ative exposure in this investigation, are reported to be the most common bacterial contaminants of metalworking fluids.\textsuperscript{10-12} From an epidemiologic perspective, the observation of precipitins such as those identified by Bernstein and colleagues does not equate with disease, but precipitins are an excellent marker of exposure. At least one other immunologic study of workers exposed to highly Pseudomonad contaminated metalworking fluid (in that case \textit{Pseudomonas pseudoalcaligenes}) found exposure-related IgG antibodies to the bacteria in workers' sera, but reported no evidence of HP in that cohort.\textsuperscript{12}

There continue to be many unanswered questions about the causes, natural history, and clinical management of HP in general.\textsuperscript{13} The role of agents other than thermophilic bacteria and the impact of high levels of exposure (characteristics that typify metalworking aerosols) are particularly interesting factors that, as they come to be better understood, may challenge widely held assumptions about HP. One example may be the relative predominance of certain lymphocyte subsets in this group of diseases.\textsuperscript{14} The report of Bernstein and colleagues provides a sign post pointing us toward an important avenue of future research in what may become an all too common occupational lung disease.

\textit{Paul D. Blanc, MD, FCCP, San Francisco}

From the Division of Occupational and Environmental Medicine, University of California, San Francisco.

Reprint requests: Dr. Blanc, Division of Occupational and Environmental Medicine, UCSF, Box 0924, San Francisco, CA 94143-0924

\textbf{REFERENCES}


7 Eisen EA, Greaves IA. Asthma in automobile workers exposed to metal working fluids [abstract]. Am J Respir Crit Care Med 1995; 151:A421


\textbf{The Quest for Normal Values}

The risk associated with extrapolating linear regressions outside the range of data on which they were based is presumably well known to the readers of \textit{CHEST}. Some predictions of pulmonary function when extrapolated to short geriatric subjects yield negative values, which if issued on a computer-generated report would embarrass most pulmonary function laboratory directors. In this issue of \textit{CHEST} (see page 663), Enright and associates have provided normal values for the age group 60 to 85, a rapidly increasing segment of our white population that has been seriously underrepresented in previous studies. Interestingly, the data from this study agree closely with linear extrapolations into this age range from the most commonly used studies.\textsuperscript{1} The subjects in this study were volunteers carefully screened to exclude history of symptoms or diseases which might impair cardiopulmonary function. Strictly, these results should apply only to this population of Northern European ancestry who for the most part were lifelong residents of the urban nonindustrial Upper Midwest. However, similarities between these studies and other white populations in the United States and Europe support wider applicability, particularly in the absence of other data.

The chief clinical application of normal values is to identify individuals outside the normal range. Discrepancies between normal value studies can be problematic. An individual could be diagnosed as having mild airways obstruction or normal lung function with the same FEV\textsubscript{1} and FVC when compared to different normal values studies. Over the years, the science of normal value studies has improved. Initial studies were performed on patients hospitalized for diagnoses other than lung disease, but undoubtedly included many heavy smokers, patients with other diseases that impair pulmonary function, and individuals who by today's
The primary determinant of maximal flow and residual volume in adults is lung elastic recoil. Reductions in recoil appear to be the primary determinant of the reduction in both vital capacity and FEV₁ with age. Differences between biologic and chronologic aging rates may contribute to the variability of flow in the elderly; but it is unlikely to be the major determinant because although the fractional variability may increase, the absolute variability is not substantially different in young adults. Muscle strength should be a determinant of lung capacity and thus a potential predictor of FVC, although in the Minnesota study, this correlation was present only in those individuals in the lowest quartile of FVC.

If lungs were aerodynamically similar, flow would scale with volume, and FEV₁/FVC would be constant except for the differential effect of age on volume and flow. Although FEV₁ and FVC are highly correlated, there is a great deal of variability in FEV₁/FVC or other indices of relative emptying rate in all ages. This is maintained because during fetal development, airways rise from different primordial tissues than the alveolar tissue which is responsible for most of the volume and elasticity of the lung. Airway size may develop independently of lung parenchymal size. This has been termed dysynaptic growth. Therefore, for a given lung elastic recoil and volume, there is considerable variability in maximal flow. There is no known biologic marker to predict maximal flow based on genetic airway size to increase the sensitivity of flow measurements for disease detection.

In summary, in spite of recent advances in documenting normal values for lung function from growth and development through senescence, the sensitivity and specificity of these measures are limited by the large between-individual biologic variability. Our understanding of the structural and functional determinants of normal variability has not resulted in improved normal predictions. Serial changes in lung function remain a far more sensitive means of detecting early disease than isolated measurements.

Joseph R. Rodarte, MD
Houston

Professor and Chief, Pulmonary and Critical Care Section, Baylor College of Medicine.
Reprint requests: Dr. Rodarte, 6550 Fannin, Houston, TX 77030

REFERENCES

Downloaded From: http://journal.publications.chestnet.org/ on 11/08/2016
Tuberculosis Chemoprophylaxis and Physicians

In the past 2½ decades a significant body of evidence has shown, unequivocally, that isoniazid (INH) chemoprophylaxis markedly reduces the risk of developing active tuberculosis (TB), especially for recently infected individuals. Isoniazid, however, has been uncommonly associated with clinically significant hepatitis and rare fatalities have been reported, especially in older adults. As a result, guidelines for administration and monitoring of INH chemoprophylaxis have been devised with the intent of minimizing the risk of toxicity.

Despite the above knowledge, however, one wonders if anyone out there is really listening. Studies show us that physicians may be the worst offenders. Data from surveys show that as few as 8% of tuberculin-reacting physicians ever take isoniazid chemoprophylaxis for their own skin test conversion, let alone complete a fully prescribed course! Although the reasons for this are not clear, it is likely that when it comes to practicing what we should be preaching, in the depths of our subconscious the imp/angel paradigm of logic most likely often warps and wobbles our decision-making.

The Paradigm May Unfold as Follows

Angel: “Your lifetime chances of getting active disease once infected can be as high as 10%. Why, you can spread disease to your loved ones! You might die of it if you don’t prevent it. It makes rational individual sense and it is good public health policy to take INH for a few months. The medical literature supports its effectiveness. Besides, you should practice what you preach . . . .”

Imp: “Don’t listen to that public health rubbish . . . they’re more worried about the masses than about you, as an individual. Who cares if every one in a while somebody dissolves their liver with INH, as long as the proletariat is protected from spreading TB to the world? Your lifetime chances of getting TB are only as low as 4%. You have no symptoms, why risk getting sick from a medicine? Remem-