vascular bed not associated with decreased volumes, the DLco/VA ratio is decreased. A concomitant decrease in both VA and DLco would occur with less specific processes such as lobar or bronchial pneumonia involving alveoli as well as interstitium and pulmonary vasculature. Measurement of DLco at a higher Fio_2 allows estimation of the volume of the capillary bed (Vc) and the thickness of the alveolar capillary membrane (Dm). Most diseases primarily reduce the capillary bed, but *P. carinii* and viral pneumonias may be unique in primarily affecting Dm.[19,20]

Since the lung has a fivefold safety factor, there can be a considerable reduction in the DLco without an abnormality in Pao_2, particularly at rest. Conversely, the DLco can be normal or even elevated when gas exchange is abnormal due to airway abnormalities (*e.g.*, asthma) or shunts (*e.g.*, atrial septal defect). Nevertheless, several studies of diverse restrictive lung diseases, many similar to the interstitial processes described in the immunosuppressed host, have shown a good correlation between DLco and exercise-induced hypoxemia.[21–23] In one series of 106 patients, a DLco of less than 50% had a sensitivity of 89% and a specificity of 93% in the detection of a 4% or greater change in oxygen saturation with exercise.[21] A DLco of less than 60% raised the sensitivity to 100% but, characteristically, decreased the specificity for oxygen desaturation to 64%.

Although the DLco measurement is noninvasive, it is complex, entailing measurement of both inspired and exhaled CO and neon or helium. Interlaboratory variations can be great (>25%), and even in the same laboratory the coefficient of variation is generally 10%.[15] Therefore, the test has been criticized as imprecise.[24] However, in experienced laboratories, measurements can be quite consistent and correlate well with pathology and hypoxemia.[22,23] Nevertheless, because of its sensitivity in detecting otherwise unsuspected disease, DLco measurement has proved to be a useful test for earlier recognition of disease and for directing management.

Even more than VC and lung volumes, it is important to recognize the value of baseline DLco measurements. Not only is there a wide range for normal predicted values (80%–120%),[16,25] but, in addition, unrecognized processes may be present in the immunosuppressed host that result in an unsuspected low DLco. This has been well documented in IV drug abusers[26] and is a not uncommon primary finding in asymptomatic HIV seropositive patients without a history of drug abuse. The availability of baseline values for comparison when symptoms do occur will improve the sensitivity and specificity of the test.

Exercise and Metabolic Studies

The analysis of exhaled gases is used to calculate oxygen consumption (Vo_2), CO_2 production (Vco_2), respiratory quotient ($RQ = Vco_2/Vo_2$), and wasted ventilation (VD).[27,28] Automated equipment now makes it possible to perform these tests continuously during rest and exercise, and even in the critically ill patient at the bedside.[29,30] These tests have utility in defining the cardiac and pulmonary limitations of the recovering patient. They may also provide insight into the metabolic and ventilation management problems in the acutely ill patient (*e.g.*, to define the level of total parenteral nutrition needed in the septic patient).[31] These measurements are costly and difficult to do accurately in acutely ill patients, are still limited in use, and therefore will not be discussed further.

PULMONARY FUNCTION TESTS IN INFECTIOUS DISEASES

Pulmonary infections account for about 75% of the pulmonary complications in the immunosuppressed host and more than 90% of pulmonary complications in those with severe neutropenia (<500 neutrophils/mm^3).[32] In patients with AIDS, pulmonary Kaposi's sarcoma, lymphoma, and interstitial pneumonia are frequently encountered,[33] but even in those with AIDS, over 90% of serious pulmonary complications in 441 patients were due to infections.[34] Early detection and diagnosis is necessary to prevent mortality, which reaches 50% or even higher if treatment is delayed.[32] In general, the infectious diseases are space-occupying processes resulting in restrictive lung disease. The VC in combination with chest radiograph defines the severity of the restrictive process, but reductions in VC without radiographic abnormalities can occur. It is the decline in VC or DLco that often directs attention to the need for additional studies such as sputum induction or bronchoscopy.

