The Effects of Smoking on the Cardiac Output at Rest and during Exercise in Patients with Healed Myocardial Infarction

By William S. Frankl, M.D., M.S. (Med.), William L. Winters, M.D., and Louis A. Soloff, M.D.

Previous work has shown that smoking increases the cardiac output at rest in normal, healthy young habitual smokers.¹,² This study was undertaken to determine if patients with healed myocardial infarction respond in a like manner, and also to test their responses during exercise.

Methods and Materials

Eight patients, five male and three female, ages 48 to 69, with documented healed myocardial infarction, were studied. Six were habitual smokers and two were nonsmokers. Each patient served as his own control. Some of the patients were studied in the fasting state and some after eating. The order of smoking and nonsmoking studies was randomly selected. Each patient smoked two standard-sized filter tip cigarettes within 10 minutes. All patients inhaled the smoke, although there was slight difficulty on this point in the two nonsmokers. Cardiac output, cardiac index, and stroke volume were measured at rest, in the supine position, by the dye-dilution technique with an indwelling Cournand brachial arterial needle, a Gilford densitometer, indocyanine green dye, and an Electronics for Medicine multichannel direct-writing recorder. Blood for these determinations was withdrawn at a constant rate with a Harvard infusion-withdrawal syringe. Calibrations were performed with the same densitometer setting and blood flow each time. Exercise was performed on a bicycle-ergometer set at 25 kilograms per watt, with the patient cycling for 3 minutes at 60 RPM, and the physiologic measurements were repeated. Each determination was performed in duplicate in six of the eight patients, and the average was calculated and recorded. All determinations agreed within 3 to 12 per cent. In one patient, only one determination was made of each measurement, and in one patient, four determinations were made of each measurement.

Results

Tables 1 and 2 summarize the results.

The cardiac output and cardiac index at rest after smoking fell in two (S.C. and M.H.) and rose in six, compared to the resting, nonsmoking study. The stroke volume fell in six. In four of these six, the cardiac rate rose sufficiently to raise the cardiac output, and in one the rate did not change and the cardiac output fell (M.H.). In one, the cardiac rate rose and the cardiac output fell (S.C.).

The cardiac output and cardiac index during exercise, after smoking, fell in three and rose in four compared to the nonsmoking exercise study. The stroke volume fell in four and rose in three. This fall was due to a drop in cardiac output and a rise in cardiac rate in two (S.C. and A.J.), an inordinate rise in rate in one (F.L.), and a fall in cardiac output and a slight fall in rate in one (E.F.).

The changes in cardiac output and stroke volume after smoking were not significant either at rest (p > 0.05) or during exercise (output p > 0.20, stroke volume p > 0.50). There was, however, a significant change in heart rate after smoking at rest (p < 0.02) and during exercise (p < 0.05). This dissociation between heart rate and stroke volume was previously noted in the healthy young habitual smoker treated earlier with glucose.¹

Discussion

Smoking in our patients with healed myocar-
cardiac infarction failed to produce the striking increases in cardiac output and stroke volume seen in young healthy habitual smokers.\(^1\)\(^2\) After our work was begun, similar differences were reported in abstract form by Pentecost,\(^3\) and in an older study by Regan and his co-workers.\(^4\) These findings are in contrast to variable results obtained with the ballistocardiogram.\(^5\)\(^6\)

Why the patient with healed myocardial infarction fails to develop a significant rise in cardiac output, cardiac index, and stroke volume in contrast to the response of the young normal subject is not clear. Such failure does not appear to be related to a longer established smoking habit, because two of our patients were nonsmokers. Unpublished work in our laboratory tentatively suggests that the catecholamine concentration of hearts from persons in the older age group with heart disease may not significantly differ from the younger person with heart disease. Perhaps the diseased heart is less capable of responding to these hormones, or perhaps less catecholamines are released from extracardiac tissues.

The data herein reported are similar to the initial effect of smoking on young habitual smokers pretreated with glucose. Under such circumstances, the smoking-induced increase in cardiac output and stroke volume are blocked but the initial increase in heart rate is not.

**Summary**

Smoking by subjects with healed myocardial infarction, in contrast to its effects on the

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**Table 1**

Findings before Smoking

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<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>SA</th>
<th>CO</th>
<th>Rest CI</th>
<th>SV</th>
<th>HR</th>
<th>CO</th>
<th>Exercise CI</th>
<th>SV</th>
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* Chronic smokers.
† Nonsmokers.

SA, surface area in M.\(^2\); CO, cardiac output in L./min.; CI, cardiac index in L./min./M.\(^2\); SV, stroke volume in ml.; HR, heart rate in beats/min.

**Table 2**

Findings after Smoking

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<tr>
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* Chronic smokers.
† Nonsmokers.

CO, cardiac output in L./min.; CI, cardiac index in L./min./M.\(^2\); SV, stroke volume in ml.; HR, heart rate in beats/min.; %Δ, Per cent change from nonsmoking state.
normal subject, fails to provoke an increase in cardiac output or in stroke volume. On the other hand, smoking does increase the heart rate in subjects with healed myocardial infarction. This dissociation between the effect on heart rate and on cardiac output and stroke volume, which was also noted in the healthy subject pretreated with glucose, indicates that increase in heart rate is mediated by different factors than those that increase stroke volume and cardiac output.

References
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