

effects of aerosolized corticosteroids on various airway tissues remain to be determined.

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Mold Control

TWO SOURCES for mold spores growing indoors have been identified. The first and, generally, more important source (exogenous) originates from outdoors but is constantly introduced into homes. The second source (endogenous) originates indoors but varies greatly from house to house. Studies in Michigan, using the volumetric Andersen sampler during frost-free periods, indicated that indoor viable mold spore counts were approximately 25 percent of the outdoor counts. Indoor mold counts varied from 10 to over 20,000 spores per cu meter. Higher indoor counts were associated with high relative humidity within the home, and generally occurred in homes with central humidification.

Similar studies done in Southern California showed a statistically significant correlation between the following outdoor characteristics and high viable mold spore counts indoors: extensive shaded areas near the house, large quantities of organic debris (fallen leaves, grass not removed after cutting, hay, compost or stands of dried weeds and grass on the lot), poor landscape maintenance and previous indoor water problems. Two indoor characteristics associated with low indoor mold isolates were the presence of a central electrostatic filtration system and compliance with dust control. In homes with continuously operated electrostatic filters, the mean viable mold spore count was 155 per cu meter, while in homes with intermittently operated units, the mean mold spore count was 344 per cu meter. In control homes the mean count was 687 spores per cu meter. A less dramatic although still statistically significant reduction in mold spore isolates occurred in homes where dust control measures were being implemented.

The effect of air-conditioning on indoor mold levels was recently studied. A statistically signifi-

cant reduction in mold spore isolates occurred in the air-conditioned homes, but no differences in the percent concentration of the major genera were noted. The investigators had expected a greater percent reduction in large mold spores if these were being trapped in the air-conditioner, but no reduction in smaller spores. Because all genera were reduced equally, the investigators felt that merely closing an air-conditioned home created a barrier to the ingress of outdoor molds resulting in lower indoor mold levels. They also noted reduction of mold isolates in air-conditioned homes with low indoor relative humidity.

It appears prudent to recommend the following program to minimize indoor mold spore concentrations. The amount of shade near a house should be reduced by avoiding plants too close to the building and by periodic pruning of trees and shrubs. Better landscape maintenance with removal of all dead vegetation within 150 to 200 feet of the house would be helpful. Dust control compliance is encouraged. Water damaged items including carpet, books, wallpaper and wicker baskets should be removed from the premises and the cause of any previous water problem corrected. Properly installed and operated central electrostatic filtration should significantly reduce the amount of viable indoor mold spores. Humidification devices including cold mist vaporizers are best avoided, especially in homes of mold-sensitive persons. Logically, use of a dehumidifier in damp areas should be helpful, but no studies have yet been carried out to verify this commonly held belief.

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Perspective on Asthma in Pregnancy

THERE IS NEARLY equal probability that maternal asthma will improve, remain the same or grow worse during pregnancy. Improvement tends to occur in the first trimester and deterioration in the second and third. Cases of severe steroid-dependent asthma are the most likely to cause deterioration in these women's conditions during their pregnancies. Slightly increased percentage rates of premature births, spontaneous abortions and new-

born deaths resulting from maternal asthma have been noted. The greatest danger to the fetus in maternal asthma is maternal hypoxia resulting in fetal hypoxia.

Several drugs used in the treatment of asthmatic patients are contraindicated in pregnancy. Preparations containing iodine may cause congenital goiter. Tetracyclines may stain the teeth. Sedatives in general are to be avoided because of their respiratory depressant effect. Hydroxyzine in large doses has been reported teratogenic in mice. The safety of the beta agonists such as terbutaline and metaproterenol has not been established in pregnancy.

Medications considered safe in the treatment of asthma during pregnancy include theophylline, ephedrine, cromolyn, penicillin and erythromycin. Corticosteroids used in the first trimester are known to cause cleft lip, cleft palate and skeletal deformities in experimental animals. They should be avoided if bronchospasm can be controlled without their use. In pregnant women with asthma who are steroid dependent, inhaled beclomethasone may allow reduction of dosage or even cessation of use of systemic steroids, although the safety of beclomethasone in pregnancy has not been established.

Because allergy skin testing and immunotherapy carry some risk of producing anaphylaxis, they should be used with special caution in pregnant patients, but need not be excluded. Ideally, asthma management should begin well in advance of contemplated pregnancies and control of this disorder should reduce both maternal and fetal morbidity and mortality.

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Reactions to Local Anesthetic Agents

SINCE FREUD AND KOLLER introduced cocaine into clinical use as a local anesthetic in 1884, various effects of local anesthetic agents have been described, including toxic effects of the drugs, systemic sympathomimetic stimulation, and vasovagal, local and allergic reactions. Rarely, preservatives such as paraben or methylparaben also may be incriminated.

Appropriate diagnoses are usually readily apparent by taking detailed histories of the episodes. Toxic effects are related to the total dose administered and include central nervous system stimulation (restlessness, dizziness and disorientation) or central nervous system depression (slurred speech, respiratory failure). Systemic sympathomimetic stimulation results from inclusion of epinephrine in the local anesthetic and includes anxiety, tremor, diaphoresis and tachycardia. Vasovagal symptoms may occur as a result of an injection of a local anesthetic; these may include apprehension, hyperventilation, hysteria and syncope. Localized swelling at the injection site often occurs and may be due to a local toxic effect of the anesthetic or to the procedure itself; however, especially when severe, a local IgE-mediated phenomenon cannot be ruled out. Pruritus, rhinorrhea, urticaria, angioedema and manifestations of bronchospasm, that is, cough and wheezing, are hallmarks of an allergic reaction. On careful scrutiny, however, only a small percentage of those presenting with questionable reactions will fit this category.

The clinical approach presumes a need to use an agent for either a necessary local procedure or for treatment of a cardiac arrhythmia. The risks inherent in the alternatives—whether to use a general anesthetic or not to use a lifesaving drug—clearly warrant provocative procedures and require an understanding of the classification of local anesthetics.

Although many pharmacologic agents have local anesthetic properties, all commonly used agents have a fundamental structure consisting of three parts: an amino group linked by an intermediate group to an aromatic residue, an ester (group I), or an amide or other bond (group II) serving to differentiate the two basic groups. Prominent among those in group I are benzocaine, butacaine (Butyn), chlorprocaine (Nesacaine), procaine (Novocain) and tetracaine (Pontocaine). These compounds will cross-react with each other. Reactions to compounds within this group have been reported much more frequently than to those within group II which represent a more diverse subset that lacks the ester bond. These compounds include dibucaine (Nupercaine), lidocaine (Xylocaine), prilocaine (Citanest), mepivacaine (Carbocaine) and proparacaine (Ophthaine). At present, it is presumed that those within group II do not cross-react with each other.

If a patient clearly has had an allergic or even