The Effects of Smoking on the Signal-Averaged Electrocardiogram in Normal Subjects*

Robert E. Sperry, M.D.; James L. Vacek, M.D., F.C.C.P.; and Gary Scott Smith, M.D.

Tobacco smoking increases the risk of sudden cardiac death, possibly by altering the substrate for propagation or sustained of ventricular tachyarrhythmias. To test this hypothesis, 15 long-term smokers without known coronary artery disease abstained from tobacco smoking for 12 h, after which they underwent SAECG before, 15 min after and 30 min after smoking two cigarettes. Other than minor lengthening of filtered QRS duration, no significant change in time-domain SAECG parameters was noted. We conclude that late potentials are not produced by cigarette smoking and that ventricular arrhythmia substrate as measured by SAECG variables is not worsened in long-term smokers without evidence of coronary artery disease.

(Chest 1991; 99:121-22)

Signal-Averaged Electrocardiography

The SAECGs were obtained from standard orthogonal X, Y and Z leads, using silver-silver chloride electrodes. The QRS signal was amplified over a bandwidth of 0.05 to 300 Hz with a gain of 1,000, 2,000 or 4,000 to maximize amplitude while not saturating the analog-digital converter. Sampling frequency was 2,000 Hz and analog-digital conversion was done with 16-bit resolution. An operator-positioned template window of 25 ms was used to accept QRS complexes with a correlation coefficient of 0.99. The QRS onset and offset were defined by computer algorithm which determined the points at which voltage exceeded the mean of the baseline plus three times the standard deviation. Noise was defined as the RMS voltage in a 25-ms interval in the midpoint of the isoelectric T:P segment. One hundred fifty to 300 beats per study were obtained with the Corazonix "Predictor" to achieve a final noise level of 0.3 μV. Acquired signals were processed by a 40-Hz high-pass bidirectional Butterworth filter, and a vector magnitude of the filtered QRS complex was obtained using the following formula: QRS vector magnitude = √X² + Y² + Z². Measured SAECG variables included the filtered QRS in milliseconds, the RMS voltage (in microvolts) of the last 40 ms of the filtered QRS, and the duration of LAS <60 μV. Previously published criteria for normalcy which have been verified in our laboratory were utilized (QRS ≤ 114 ms; RMS > 20 μV; LAS ≤ 38 ms).

Statistics

Descriptive statistics are expressed as mean ± 1 SD. Comparison of pre- and post-smoking SAECG parameters was made by the paired Student t test.

RESULTS

One patient's baseline 12-lead ECG demonstrated a short P-R interval consistent with a preexcitation syndrome but a normal QRS complex. This patient had no history of palpitations, syncope or other cardiac disease. The other 14 patients' ECCs were normal with no evidence of any type of intraventricular conduction delay. The patients' SAECG parameters are shown in Table 1. The only significant change in post-smoking parameters when compared to pre-smoking parameters was the filtered QRS when measured at 30 min after smoking was completed. Although

*From the University of Kansas Medical Center, Section of Cardiovascular Diseases, Kansas City, KS, and Mid America Heart Institute, Kansas City, MO.

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Reprint requests: Dr. Vacek, 4320 Wernall, Suite Kansas City, MO 64111

SAECG = signal-averaged electrocardiogram; RMS = root mean square; LAS = low-amplitude signals
Table 1 — Signal-Averaged Electrocardiogram Parameters

<table>
<thead>
<tr>
<th></th>
<th>Pre-smoking</th>
<th>15 min</th>
<th>30 min</th>
<th>Post-smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>QRS (ms)*</td>
<td>95.7 ± 15.0</td>
<td>95.9 ± 14.4</td>
<td>97.0 ± 14.9†</td>
<td></td>
</tr>
<tr>
<td>RMS (µV)†</td>
<td>58.1 ± 42.0</td>
<td>57.8 ± 38.4</td>
<td>45.8 ± 29.1</td>
<td></td>
</tr>
<tr>
<td>LAS (µS)‡</td>
<td>30.8 ± 9.0</td>
<td>30.4 ± 8.1</td>
<td>30.5 ± 8.6</td>
<td></td>
</tr>
</tbody>
</table>

*Filtered QRS duration.
†RMS voltage of last 40 ms of QRS.
‡LAS <40 µS.

$p < 0.01$ vs pre-smoking value, all other values NS when compared with those of pre-smoking period.

statistically significant, this difference was very small. No patient in this study demonstrated evidence of late potentials after smoking. Adequate tobacco exposure was demonstrated by the fact that nicotine levels increased from a pre-smoking value of 6.3 to 18.5 ng/ml 30 min after smoking ($p < 0.001$). The initial value is consistent with previously reported morning pre-smoking values in chronic smokers after an overnight tobacco fast.5,7 No patient demonstrated symptomatic or electrocardiographic evidence of ventricular ectopic activity during the course of the study.

**Discussion**

**Impact of Tobacco Smoking**

Tobacco smoking is well recognized as a risk factor for sudden cardiac death as well as other manifestations of coronary artery disease.1,2,9,10 Tobacco smoking appears to have an independent impact on sudden death occurrence over and above its contribution to the development of coronary artery disease.2 Many physiologic sequelae of tobacco smoking may contribute to sudden death occurrence by enhancement of ventricular arrhythmogenesis. Cigarette smoking enhances platelet activation, increases sympathetic discharge and in experimental animals has been shown to lower ventricular fibrillation threshold and to be arrhythmogenic.7,9,10 In one study ventricular fibrillation threshold was lower in normal animals as well as those who had suffered experimental infarction.10 However, studies in humans have demonstrated neither an increase in the frequency or severity of ventricular arrhythmias after cigarette smoking nor increased inducibility of ventricular arrhythmias at the time of electrophysiologic study.9,10 However, the potential exists that while ventricular ectopy per se is not enhanced by the constituents of tobacco smoke, the substrate for the propagation and continuation of ventricular arrhythmias may be altered by smoking. Our study was designed to see whether the substrate for sustained ventricular arrhythmias is altered by smoking when studied by evaluation of the time domain SAECG variables. Other than a very minor lengthening of the filtered QRS duration at 30 min after smoking, no significant alteration in SAECG variables was noted. Our data suggest that it is unlikely that smoking per se changes the ventricular arrhythmia substrate as measured by time domain SAECG analysis in patients without a known history of coronary artery disease.

**Limitations**

Sudden death due to ventricular arrhythmias in patients with demonstrated coronary artery disease may be associated with increased ventricular ectopic activity which occurs during periods of transient ischemia. It has been demonstrated that smoking may induce ischemia, often silent, in patients with known coronary artery disease.8,13 It is possible that smoking in patients with known coronary artery disease produces ischemia and hence contributes to sudden cardiac death in this fashion, perhaps augmented by the increased sympathetic discharge associated with tobacco smoke inhalation.

**References**

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