embarrassing setting. Such persons deserve empathy and understanding. At the same time, the temptation to amend smoke-free policies to provide a smoking area for such persons is a simplistic answer and one to be avoided.

Another issue is that in many hospitals exceptions from the smoke-free policy have been made for chemical dependency or inpatient psychiatric units. The perception has been that these areas should be exempt because of the behavioral difficulties that the patients would experience if they were no longer allowed to smoke. The experience at the Minneapolis VA and the successful implementation of smoke-free policies in psychiatric hospitals in the state of Oregon indicates otherwise. Patients deserve the same consideration with regard to breathing clean indoor air irrespective of the disease for which they are hospitalized. Furthermore, to continue to allow smoking and to not address nicotine dependence in addictive disorders units seems to contradict the goals of these treatment providers, which is to provide complete care and address all addictions that the patient might have. An analogy would be for a patient with known quadruple coronary disease to undergo surgery, but only have a bypass performed on three of the vessels, even though all four were operable.

Finally, the issue of a physician writing orders for a patient to smoke in a smoke-free hospital is fraught with medicolegal implications and should be undertaken only with careful consideration and under the most unusual circumstances. It is especially important that any such exception have the active support of the nursing staff.

Hudzinski and Frohlich are to be commended for assembling the information that they have provided. It is only with such data that other medical institutions around the country will be encouraged to move forward with smoke-free policies. Such policies are long overdue. To continue to allow smoking in such facilities is inconsistent with the leadership role that medicine should take when facing this critical national health issue. The ramifications of a smoke-free policy in a medical setting are far-reaching. The ultimate impact of a more universal implementation of such policies could go well beyond any form of intervention that is currently available and could serve as a major foundation for efforts to help patients stop smoking. Physicians and hospital administrators need encouragement to move along toward the future when smoking will no longer be allowed within the confines of our medical institutions.

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As the Liver Goes, So Goes the Lung

In the 106 years since the initial recognition by Fluckiger and Vorkommen1 of cyanozis accompanying cirrhosis, the spectrum of liver-associated abnormalities that cause hypoxemia has expanded.2,4 Right-to-left shunt through intrapulmonary arteriovenous communications,2,3 pleuropulmonary, and porto-pulmonary anastomoses,4 rightward shift of the oxyhemoglobin dissociation curve, diffusion impairment, and ventilation-perfusion mismatching accompanying pulmonary hypertension, atelectasis, and/or pleural effusions are all mechanisms that can conspire against normal oxygenation in patients with liver disease. Among these various causes of liver-associated hypoxemia, intrapulmonary shunt through arteriovenous channels has attracted attention5 recently as a mechanism that can cause hypoxemia singlehandedly and as a phenomenon that appears to be associated distinctively with hepatic disease.

Confirmation that intrapulmonary arteriovenous channels occur in liver-associated hypoxemia comes from two major lines of evidence: (1) anatomic studies, in which the channels are sometimes visualized angiographically in vivo and/or demonstrated post mortem in latex-injected lungs,6 and (2) physiologic studies using several diagnostic techniques, including 99M technetium macroaggregated albumin perfusion scans to demonstrate right-to-left shunt,7,9 contrast-enhanced echocardiography to distinguish intracardiac from intrapulmonary sources of shunt,10 and multiple inert gas techniques.11
Recent interest and investigative activity aside, basic questions about liver-associated hypoxemia and arteriovenous communications remain unanswered. For example, we remain uncertain about the pathogenesis of these intrapulmonary arteriovenous channels. What is the relationship of the hypoxemia accompanying liver disease to failure of hepatic synthetic or metabolic functions? How does the presence of these channels anatomically correlate with their physiologic impact, and where disparity between anatomy and physiology exists, why?

To the clinician, even more pressing is the question of whether the hypoxemia accompanying liver disease is reversible. To the extent that reversibility is possible, what is the role of liver transplantation and can we predict preoperatively whether benefit can be expected from a transplant? Can nonsurgical strategies (eg, drugs, pheresis techniques) be useful in predicting the response to transplantation or in improving shunt without the need for liver transplantation?

Available experience about the reversibility of intrapulmonary shunts in liver-associated hypoxia is sparse, but impressions about reversibility are mixed. As with patient 37 in the accompanying study (see p 1165) (who showed no reversal of pretransplant hypoxemia when reexamined on the 25th day after liver transplant), experience with some hypoxemic cirrhotic patients undergoing liver transplantation has been disappointing. Based on the added surgical risk imposed by these patients' hypoxemia and the possibility of worsened postoperative hypoxemia that is incompletely correctable by oxygen supplementation, some investigators regard severe preoperative hypoxemia (ie, room air PaO₂<50 mm Hg) as an absolute contraindication to liver transplantation.

