticals Limited for financial support for S.F. We would also like to thank Nigel P. Barker, Statistics Section, I.C.I. Ltd, for the statistical analysis.

References