Obstructive airways disease can occur either by direct involvement of the airway (bronchitis, bronchopneumonia) or via as yet ill-defined hypersensitivity or irritant factors.[35] Airway obstruction may not be evident from the usual physical signs such as wheezes and rhonchi, because flows are low in the immunosuppressed host, who commonly has restrictive lung disease with low tidal volumes. Spirometry may be misleading as well when the patient is unable or refuses to fully exhale because of pain or cough. Bronchodilator aerosols may be beneficial and indicated in any acute pulmonary infection because of the benefits of increased

ciliary activity,[36] airway smooth muscle relaxation, and a reduction in the cough stimulus.

Bronchiolitis obliterans is an inflammatory process thought to be related to preceding viral infections.[37] It is now a common sequela of heart–lung transplantation[38] and graft-vs-host disease.[39] It is a disease in the distal airways where flow is low and the usual signs of wheeze and rhonchi may not be present. Early recognition will be missed without spirometry.

Gas exchange, in particular hypoxemia, is the most critical determinant of the severity of lung disease. When patients are symptomatic, arterial blood gas values or pulse oximetry may indicate disease more or less severe than that suggested by signs and symptoms and may alter management decisions regarding hospitalization or even intensive care unit (ICU) admission. On the other hand, some diseases may be very subtle in onset with vague symptoms of fatigue, malaise, or low-grade fever; DLco measurements or exercise hypoxemia may provide objective evidence of significant or progressive disease. This picture is not unusual with the most common opportunistic infection in the AIDS population, *P. carinii* pneumonia. Since this entity has been most extensively studied it will be discussed next and used as the primary model for the use of PFTs in managing the immunosuppressed host.

Pneumocystis carinii Pneumonia

Even before the AIDS epidemic, *P. carinii* was a common opportunistic infection in immunosuppressed patients, but attack rates were generally less than 1% per year.[40] The frequency and characteristics of the disease are quite different in the AIDS population.[41] It is the primary diagnosis in 64%, and over 80% of AIDS patients will experience at least one episode of *P. carinii* pneumonia.[34,35,42,43] In the non-AIDS patient, *P. carinii* pneumonia is more acute in onset.[42,44] In the AIDS patient, *P. carinii* pneumonia often develops slowly and subtly, with only the nonspecific signs of nonproductive cough, dyspnea on exertion, low-grade fever, and, in 10% to 25%, a normal chest radiograph.[41,45,46] Mortality in immunosuppressed children is less than 20%, but it is in excess of 30% in adults.[44] Mortality may be decreasing, perhaps due to earlier diagnosis and more effective treatment.[34] Prevention may be very successful with trimethoprim sulfa (TMS), three days per week, in the non-AIDS immunosuppressed host.[47] Toxic reactions to TMS and other drugs used for prophylaxis are more common in the AIDS patient, so that prophylaxis is less successful, with recurrence rates in excess of 30%.[43] Recently pentamidine aerosol every two to four weeks has gained wide acceptance and has reduced recurrence rates.[48–51] This treatment has resulted in yet a more subtle onset of disease that may be cystic and limited to upper lung fields and confused with tuberculosis.[52–55] Common, early manifestations of *P. carinii* pneumonia are abnormalities in pulmonary function. Therefore, PFTs may be helpful in earlier diagnosis and improved management.

There have been a number of studies of PFTs in AIDS patients with *P. carinii* pneumonia, summarized in Table 12-1.[19,56–67] A reduction in VC or TLC below 80% of predicted occurs in less than 50% of patients with *P. carinii* pneumonia and is neither sensitive nor specific. A reduction in VC of 10% or more than 400 cc from a previously determined baseline value may be a simple and more sensitive indicator of early disease, but prospective studies to test this hypothesis are needed.

The FEV_1/FVC is characteristically high in *P. carinii* pneumonia as it would be with other interstitial pneumonitis due to the increased lung elastic recoil. However, 20% of patients with *P. carinii* pneumonia may have a low FEV_1/FVC ratio indicative of airway obstruction not explained by cigarette smoking or other obstructive airways disease.[59,61] The etiology is unclear, but increased airway reactivity associated with altered immunologic states has been suggested.[35,59] Recognition is important so that bronchodilator treatment can be started. When doses of 100 mg or more of aerosolized pentamidine are used for treatment or prophylaxis, bronchospasm can occur without clinical signs or symptoms. Therefore, spirometry before and after the first pentamidine aerosol treatment is routinely done at our institution to determine the need for bronchodilator therapy prior to the pentamidine aerosol therapy.

