heart catheterization will be needed to accurately measure both left and right heart pressures.

Another interesting but disturbing observation is the very poor mortality rate observed in the subgroup of individuals with concomitant left ventricular dysfunction. Possibly, the preservation of cardiac output is as important in patients with IPF-associated PH as in those with idiopathic pulmonary arterial hypertension.

So, what will it take to change the fatalism with which many pulmonary physicians approach IPF? Since corticosteroid and antifibrotic therapies have not yet proven to be beneficial in randomized trials, might there be other options to improve IPF outcomes? Should we begin ordering serial echocardiography or measurements of brain natriuretic peptide levels\(^5\) to define the frequency of PH with rest and exercise as a target for therapy? The answer will be realized after the next round of clinical trials directed at IPF-induced PH, targeting functional outcomes.

Because IPF and most interstitial lung diseases are heterogeneous, some healthy blood vessels likely remain. These blood vessels, which are localized in areas of preserved lung parenchyma, might be amenable to vasodilator therapy, particularly if medial hypertrophy in response to pressure overload has developed. One challenge will be to match ventilation and perfusion to avoid worsened exercise hypoxemia.

There are other diseases in which so called fixed pulmonary artery hypertension has proven amenable to drug therapy. The response of thromboembolic PH\(^6,7\) and sarcoidosis\(^8\) to epoprostenol therapy serves as an example of how those blood vessels remaining in the lung may vasodilate and marginally improve cardiac output, pulmonary vascular resistance, and symptoms. Future trials in IPF patients will use the data of Nadrous et al to risk stratifying the patients and justify medication trials. These trials seem justified in the investigation of this devastating pulmonary disease.

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**Airflow Limitation as a Screening Tool**

Too Relevant To Ignore, Too Conspicuous To Apply?

Clinical logic tells that loss of pulmonary function heralds decline in (respiratory) health status, and the study of Xie et al\(^1\) in this issue of *CHEST* (see page 2448) may help further explore this logic. Pulmonary function decline gains importance when its impact on the general population is taken into account. Our study in family medicine, the Detection, Intervention and Monitoring of COPD and Asthma (DIMCA) program, for example, detected in 1991 a persistently reduced lung function or increased bronchial hyperresponsiveness in 7.7% of the general population, whereas another 12.5% showed a rapid decline in lung function (> 80 mL/yr) in combination with signs of bronchial hyperresponsiveness.\(^2\) A further 19.4% showed mild objective signs of COPD or asthma. However, research failed to directly confirm intuition; in analyzing airflow limitation in conjunction to other patient characteristics, a poor correlation was found between FEV\(_1\) and respiratory signs and symptoms.\(^3\) In the early stage, respiratory symptoms and airflow limitation form more or less independent aspects of
Chronic obstructive respiratory disease, and this helps explain the underpresentation by patients and underdiagnosis by (family) physicians of asthma and COPD. With its resistance to interventions other than smoking cessation, decline in FEV₁ appears to lose its central position in monitoring and managing COPD.

COPD is the end stage of a development over a long period of time, and this requires longitudinal research. So far, population studies have mainly focused on the rate of FEV₁ decline in relation to mortality or smoking behavior, whereas several randomized controlled trials on inhaled corticosteroid treatment also related health status outcome to the progression of the disease. As yet, there are no studies on early presentation of symptoms and their prognostic value for the development of COPD in the long term. In the mean time, Xie et al. present a longitudinal study demonstrating that the presence of airflow limitation predicted health-related quality of life 9 years later. Although the correlations found were weak, they pointed in the same direction in all domains. The findings of Xie et al. are in agreement with previous studies that showed that impaired health and functional status are independently associated with the severity of airflow limitation in subjects with undiagnosed disease, as well as in patients with established COPD. In our DIMCA program, a representative sample of subjects with formerly undiagnosed disease recruited in family practice was screened for respiratory symptoms and airflow limitation. Although the long-term follow-up results from the DIMCA cohort have not been published yet, our preliminary analyses show that baseline lung function is indeed associated with future respiratory health status as measured with the Chronic Respiratory Questionnaire. After 5 years, the group of subjects who were initially at risk for chronic respiratory disease (who also had a lower mean baseline postbronchodilator FEV₁) showed a mean Chronic Respiratory Questionnaire score that was 0.3 point lower than in the subjects who initially had good respiratory condition (5.95 ± 0.89 points vs 6.24 ± 0.56 points [± SD], p = 0.007). The DIMCA study also showed that impairment of quality of life due to dyspnea and fatigue and variability in lung function are better predictors of physician consultation than the mere presence of respiratory symptoms or a (gradually) reduced lung function.

