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DIFFICULTIES IN DIAGNOSING AND TREATING POLYMYALGIA RHEUMATICA

Polymyalgia rheumatica (PMR) is a common disease in the population over age 50 years; the incidence approaches 0.01%. Because this syndrome is often associated with giant cell arteritis (GCA), which if untreated can cause blindness and death, early recognition and treatment is necessary.

DIAGNOSIS

The most commonly used diagnostic criteria for PMR have been arrived at by convention and consensus. Recently, a British group led by H.A. Bird established the sensitivity and specificity of a large number of signs and symptoms thought helpful in the diagnosis of PMR. Seven of these features were most discriminative: 1) bilateral shoulder pain, 2) onset over a period of two weeks or less, 3) initial Westergren sedimentation rate of 40 mm/h or greater, 4) morning stiffness lasting one hour or longer, 5) age over 65 years, 6) depression or weight loss and, 7) bilateral upper arm tenderness. When three or more of these criteria or one criterion and palpable abnormality of the superficial temporal artery were present, the likelihood that the diagnosis would be PMR was 92% sensitive and 75% specific.

TREATMENT

Many authorities suggest that PMR should be treated initially with daily doses of prednisone ranging from 15 to 60 mg. Many of these same authorities feel that if GCA is present, 40 to 60 mg of prednisone per day should be given and continued for at least four weeks. Another British group headed by A.R. Behn reported a prospective study of 176 patients with PMR and/or GCA. An initial daily dose of prednisolone 10 mg for PMR and 20 mg for GCA adequately controlled disease signs and symptoms in more than 90% of patients. This work has recently been corroborated by others. Studies like this suggest that we must look critically at the use of high-dose corticosteroid in the treatment of either of these syndromes.

These studies also allow the use of lower-dose corticosteroid in selected patients with these diseases, such as patients with insulin-dependent diabetes, in whom high-dose corticosteroid would likely result in a serious adverse effect.

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PASSIVE SMOKING: NUISANCE OR HEALTH RISK?

Without question, sidestream smoke emitted from the tip of a burning cigarette is biologically hazardous. Indeed, it is more dangerous than mainstream smoke inhaled by the smoker. Sidestream smoke, which is not filtered through the unburned tobacco and cigarette filter, has a lower combustion temperature. Consequently, all substances in tobacco smoke are in greater concentration in sidestream smoke, and in vitro it is more carcinogenic than mainstream smoke. The issue is whether nonsmokers' exposure to sidestream smoke, or "passive smoking," is enough to pose a health hazard is a subject of much scientific and public debate.

Only two aspects are no longer debated: Children exposed to parental smoking clearly are at risk of impaired health, and passive smoking by adults does not result in clinically significant chronic obstructive pulmonary disease (COPD). The data on lung cancer are controversial; the data on asthma are limited to individuals whose asthma was stable or well controlled at the time of testing. Few studies have been done on the relationship between involuntary smoking and cardiovascular disease.

INFANTS AND CHILDREN

It is well documented that parental smoking

threatens the health of children. The risks are greatest for children exposed during the first two years of life. The incidence of hospitalization for bronchitis and pneumonia is higher among children of smokers. They have increased airway reactivity and greater frequency of respiratory illnesses and symptoms, including cough, phlegm production, and wheezing. About 20% of childhood asthma is caused by parental smoking.

Smoke-exposed children also have smaller rates of increase in lung function as the lung matures. Passive smoking as a child may lead to a detriment of 3% to 5% in FEV₁ levels at age 20. This is probably clinically insignificant; the question is whether subtle damage predisposes to lung disease in adulthood, such as COPD among smokers.

LUNG CANCER

The risk of lung cancer to passive smokers remains the area of greatest controversy despite the Surgeon General's estimate that 5,000 deaths per year in the U.S. are due to lung cancer caused by passive smoking. Although sidestream smoke is unquestionably carcinogenic, it is uncertain whether exposure is adequate to overcome a biological threshold effect to cause cancer. It is estimated that passive smoking is equivalent to actively smoking 0.1 to 1.0 cigarette per day.

By extrapolating from the known risk of lung cancer in heavy smokers, the expected relative risk (assuming no threshold effect) from such low-level exposure is about 1.03 to 1.38. That is, a nonsmoker who experiences significant passive smoking might be expected to have a 3% to 38% increased risk of lung cancer compared to a nonsmoker who is not exposed to environmental tobacco smoke.

At least 13 epidemiologic studies in several countries have investigated the relationship between passive smoking and lung cancer. All but two or three of these studies report a modest elevation of the risk of lung cancer among involuntary smokers, but in only six studies was the positive association statistically significant. Pooled results of all studies suggest that heavy passive smoking does increase the risk of lung cancer by about 10% to 30%. This conclusion is not accepted by all experts, as most of the studies have some elements of bias, such as possible misclassification of lung cancer and passive smoke exposure, or interviewer and respondent bias.

ASTHMA

Three studies have evaluated the effects of passive

smoking on persons with a history of asthma; nearly all subjects had relatively inactive or well controlled asthma when tested.

One study (Shepard et al. *Environ Res* 1979; 20:392–402) demonstrated no effect on lung function in asthmatic persons exposed to two hours of passive smoking in an enclosed room. However, in a similar investigation, Dahms et al (*Chest* 1981; 80:530–534) found a 20% decrease in FEV₁. In our assessment of the acute effects of passive smoking on lung function and bronchial reactivity, spirometry remained stable and we found no change in airway reactivity (Wiedemann et al. *Chest* 1986; 89:180–185).

Although these three studies overall suggest no acute respiratory risk to young asymptomatic patients with a history of asthma, some important aspects have not been evaluated, including the delayed effects of exposure, the effects of chronic exposure, and the effect on patients who have active asthma.

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APNEA: EASY TO DIAGNOSE, DIFFICULT TO TREAT

Sleep apnea, which affects 1% of the population, has far-ranging consequences, including a spouse who refuses to share the patient's bed because of loud snoring, daytime hypersomnolence that can be incapacitating, and cardiorespiratory sequelae that may be life-threatening.

CHARACTERISTICS

Apnea is defined as cessation of airflow for at least 10 seconds during sleep. The apnea index is the number of apneas per hour. A person with sleep apnea syndrome has an apnea index greater than 5, with apneas occurring during both REM and non-REM sleep.

Early symptoms of sleep apnea are nocturnal and are characterized by loud snoring and episodic breathing