A reduction in the DLco has been uniformly reported to be almost 100% sensitive for *P. carinii* pneumonia,[56–61,64] but again, is nonspecific. This is particularly true in the IV drug abuser, who frequently already has a decreased DLco,[26] apparently from capillary obstruction from IV contaminants. The same may also be true for non-drug-abusing AIDS patients. We have found that a large number of AIDS patients have a low DLco without prior *P. carinii* pneumonia or other obvious lung disease (Fig. 12-3,B). A recent report on 24 asymptomatic, HIV-positive individuals found 12 (50%) had a DLco below 80%.[68] Similar findings have been reported in patients with AIDS-related conditions (ARC) and AIDS without known pulmonary complications.[69] Therefore, a baseline DLco determination is essential if the DLco is to be useful in the ongoing management of the AIDS patient.

The pathophysiology of *P. carinii* pneumonia is somewhat unique in that the organism appears to

Table 12-1. Pulmonary Function in Patients with *P. carinii* Pneumonia

Pt. Group, Reference	No.	FVC <80% of Pred. (No.)	FVC % Pred.	TLC <80% of Pred. (No.)	TLC % Pred.	FEV_1/FVC < Pred. (No.)	FEV_1/FVC Mean (%)	DLco <80% of Pred. (No.)	DLco % Pred.	O_2 Rest Abn. (No.)	O_2 Rest Mean (mmHg)	O_2 Exercise Abn. (No.)
Acute PCP[57–60]		8/15	78	5/15	89	5/24		27/27	43	18/28		20/22
KS		3/8	94	0/4	112	12/12		12/14	41	14/15		6/15
CMV		1/4	100	1/4	112	5/12		6/8	65	6/8		2/7
Acute PCP[61]	36	21/36	74	15/36	83	8/36	81	35/36	50	10/22		20/22
Acute PCP[62]	43											
Survivors	30									24/30	30†	
Nonsurvivors	13									12/13	45†	
Acute PCP[63]	25		52		67				60		69‡	
Non-PCP	7		73		78				77		73‡	
Acute PCP[64]	30		53				84	30/30	56			
Non-PCP	24		84				76		75			
Acute PCP[19]	20		57		71		78	20/20	58			
Non-PCP	16		83		95		82	16/16	61			
Recovered from PCP	10				88				80			
Acute PCP[56]	9	3/9		4/9		0		8/9		5/7		
Non-PCP	7	2/7		3/7		0		6/7				
Late recovery	6	3/6		2/6		1/6		3/6				
Acute PCP[66,67]	13		52		73		78		60		74‡	
Recovered from PCP	13		78		84		77		62		88‡	
Acute PCP[65]	7									7/7		
Recovered from PCP	7	2/7		2/7		1/7		3/7		2/5		2/7

*Abnormal: $P_{A-a}O_2$ < 20 mmHg or PaO_2 < 80 mmHg or O_2 saturation < 95%.
†$P_{A-a}O_2$.
‡PaO_2.

Comments: These comments refer to the studies cited in the table, in the order shown.

Stover's group[57–60]: Restrictive lung disease occurred in 30% to 50% of patients with *P. carinii* pneumonia but with a lower frequency in those with Kaposi's sarcoma and CMV infection. Obstructive airways disease occurred in 20% of patients with *P. carinii* pneumonia but in 100% of those with Kaposi's sarcoma. DLco values and an increase in $P_{A-a}O_2$ with exercise best discriminated *P. carinii* from other infections.

Nisam *et al*[61]: Only 50% of patients had restrictive lung disease and 22% had possible obstructive airways disease (FEV_1/FVC less than predicted), but over 90% had abnormal DLco values and exercise O_2.

Brenner *et al*[62]: The mean $P_{A-a}O_2$ was lower in survivors ($P = .05$), but values were normal (<20 mmHg) in only six of 30 survivors.

Bigby *et al*[63]: Restriction and low DLco values were most common in patients with *P. carinii* demonstrated in sputum; patients without *P. carinii* pneumonia had CMV, Kaposi's sarcoma, or tuberculosis.

