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Adverse Anesthesia Events in Children Exposed to Environmental Tobacco Smoke

Exposure to Environmental Tobacco Smoke and the Risk of Adverse Respiratory Events in Children Receiving General Anesthesia

AT a time when the "tobacco settlement" is found in the headlines everyday, and as we try to sort out not only the health effects of smoking on the smoker but also the effect of environmental tobacco smoke (ETS) on the nonsmoker, it is appropriate that Anesthesiology publish a seminal report on the risk of adverse respiratory events in children receiving general anesthesia. 1

It is also appropriate to focus on the health effects of tobacco at a time when the emphasis in the media is on the egregious behavior of the tobacco industry. Smoking, on the decline until only a few years ago, is now more than ever a threat to the public's health because the prevalence of adult smoking has ceased to decrease and about 6,000 American teenagers try their first cigarette each day. Of these, 3,000 will become regular smokers (1,600 girls and 1,400 boys). Even though two thirds of the teenagers will regret taking up the habit and will want to quit, three quarters of them do not succeed because of their addiction to nicotine. Sadly, about one third of those who cannot quit will die prematurely and poorly of a smoker's death.

The damages of ETS have been confirmed by recent studies focusing on urinary cotinine. The adverse respiratory events reported by Skolnick *et al.* were tied to urinary cotinine concentrations, an accurate and unambiguous means of assessing the patient's exposure to ETS. Such cotinine studies are scientifically sound and were used, for example, in volunteer flight attendants and passengers in the studies carried out by the Public

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Health Service to assess exposure to environmental tobacco smoke on air flights. These studies resulted in the federal ban on smoking for domestic flights on American carriers, which rapidly spread to flights overseas and then to foreign airlines as well.

Cotinine concentrations avoid the uncertainties that arise when questioning parents who smoke. Smokers tend to underestimate the number of cigarettes smoked per day, exaggerate the isolation of children in the home from smokers, and do not always tell the truth when they think that smoking makes them culpable for their children's health problems.

Of the 499 children in the study, those with the highest cotinine concentrations (≥40 ng/ml) experienced a 42% incidence of adverse airway complications. The percentage of patients with adverse events was 33% with moderate cotinine concentrations (10-39.9 ng/ml) and 24% with low cotinine levels (<10 ng/ml). The authors found a strong association between the passive inhalation of tobacco smoke and airway complications in children receiving general anesthesia—the relationship is greater for girls and for those whose mothers have a lower level of education. They conclude that environmental smoke should be regarded as a risk factor in children undergoing general anesthesia.

My pediatric, surgical, and public health background suggest another conclusion: children who exhibit adverse respiratory events during general anesthesia—and their mothers (families)—are prime targets for educational programs about the dangers of ETS in child-hood. Such children who are adversely affected by ETS are likely to be those with the more severe health effects of actual smoking. I am especially concerned that the increase in teenage smoking is more prevalent in girls, a gender trend confirmed in the studies reported here.

In as much as the accepted and conservative number of nonsmokers who die each year from ETS exposure is more than 50,000, anything we in the medical profession can do to educate children and their families about the risks of ETS has to be worthwhile.

The adverse respiratory events reported here in children during general anesthesia add significant validity to accumulating evidence on the risks of ETS in children. These risks include alteration of pulmonary function, exacerbation of asthmatic attacks, level of bronchial response in asthma, wheezing in young children, upper and lower respiratory tract illness, and middle ear effusion.

Any educational effort aimed at encouraging smokers to quit and to protect their children from ETS is an uphill task. The trigger event that could be used in this circumstance would be the reporting by anesthesiologists to surgeons and pediatricians of the patients' families of the occurrence of adverse respiratory effects during general anesthesia and the likelihood that they were associated with exposure to ETS. I have long advocated that on the initial assessment of new pediatric patients, history-taking include exposure to environmental tobacco smoke. Parents, especially new first-time parents, want to do the right thing for their children, and this is the best time for a physician's

admonition about ETS exposure to make an impact. There are many simple preventive measures that never occur to parents that can save lives, such as suggesting to a parent who believes a handgun must be kept in the home that the bullets for the weapon should be hidden in another place.

When all of the detrimental effects of ETS on children are considered, especially those in asthmatic children, smoking in households where there are children may well become the next issue in child abuse.

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Reference

1. Skolnick ET, Vomvolakis MA, Buck KA, Mannino SF, Sun LS: Exposure to environmental tobacco smoke and the risk of adverse respiratory events in children receiving general anesthesia. Anesthesiology 1998;88:1144-53

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How Does µ-Opioid Receptor Blockade Work in Addicted Patients?

WHEN I first heard the CNN broadcast and then later read the *New York Times* story about patients who receive general anesthesia to break opioid addiction, my immediate response was, "Well, sure it will work—until the patient wakes up. An addict cannot take drugs while asleep, but the basic biochemistry of his or her brain will not change." But on further consideration, I think it is possible that the mechanism that produces the unconscious state during general anesthesia and prevents an addict from "feeling" may

indeed permanently alter brain biochemistry. A crazy thought? In this issue of Anesthesiology, Kienbaum *et al.*¹ describe the rapid detoxification of addicts during general anesthesia with the use of μ -opioid receptor blockade. The investigators do not speculate about the mechanism behind this therapy. My question is, does the therapy really work? To call the therapy effective, Kienbaum *et al.* must show that in the long term, brain biochemistry in their subjects was changed. Or failing that, proof is needed that the subjects abstained from drugs long term.

The study, which presents one therapy to reverse the biochemistry of addiction, may teach us something about the biochemistry of anesthesia. We know that opioids and general anesthetics suppress the autonomic nervous system. Many studies have suggested that these drugs work synergistically rather than additively. In the 1970s, I spent a year at NIH trying to confirm the changes in brain receptors caused by opi-

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This Editorial View accompanies the following article: Kienbaum P, Thurauf N, Michel MC, Scherbaum N, Gastpar M, Peters J: Profound increase in epinephrine plasma concentration and cardiovascular stimulation following μ -opioid receptor blockade in opioid-addicted patients during barbiturate anesthesia for acute detoxification. ANESTHESIOLOGY 1998; 88:1154-61.