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Editorials

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Electronic Cigarettes

No Such Thing as a Free Lunch…or Puff

As practitioners of pulmonary and cardiac medicine, many of us have no doubt been asked by our patients who smoke about so-called "electronic cigarettes" (e-cigarettes). These devices, termed electronic nicotine delivery systems (ENDS) by the World Health Organization, have been available in the US market since 2007. Our patients have likely heard far more about these devices through marketing, chat rooms, and word of mouth, than we as physicians have through the medical literature. Because ENDS are not currently regulated by the US Food and Drug Administration (FDA) as medical devices—recent court decisions, denied the agency the right to such oversight—manufacturers of ENDS have not been required to establish either safety or efficacy, and we have had few data with which to answer our patients' queries about these products. Are e-cigarettes a smoking cessation tool? Are they a harmless alternative to cigarettes, as manufacturers claim?

In this issue of *CHEST* (see page 1400), Vardavas et al[1] evaluated the immediate effect of e-cigarette vapors on airway mechanics. Subjects, healthy smokers without chronic lung disease, inhaled the vapors of a commercially available e-cigarette for 5 min. A comprehensive analysis of lung function, including oscillometry and spirometry, revealed that after inhaling e-cigarette vapors, subjects had a significant increase in airway resistance. There were no significant effects on FEV_1, FVC, or FEV_1/FVC ratio. Subjects also had measurable, significant decreases in fraction of exhaled nitric oxide (FENO). Control subjects inhaling vaporless control cigarettes did not have any changes in airway resistance or FENO. This pattern of changes in airway mechanics and FENO experienced by subjects using e-cigarettes is very similar to that seen shortly after inhalation of tobacco smoke.[2] The implication is that with long-term exposure to ENDS, it is plausible that, as with cigarette smoking, there is the potential for more permanent changes in lung function. The study's authors correctly point out that this is conjectural and that further research on the long-term effects of ENDS is needed.

Numerous manufacturers market ENDS, but the devices share key design features. They use a battery-operated heating device that vaporizes a nicotine-containing solution from a replaceable cartridge in a process triggered by the pressure drop that occurs when the user inhales from the device. They resemble cigarettes, and, in addition to providing nicotine in inhaled form, replicate some of the behavioral aspects of cigarette smoking. Cartridges come with various concentrations of nicotine, and refill solutions containing large amounts of nicotine are available.[3] The most common vehicle in which the nicotine in such cartridges is contained is propylene glycol, though other chemicals may be used. The efficiency of systemic nicotine delivery with the e-cigarette is highly variable,[4] and the pharmacokinetics of nicotine delivery appear to more closely resemble those of nicotine replacement therapies than that of cigarettes.[5] Marketing materials for ENDS commonly emphasize the safety of the devices in comparison with cigarettes, claiming that the aerosols or vapors delivered by the devices are "tar free" and lack the carcinogens and other chemicals found in cigarette smoke. Because of concerns about running afoul of the FDA, ENDS are not specifically marketed as smoking cessation aids.

Users of ENDS tend to be former or current smokers who choose ENDS for a variety of reasons, including the markedly reduced cost compared with cigarettes, the ability to use the devices in settings in which cigarette smoking is prohibited, the curbing of nicotine cravings, and the perceived lower toxicity compared with traditional tobacco cigarettes.[6] No randomized controlled clinical trials have evaluated ENDS as smoking cessation tools. ENDS appear to be most commonly used by current smokers as an alternative to cigarettes rather than as cessation tools, although some ENDS users do reduce or eliminate cigarette use.[7] Given the relatively short time that ENDS have been available, long-term patterns of use are not clear, and it is plausible that, as with cigarettes and other forms of nicotine, some users will use the devices for prolonged periods. We as physicians, and they as our patients, need and deserve

the information necessary to make informed choices and recommendations about the safety of these devices as alternatives to cigarettes and other available therapies for smoking cessation. Although the FDA has pledged to regulate these devices as tobacco products, it is unclear what this will entail and how it will change the availability, marketing, manufacturing, and quality control of these devices.

The study by Vardavas and colleagues,[1] although modest in its size, scope, and conclusions, provides some much needed data on the potential harm of e-cigarettes. Clearly, more studies are needed on the long-term effects of these devices, especially in patients with chronic airways disease. In the interim, we now have enough information to state that the use of the ENDS does cause at least short-term adverse effects that are similar to those of cigarettes, and to tell our patients that there is no such thing as a free lunch… or, in the case of ENDS, a (harm-) free puff.

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The Pharyngeal Airway

Is Bigger Really Better?

In this issue of *CHEST* (see page 1407), Yamashiro and Kryger[1] report the results from a consecutive series of patients with obstructive sleep apnea (OSA) who underwent CT scans. The authors observed important associations between pharyngeal airway length and various risk factors for OSA (including age and sex) and postulated that "laryngeal descent," perhaps having evolved for speech, may have consequences from the standpoint of pharyngeal mechanics. The article helps to corroborate existing literature and provides an opportunity to consider the mechanical basis underlying pharyngeal airway collapse in humans.

In discussing pharyngeal airway length, an important distinction must be made between the intrinsic length of the airway vs the length that may result from changes in longitudinal tension. In the former, physical principles as well as computational modeling and human experimental data all support the concept that a longer airway is more vulnerable to pharyngeal collapse during inspiration than would be a shorter airway.[2] For a given negative intrapharyngeal pressure, the force per unit area would favor collapse in the longer airway as compared with a shorter one. Moreover, the radius of curvature (in the longitudinal direction) will be larger in a long airway than it will be in a short airway, and based on the law of Laplace, less negative intrapharyngeal pressure would be required to reduce airway volume through longitudinal bending; the longer airway is more susceptible to collapse. On the other hand, if an airway is lengthened through increased longitudinal tension, it may be less susceptible to inspiratory collapse due to stiffening in the airway walls through changes in the tube law.[3]

Similar arguments can be made regarding the diameter in the pharyngeal airway, which could be intrinsically small or could be made smaller by extraluminal forces (eg, parapharyngeal fat deposition).[4] In the case of the intrinsically small-diameter airway, one would predict protection from collapse based on the small area upon which a given intrapharyngeal negative pressure is acting. In other words, based on the law of Laplace, increased negative pressure is required to collapse a tube with small radius of curvature (ie, small diameter airway) than that required for a larger one; the smaller one is less susceptible