Hopewell *et al*[64]: FVC, FEV_1/FVC, and DLco values were significantly lower in patients with *P. carinii* infection, but there was considerable overlap. Only DLco was 100% sensitive for *P. carinii* pneumonia.

Sankary *et al*[19]: Patients were selected for study because of low DLco values. A low DLco value due to low Dm increased in ten of ten patients after treatment, but the mean Vc did not change.

Coleman *et al*[56]: In the acute group, less than 50% had restriction, 89% had low DLco values, and 71% had abnormal O_2 values. On recovery from *P. carinii* pneumonia the restriction and perfusion defects persisted in 50%; airway obstruction persisted in ony one of six.

Leoung *et al*,[66] Wharton *et al*[67]: All patients exhibited improved lung volumes and O_2 values, but DLco decreased in two and mean values remained low.

Suffredini *et al*[65]: Long-term (15 to 21 months) survival from *P. carinii* pneumonia occurred after renal transplantation. Mild abnormalities persisted in five of seven patients.

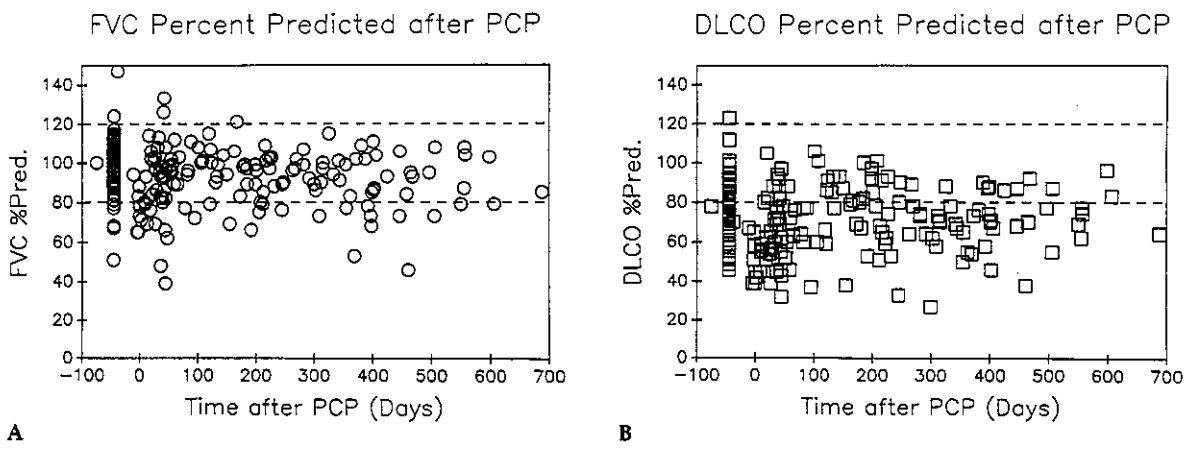

Figure 12-3. FVC and DLco measurements in a random population of HIV-positive patients referred for pentamidine aerosol prophylaxis. The majority were studied at variable times after a first bout of *Pneumocystis carinii* pneumonia (PCP). (**A**) Before PCP, only 5 (8%) of 61 had low FVC values and in most patients the FVC returned to normal after PCP. (**B**) Before PCP, 29 (48%) of 61 had low DLco values. Some had other pulmonary disease, but in most the explanation for the low DLco was unknown. After PCP, the DLco remained abnormal (<80% of predicted) in the majority of patients.

produce a true thickening of the alveolar–capillary membrane characterized by a reduction in the membrane component (Dm) of the DLco equation rather than a decrease in the capillary bed, Vc.[19] Unfortunately, measurement of Dm is costly, cumbersome, and unlikely to yield a specific etiologic diagnosis since similar reductions in Dm have been described in other (viral) pneumonias.[20]

The Sao_2 at rest is neither sensitive nor specific but during exercise may approach 100% sensitivity, like the DLco.[56–63,65] In an excellent review of PFTs in AIDS,[58] the authors suggested that a 10 mm Hg increase in the A-a gradient from rest to exercise may differentiate *P. carinii* pneumonia from other conditions such as pulmonary Kaposi's sarcoma and cytomegalovirus (CMV) pneumonia. However, the wide range of severity of pulmonary involvment with *P. carinii* pneumonia and other diseases precludes high specificity of any of the PFTs, including the $A–ao_2$ gradient. The ultimate diagnosis and management, once a significant decline in DLco or oxygenation is detected, will depend on sequential evaluation of radiologic, microbiologic, and bronchoscopic data.[70]

