

## Smoke-free Legislation and Acute Coronary Syndrome

**TO THE EDITOR:** Pell et al. (July 31 issue)<sup>1</sup> report that smoke-free legislation in Scotland was strongly associated with a decreased risk of the acute coronary syndrome. Nested within their study is a critical and remarkable observation: smokers are also at substantial risk from exposure to secondhand smoke and benefit from its cessation. My group and others have also shown that smokers have adverse respiratory health effects from exposure to secondhand smoke.<sup>2,3</sup> Bartenders, whether smokers or nonsmokers, have rapid improvement in their respiratory health after smoke-free-workplace legislation is instituted.<sup>4,5</sup> This point is important, because most previous research on the health effects of secondhand smoke has excluded smokers on the basis of the assumption that passive smoking is inconsequential as compared with active smoking. We are now learning that secondhand smoke, like other air pollution, affects both smokers and nonsmokers. Research and public health efforts that address

exposure to secondhand smoke need a broader focus, to include all persons, regardless of personal smoking status. It is time to clear the air, for smokers and nonsmokers alike.

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## FDA Regulation of Tobacco

**TO THE EDITOR:** In 1998, Mark Berling, chief legislative counsel of Philip Morris, drafted specifications for regulation of tobacco products by the Food and Drug Administration (FDA) that would ensure the continuing profitability of the Marlboro brand, provide a shield against litigation, and protect cigarettes from competition from less-toxic, smokeless tobacco products.<sup>1</sup> The current Family Smoking Prevention and Tobacco Control Act (H.R. 1108/S. 625) discussed by Brandt in his Perspective article (July 31 issue)<sup>2</sup> was negotiated between Matthew Myers of the Campaign for Tobacco-Free Kids and Mr. Berling<sup>3</sup> for purposes of securing an FDA bill with full support from our nation's largest cigarette maker. The text conforms to Mr. Berling's 1998 specifications.

Despite the optimistic wording of the summaries used to attract endorsement and sponsors, we believe that this bill is so distorted in favor of Altria-Philip Morris that, if passed in its current form, it will do more harm than good in terms of future levels of teen smoking and future

rates of tobacco-related illness and death. It can protect cigarettes or it can protect the public's health. It cannot do both.

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**THE AUTHOR REPLIES:** Nitzkin is incorrect in suggesting that the current FDA bill is based on the Berling memorandum. Philip Morris introduced such a bill in 2001, and it was broadly rejected by tobacco-control advocacy groups, including the Campaign for Tobacco-Free Kids. Furthermore,

Nitzkin is wrong to claim that the currently pending bill was negotiated between Philip Morris and Matthew Myers of the Campaign for Tobacco-Free Kids. The current bill was the work of Senator Ted Kennedy (D-MA) and then-Senator Mike DeWine (R-OH), who have consistently been among Congress's strongest supporters of tobacco control.

The pending legislation differs markedly from the proposals previously put forward by Philip Morris. The bill has provisions that would require serious changes in the marketing and manufacture of cigarettes in the decades ahead, including the authority for the FDA to monitor and reduce levels of dangerous additives and nicotine. The

improved warning labels — just one provision of the bill — could save thousands of lives.

Nitzkin is certainly entitled to oppose the legislation, but he fails to offer any evidence for his claim that the bill will lead to increases in teen smoking and tobacco-related mortality. His position conflicts with the positions of virtually all the leading public health and medical organizations that are committed to reducing the burden of disease that cigarettes generate.

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## A New ECG Sign of Proximal LAD Occlusion

**TO THE EDITOR:** Recognition of characteristic changes in an electrocardiogram (ECG) that are associated with acute occlusion of a coronary artery guides decisions regarding immediate reperfusion therapy.<sup>1-3</sup> Working from our primary database of percutaneous coronary interventions, which includes records of the ambulance, or admission, ECG (performed on first medical contact with the patient), the preprocedural ECG, and the coronary angiogram, we describe a new ECG pattern without ST-segment elevation that signifies occlusion of the proximal left anterior descending coronary artery (LAD). Instead of the signature ST-segment elevation, the ST segment showed a 1- to 3-mm upsloping ST-segment depression at the J point in leads  $V_1$  to  $V_6$  that continued into tall, positive symmetrical T waves. The QRS complexes were usually not widened or were only slightly widened, and in some there was a loss of precordial R-wave progression. In most patients there was a 1- to 2-mm ST-elevation in lead aVR (see Fig. 1 for representative examples of this ECG pattern). We recognized this characteristic ECG pattern in 30 of 1532 patients with anterior myocardial infarction (2.0%).

Although tall symmetrical T waves have been recognized as a transient early feature that changes into overt ST elevation in the precordial leads, in these patients this pattern was static, persisting from the time of first ECG until the preprocedural ECG was performed and angiographic evi-

dence of an occluded LAD was obtained (i.e., 30 to 50 minutes). The ECGs with this pattern were on average recorded 1.5 hours after symptom onset. Collateral filling of the LAD ranged from Rentrop class 0 to class 3, and a wraparound LAD was present in 50% of patients. There was no evidence of involvement of the left main stem of the coronary artery, nor was there evidence of significant disease in the coronary arteries supplying the posterior or posterolateral myocardial territories. Potassium levels on admission were normal ( $3.9 \pm 0.5$  mmol per liter). Despite successful procedures in all cases, there was considerable loss of myocardium, with a median creatine kinase MB peak of  $342 \mu\text{g}$  per liter.

The electrophysiological explanation of the observed ECG pattern remains elusive. We could not establish patient characteristics, nor could we identify coronary angiographic characteristics that were unequivocally associated with the ECG pattern described as compared with a pattern of anterior ST elevation. Theoretically, an anatomical variant of the Purkinje fibers, with endocardial conduction delay, could be present. Alternatively, the absence of ST elevation may be related to the lack of activation of sarcolemmal ATP-sensitive potassium ( $K_{\text{ATP}}$ ) channels by ischemic ATP depletion, as has been shown in  $K_{\text{ATP}}$  knock-out animal models of acute ischemia.<sup>4</sup> It is of great importance for physicians and paramedics involved in the triage of patients with chest pain