It seems that the impact on quality of life comes next to the impact on mortality (in the study of Xie et al.1, an important reason for loss to follow-up among those with initially low FEV₁) and morbidity. In a recent article, Thomas and Levy state that “as COPD is an illness that has no cure, we must continue to strive for early detection and active management at all stages of the disease. The cycle of inactivity, social isolation, depression and de-conditioning so well described in COPD should be prevented.” Xie et al. conclude that the decrease of quality of life is largely mediated through the development of chronic respiratory symptoms. If symptoms do predict morbidity (and mortality) after all, early detection of COPD can be easily implemented in primary care practice by the use of office spirometry. Further research should point out whether early access to proactive care and thus to prophylactic vaccination, pharmacologic and nonpharmacologic treatment options, and early lifestyle interventions (including smoking cessation, improved diet, and increased exercise) prevents further deterioration of quality of life.

It remains an interesting question how many of the individuals in the study by Xie et al. actually had COPD. On the group level, impairment of baseline pulmonary function had a negative impact on quality of life in future years. However, the correlation of FEV₁ with quality of life was accompanied by a broad spread: where some suffer, others with the same degree of airflow limitation lead a relatively unimpaired life. This reduces the possibility to predict the individual course and consequently early detection of COPD.

And this brings us back at the heart of the matter. FEV₁ has been regarded a method for early detection of COPD, thus creating the opportunity of early intervention. In fact, this was the underlying objective of the DIMCA program. With an observation spanning 10 years, it has now become obvious to us that a firm individual clinical diagnosis is necessary for the interpretation of (group) correlations and progression in early stage COPD. The way forward is expected to come from adding analysis of individual medical histories over time to the population studies, in order to fine-tune this information and build evidence-based case-finding: detection and monitoring of patients with airflow limitation in order to prevent further loss of quality of life.

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Implementation of Guidelines on Hospital-Acquired Pneumonia

Is There a Clinical Impact on Outcome?

Hospital-acquired pneumonia (HAP) is a common and serious clinical problem. In fact, it is the second most frequent nosocomial infection and the first in the ranking of mortality. The incidence of HAP acquired outside the ICU has recently been studied by Sopena et al in a multicenter study reporting an incidence of 3 ± 1.4 per 1,000 hospital admissions and a crude mortality of 26% (14% of attributable mortality). Ventilator-acquired nosocomial pneumonia (VAP) is a form of HAP that develops during mechanical ventilation. The overall incidence ranges from 9 to 27% of intubated patients, accounting for a very high attributable mortality (range, 33 to 50%). This variability in the incidence and mortality of VAP reflects the different populations studied, the different methods used for diagnosis, as well as the different methods and prevention programs implemented.2,3

There are some medical interventions that can modify the outcome of HAP and VAP. The most effective intervention is early and appropriate administration of antibiotics.4,5 Initial appropriate antibiotic treatment is associated with lower mortality, shorter length of stay, and lower number of complications.4 Multidrug-resistant (MDR) microorganisms are responsible for a great proportion of inadequate initial treatments.4

The protocolization of the management of diseases is a key issue in modern medicine and should be carried out following evidence-based recommendations. In the last 10 years, several guidelines have been published on respiratory infections such as community-acquired pneumonia, COPD exacerbations, and HAP. The utility of these guidelines has been clearly demonstrated. For example, using a “before-and-after” design, Dean and colleagues6 showed a decrease in mortality (from 13.4 to 11%) after the implementation of the old American Thoracic Society (ATS) guidelines for CAP in a group of hospitals.

In the study published in this issue of CHEST (see page 2778), with the use of an observational cohort study with preguideline and postguideline data collection, Soo Hoo and colleagues7 studied the impact of the implementation of the 1996 ATS HAP guidelines8 in 61 episodes of severe HAP. Interestingly, implementation of the guidelines resulted in a higher percentage of adequately treated patients (81% vs 46%) and a lower mortality at 14 days (8% vs 43%). Appropriate imipenem use occurred in 74% of the cases with no increase in the imipenem-resistant microorganisms at the end of the study. This study confirms that the implementation of evidence-based protocols decreases the mortality in HAP. This type of study is absolutely necessary to convince healthcare authorities and medical communities of the importance of providing resources for the development and implementation of guidelines. These com-