With recovery from *P. carinii* pneumonia, the VC and resting Po_2 usually return to normal (Figs. 12-3,A and 12-4), but the DLco may remain abnormally low in a large percentage of patients (see Fig. 12-3,B).[56,65–67] The time course of recovery may be quite rapid (three to four weeks) (see Fig. 12-4,A), even with diffuse and extensive radiolographic findings. Abnormally low DLco values, on the other hand, may persist for months (see Fig. 12-4, B), as previously shown for viral pneumonias[20] and ARDS.[71,72] Exercise tolerance and oxygenation also return toward normal, but the limited data reported suggest that some mild decrease in oxygenation and exercise tolerance may persist.[65]

Cystic changes and spontaneous pneumothorax have been reported as an unusual manifestation of *P. carinii* pneumonia but may become more frequent as prophylaxis with pentamidine aerosol changes the pattern of relapse.[54,55] Acute onset of dyspnea or a decline in VC should indicate the need for an expiratory chest radiograph to detect pneumothorax.

Since the mean time to recurrence of *P. carinii* pneumonia may be as little as four months in patients without prophylaxis,[48] and even with aerosol pentamidine prophylaxis recurrence rates of 21% in nine months have been observed,[48] it is reasonable to monitor AIDS patients for early detection of the disease. A reduction of 10% (or 400 mL) or greater in VC may be used as an indicator of relapse, but the increased frequency of hyperreactive airways or bronchial infections may also cause a decline in VC; however, these reductions should be associated with obstructive airways disease and reversed with bronchodilators, while a decrease due to interstitial disease would not.

Since the decline in Pao_2 or an increase in $PA–ao_2$ during exercise is a more sensitive indicator than VC, either of these measurements should detect *P. carinii* pneumonia earlier. Resting $A–ao_2$ has been shown to be a good prognostic indicator of survival.[62] Earlier detection of disease, when only exercise o_2 is abnormal, may be one reason for the decreased mortality from *P. carinii* pneumonia in

