exposure or the individual hosts response to the MAC after exposure? After all, these are not called atypical mycobacteria for nothing.

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Pulmonary Vascular Involvement in Pulmonary Histiocytosis X

To the Editor:

We read with interest the communication by Harari et al (April 1997) regarding their finding of pulmonary hypertension in a group of severely affected patients with pulmonary histiocytosis X (PHX) who had been referred for lung transplantation. Their findings are intriguing and add to data indicating the importance of the pulmonary vascular involvement in this disease. Vascular involvement has frequently been described pathologically in PHX. Travis and coworkers found evidence of vascular involvement in 80% of biopsy specimens. The pathophysiologic significance of this vascular involvement has received little attention.

We reported the pulmonary function and exercise performance of a cohort (n = 23) of less severely affected patients with PHX. The group demonstrated normal lung volume (total lung capacity, 90% predicted) and mildly altered spirometry (FEV1 = 77% predicted, FVC/FVC = 80%) with a disproportionate reduction in diffusing capacity for carbon monoxide (DCO, 59% predicted). The exercise performance for the group was reduced (workload at maximum exercise, 54% predicted) with an abnormal dead space to tidal volume ratio (41%, at rest), which failed to fall with exercise (41%, at peak exercise). This combination of a low DCO and abnormal physiologic dead space ventilation (Vd/Vt) strongly suggest the presence of pulmonary vascular abnormalities. Finally, linear regression analysis demonstrated that these parameters (Vd/Vt and DCO) together explained 55% (partial r2 = 0.55) of the variability in exercise performance of the group [maximum workload achieved = 0.884×(0.008×Vd/Vt at rest)−(0.002×residual volume)+(0.0044×DCO); r2 = 0.73]. We concluded that the functional limitation in activity these patients experience, as reflected by diminished exercise performance, may be due in large part to pulmonary vascular involvement by the disease process.

As yet, there have been no therapeutic trials with interventions aimed at vasodilation as a primary treatment for this condition. Our experience and that of Harari et al suggest that this might be a useful avenue to pursue.

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Usefulness of Walking Test for Arterial Oxygen Desaturation Screening in General Population

To the Editor:

Patients with COPD often are hypoxic during exercise, even though they are not hypoxic at rest, and this exercise-induced hypoxemia may lead to pulmonary hypertension and heart failure. The early diagnosis of exercise-induced hypoxemia may facilitate early use of appropriate therapeutic approaches which, in turn, may improve the patient’s quality of life and prognosis.

To evaluate the frequency of exercise-induced hypoxemia in an apparently healthy population, a 5-min walking test with simultaneous measurement of oxygen saturation by pulse oximetry was performed by 360 subjects during a medical health examination. No subject had a previous history of lung disease or abnormalities on chest radiography. During the walking test, each subject was equipped with a pulse oximeter and was encouraged to walk as fast as possible. None of the subjects had hypoxemia before the exercise test.

Forty-one (11.4%) subjects developed oxygen desaturation during the walking test. Further investigation disclosed that, among these 41 subjects, there were 24 with COPD, 2 with arthritis rheumatoid-associated lung disease, 1 with diffuse panbronchiolitis, and 4 with severe scoliosis. The causative factor of exercise-induced hypoxemia could not be clarified in 10 subjects. The Hugh-Jones score and the pulse oxygen saturation at rest were not significantly different between subjects with and without oxygen desaturation (Table 1).