these patients with no structural heart disease would lead to multiple replacements of generators over years with their inherent complications (especially infections) and their considerable economic cost. Our results support that in patients with idiopathic VF, one should elect to base treatment on results of electrophysiological drug testing, especially with class IA agents. Of course, this would be possible only for patients with inducible ventricular tachyarrhythmias at control study. Efforts should be made to increase sensitivity of programmed stimulation protocols in these patients. In our opinion, the use of repetition of extrastimulation may be very useful and should be systematically included in these protocols.5,6

Bernard Belhassen, MD
Sami Viskin, MD
Department of Cardiology
Tel-Aviv Medical Center
Tel-Aviv, Israel

References

Sidestream Cigarette Smoke

A recent study by Penn and Snyder in Circulation (‘‘Inhalation of Sidestream Cigarette Smoke Accelerates Development of Arteriosclerotic Plaques.’’ 1993;88:1820-1825) contains technical errors and omissions in the inhalation exposure methodology that prevent a useful analysis of the morphometry data presented.

The authors state that ‘‘moderate levels were chosen so that exposure conditions would be more relevant to those encountered in indoor environments by passive smokers.’’ No reference was given to target concentrations or how these targets would relate to field measurements. Instead, the reader finds that the mean values obtained in four identical chambers (no reason is given as why four separate units were needed) for total suspended particulates (TSP) were between 7.5 and 8.5 mg/m3. A recent review of TSP concentrations in 951 smoking homes and 905 nonsmoking homes indicated an average of 0.049 mg/m3 for the former and 0.022 mg/m3 for the latter, for an overall difference (which may be due to smoking) between the two of 0.027 mg/m3. This is more than 300-fold less than the ‘‘moderate’’ concentrations used by Penn and Snyder. Clearly, the statement made in the ‘‘Discussion’’ (‘‘comparable to the dose that can be expected under heavy smoking conditions at home’’) brings a new insight as to what constitutes ‘‘heavy’’ smoking for these authors, especially as their cockerels were exposed to this massive concentration continuously for 6 hours per day. The calculations made by Penn and Snyder in the ‘‘Discussion’’ contain a number of assumptions and produce results that are starkly at variance with actual field measurements.2 Holcomb3 showed similarly small differences in TSP concentrations between smoking and nonsmoking restaurants, offices, and transportation.

In our laboratory,3 we have shown that there are no histopathological effects in the vascular systems of rats exposed to aged and diluted sidestream smoke at the grossly exaggerated TSP concentration of 10 mg/m3 for 6 hours per day for 13 weeks (370 times the mean difference noted above between smoking and nonsmoking homes).

Cigarette mainstream smoke, sidestream smoke, and environmental tobacco smoke (ETS) are not synonymous.4 The aging process, a major factor in the formation of ETS from sidestream smoke and exhaled mainstream smoke,2 is not present in Penn and Snyder’s design. Exposure to fresh sidestream smoke, as generated by Penn and Snyder, is totally inappropriate. It is then unclear how much of the aerosol presented to the cockerels was actually inhaled or ingested by them; whole-body exposures are known to produce large body burdens by the latter route compared with the former.2

No reasons are given for the extensive modification of the smoke generator2 to remove design features that were incorporated to produce standard puffs and smoke with a constant composition. Together, these unreferenced modifications from our original design2 would produce an extremely variable aerosol over the 6 hours of exposure (no data presented); the nonstandard methods preclude verification of the results by other researchers.

Chris R.E. Coggins, PhD
Research and Development
R.J. Reynolds Tobacco Co
Winston-Salem, North Carolina

References

Reply

It is fanciful (and wrong) for Coggins to state that our study contains errors and omissions that ‘‘will prevent a useful analysis of the morphometry data presented.’’ The reasons for our position follow.

Coggins correctly quotes our choice of exposure conditions and then takes us to task for not predetermining target concentrations of smoke surrogates. Our goal, stated clearly in the last paragraph of the introduction, was ‘‘to address directly the question of whether inhalation of sidestream (SS) smoke accelerates aortic plaque development.’’ Smoking advocates have expended a great deal of energy minimizing the health hazards of SS smoke (eg, see Coggins’ References 3 and 4).2 We have now shown unequivocally that inhalation of SS cigarette smoke alone, without additional experimental manipulations such as enhanced cholesterol intake, markedly accelerates arteriosclerotic plaque development. Indeed, Coggins appears to concede this point in his letter and instead questions the relevance of the concentrations of smoke surrogates. Smoking advocates consider total suspended particulate (TSP) measurements more reliable surrogates for SS cigarette smoke concentrations than carbon monoxide.
Sidestream cigarette smoke.
C R Coggins

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