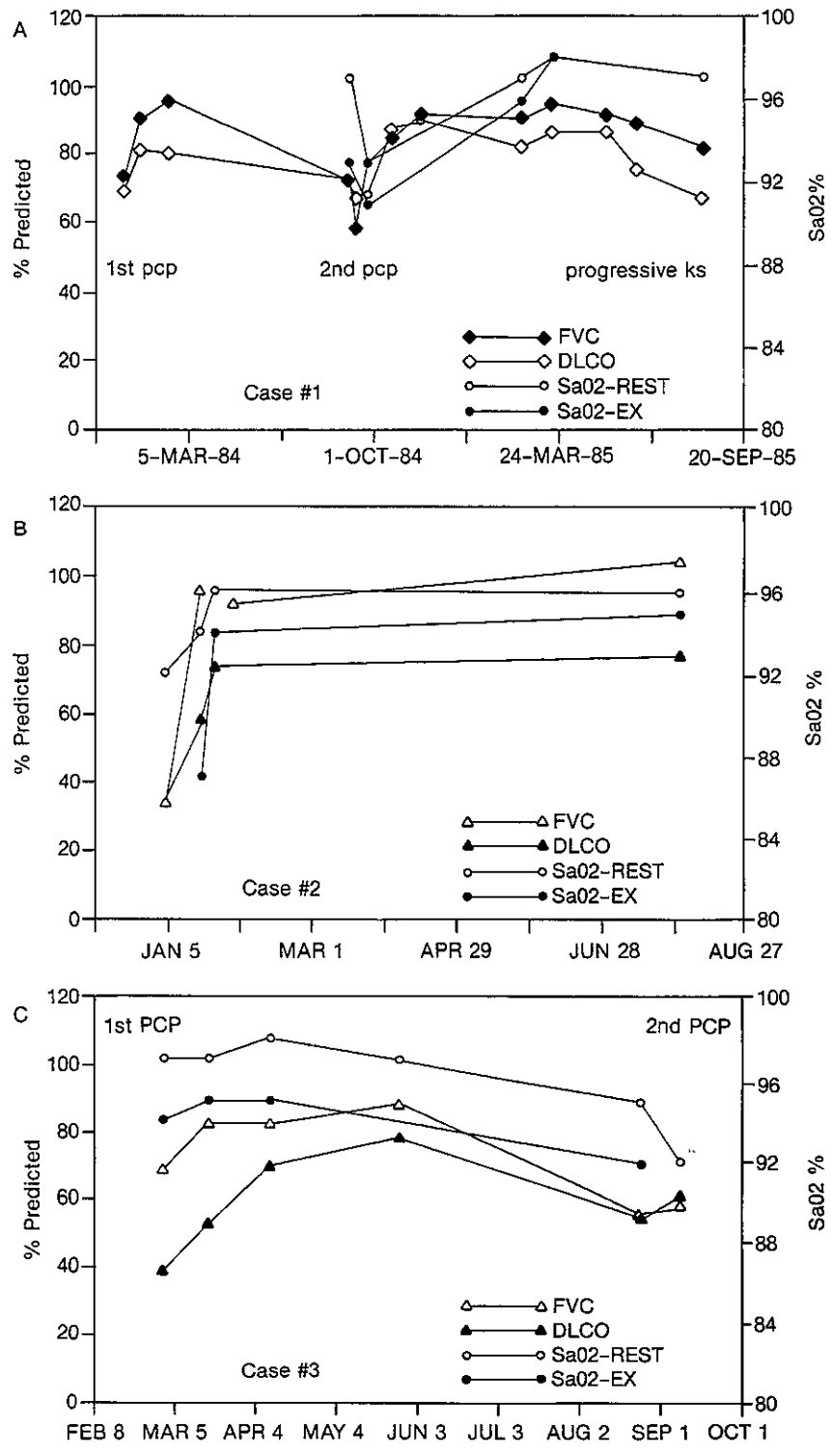

Figure 12-4. Serial studies of FVC, DLco, and Sao_2 in three patients after *P. carinii* pneumonia. (**A**) FVC returned to normal but DLco remained low after first PCP. All values declined acutely with second bout of PCP, returned to normal, then gradually declined again as pulmonary Kaposi's sarcoma progressed. (**B**) Despite diffuse, extensive pulmonary infiltrates on chest radiographs, severely decreased FVC and DLco values, and moderate hypoxemia with minimal exertion (Sao_2 = 87%), all parameters improved within one month, but DLco remained less than 80% of predicted. (**C**) Note increased sensitivity of DLco and exercise Sao_2 during acute PCP and the persistence of decreased Sao_2 with exercise and decreased exercise tolerance following the first bout of PCP.

recent studies. However, arterial blood sampling during exercise is invasive and not as readily available as pulse oximetry, which may provide equivalent information. Prospective trials are needed to test this hypothesis. In our experience, pulse oximetry and DLco are complementary and of about equal sensitivity and specificity. Because oximetry can be done by the practicing physician in her office or by a nurse or respiratory therapist in a clinic, it may be the preferred test for monitoring the AIDS patient.

Parasitic pneumonias other than *P. carinii* pneumonia have been described in the immunosuppressed host.[73] Of these, pulmonary toxoplasmosis may be underdiagnosed. In one series of 38 patients with disseminated toxoplasmosis, more than one third had pulmonary involvement.[74] How often the pulmonary involvement was clinically significant, however, remains in question. A common pathologic finding at autopsy is extensive interstitial pneumonitis with hyaline membrane formation, thickened alveolar septa, and prominent aveolar lining cells.[75] PFT results similar to those in a patient with *P. carinii* pneumonia might be hypothesized from these findings, but data are not available.