On the other hand, controversy exists about the role of liver transplantation because reduction of liver-associated intrapulmonary shunt and resolution of digital clubbing has been observed in other patients following spontaneous recovery from severe liver disease (severe hepatitis and parasitic infection) and following liver transplantation. Specifically, three available studies have observed improvement in liver-associated hypoxemia following liver transplantation. As early as 1968, Starzl and colleagues reported reduction in intrapulmonary shunt following liver transplantation in three hypoxicemic children. In a recent preliminary communication using serial multiple inert gas studies, Ekriksson and colleagues have reported improved oxygenation and resolution of intrapulmonary shunt and areas of low ventilation-perfusion ratio in six patients following liver transplantation. Finally, Stoller et al have reported reversal of hypoxemia and digital clubbing following liver transplantation in a 39-year-old woman with primary biliary cirrhosis and marked hypoxemia.

Clearly, the available experience is divided and does not permit a clear consensus on the advisability of liver transplantation for patients with liver-associated hypoxemia. Nevertheless, in view of the available reports that liver transplantation can at least occasionally reverse hypoxemia, much greater attention must turn to predicting which patients can be benefited.

The current report by Krowka and colleagues (see page 1165) is an important beginning in this effort. Their observation that contrast-enhanced echocardiography demonstrates intrapulmonary shunt in 9.7 percent of normoxemic liver transplant candidates vs 28.6 percent of hypoxemic candidates suggests a disparity between anatomic and physiologic evidence of intrapulmonary shunt. The normoxemic patients with abnormal echocardiograms (9.7 percent) presumably have a shunt which, for currently unclear reasons, has little physiologic impact. We may hope that future investigation of patients like these 9.7 percent (both pretransplant and posttransplant) will clarify why anatomically demonstrable shunts can be physiologically unimportant. Furthermore, serial evaluation of such patients following liver transplantation, using both contrast-enhanced echocardiography and other clinical techniques for measuring right-to-left shunt, may better identify patients most likely to experience pulmonary improvement following liver transplantation. In this way, we can better identify those patients for whom the lung will go as the liver.

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Adiaspiromycosis
An Uncommon Disease Caused by an Unusual Pathogen

Adiaspiromycosis is a pulmonary disease caused in humans by the fungus *Chrysosporium parvum var crescens* (*Emmonsia crescens*), a soil saprophyte of cosmopolitan distribution. The disease was first described in Arizona rodents by Emmons and Ashburn in 1942, and the first human case was reported from France in 1964. Subsequently, human infection was reported from Czechoslovakia, where most of the work characterizing this disease and its etiologic agent has been done, and from the USSR, Central America, and Venezuela. Although adiaspiromycosis occurs in rodents and other small mammals throughout the world, no autochthonous human cases have been reported from the United States.

The term “adiaspiromycosis” derives from the conidia of this fungus, the adiaconidia, which exhibit the unique property of progressive enlargement, without replication, at elevated temperature (approximately 37°C). An inhaled conidium, 2-4 μm in diameter, can grow to 200-400 μm or more, which results in a millionfold expansion of its volume. The pulmonary lesions produced by the enlarging conidia consist of discrete or confluent fibrotic granulomas, each containing one or more spherical, thick-walled adiaconidia that, when mature, are just large enough to be seen with the unaided eye. Proliferation or replication of the adiaconidia does not occur in human tissues. The granulomas therefore maintain a bronchiolecentric pattern of distribution indicative of an inhalational portal of entry, and secondary dissemination within or beyond the lungs does not appear to happen, despite an unconvincing claim to the contrary. Symptomatic disease has been attributed to inflammation and fibrosis associated with the granulomatous inflammatory reaction, and to displacement and compression of terminal airways and alveoli by massive numbers of progressively enlarging but sterile adiaconidia.

Because adiaspiromycosis occurs so infrequently in humans, little clinical experience with it has accumulated. The Czechoslovakian workers have proposed a clinicopathologic classification based upon the density of granulomas found in the lungs. *Solitary adiaspiromycotic granuloma* is an incidental finding in lung tissue removed for another reason; patients with this form of the disease have no symptoms or radiographic abnormalities attributable to the fungal infection. *Disseminated granulomatous pulmonary adiaspiromycosis* is a localized or diffuse bilateral pulmonary disease that has a finely nodular or reticulonodular radiographic pattern; only those patients with the densest, most severe bilateral involvement have had symptoms, which include fever, cough, and progressive dyspnea. No more than half a dozen symptomatic patients have been described. The diagnosis is usually established by lung biopsy. Pathologists unfamiliar with this disease are likely to mistake the adiaconidia for helminthic parasites, aspirated starch granules of lentil pneumonia, or more familiar large-form fungal pathogens such as *Coccidioides immitis* or *Rhinosporidium seeberi*.

In the present issue of *Chest* (see page 1171), Valente Barbas Filho et al report two Brazilian patients with progressive respiratory failure attributed to disseminated granulomatous pulmonary adiaspiromycosis, demonstrated by lung biopsy. Both patients recovered, and both were asymptomatic with normal chest radiographs and pulmonary function studies six months and one year later. The authors are appropriately cautious about advocating the use of systemic antifungal chemotherapy even for symptomatic disease. Published evidence indicates that the thick-walled adiaconidia are sterile cells incapable of replication in host tissues. Resolution of the disease without adequate therapy in these two patients further supports a cautious attitude about the need for specific antifungal therapy. The role of corticosteroids in the treatment of adiaspiromycosis remains largely unexplored. There is clearly much that remains to be learned about this uncommon and fascinating disease.

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