Bacterial Bronchopneumonia

Although emphasis is placed on opportunistic infection, bacterial bronchopneumonia also occurs frequently in the immunocompromised host. Among infectious diseases, bacterial pneumonia and septicemia are the leading causes of death in patients with malignancies and renal transplant patients, with mortality rates of 33% to 76%.[76] Bacterial pneumonia in AIDS patients is less common; in one series only 11 of 441 pulmonary complications were related to bacteria.[34] Community-acquired pneumonia is more common when AIDS is due to IV drug abuse, in which setting up to 10% of cases are bacterial in origin.[77] A cough productive of purulent sputum and obstructive airways disease diagnosed with spirometry suggest this diagnosis. If pulmonary infiltrates are present, some decline in VC and DLco may be expected, proportional to the radiolographic appearance. A disparity between these findings should point to other interstitial pneumonias. Unusual presentations of bacterial pneumonia in the immunosuppressed host, however, are being recognized; for example, *Hemophilus influenzae* may present as an interstitial process that is clinically and radiographically indistinguishable from *P. carinii* pneumonia.[78]

Sputum evaluation and radiography are clearly most important in diagnosis, but monitoring gas exchange is critical in the management of bacterial pneumonias. Hypoxemia can be more severe than viral pneumonia, and ventilation/perfusion abnormalities persist even after resolution of abnormalities on the chest radiograph.[79] This is particularly true in children, in whom bronchiolitis may be associated with ventilatory failure.[80] Severe hypoventilation ($Paco_2$ > 65 mmHg) signifies a poor prognosis[81] if not recognized promptly and corrected with ventilatory assistance.

Lobar pneumonia, when present, is associated with a decline in VC. The frequent association of pleuritic pain and splinting can further decrease the VC.[82] Hypoxemia and hyperventilation are characteristic.[83] With extensive bronchopneumonia and airways obstruction, ventilatory failure can occur; Pco_2 elevations can be expected if the VC or FEV_1 is less than 25% of predicted in acute obstructive airways disease.[84]

Viral Pneumonias

CMV is extraordinarily common in some immunosuppressed populations. It was documented post mortem by culture and inclusion bodies in 39 of 54 autopsied AIDS patients, 31 of whom had pneumonitis.[85] It has also been seen in over 30% of 386 bone marrow transplant patients[39]; the peak incidence occurs eight weeks after transplantation. CMV is commonly found in association with *P. carinii* pneumonia, but CMV itself can cause a severe diffuse interstitial pneumonitis that would have a PFT profile very much like that of *P. carinii* pneumonia with a decreased DLco and hypoxemia.[57–59] In a small series, it was suggested that the increase in the $A–ao_2$ gradient during exercise was less with CMV than with *P. carinii* pneumonia,[58,59] but this will clearly depend on the severity of the disease; distinguishing the two depends on cultures, bronchoscopic lavage, and biopsy. Deteriorating gas exchange during treatment for *P. carinii* pneumonia may identify patients with superimposed CMV pneumonitis and the need for specific antiviral therapy.

Pulmonary infections with herpes simplex virus occur less frequently than CMV pneumonitis in the immunosuppressed host. Both a focal pneumonic process (often with concomitant herpetic tracheitis and esophagitis) and a diffuse interstitial process (often with dissemination of disease to other organs) have been described.[86] Therefore, diverse PFT changes can be expected, and the diagnosis will depend on culture and biopsy results.

The greatest risk of varicella–zoster infection is in bone marrow transplant patients with disseminated disease, occurring in 15%,[87] but is not reported in AIDS.[33] Multiple peribronchiolar lesions can lead to respiratory failure and death.[88] Again,

chronic impairment in gas exchange has been demonstrated by decreased DLco, even after resolution.[89] In a series of 11 children studied one to 16 years after a serious varicella infection, three had restrictive defects, two had reduced DLco values, while none had obstruction; gas exchange was normal in all.[90]

Viral or "atypical" pneumonia was extensively studied and reported by Berven in 1962.[20] He showed that DLco may be impaired for several months after clearing of the radiolographic abnormalities. He further demonstrated the reduction in DLco was due to the membrane component (Dm), as has been recently shown with *P. carinii* pneumonia.[19] This finding is therefore not unique to *P. carinii* pneumonia. Klocke and colleagues could not confirm Bervan's findings, but noted that their patients had milder pneumonia.[91]

Mycobacterial and Fungal Pneumonia

There is an increased incidence of tuberculosis in immunosuppressed patients[92] and in patients with AIDS.[33,93] Extrapulmonary dissemination and miliary disease can cause severe hypoxemia with an ARDS-like picture.[94] Hematogenous tuberculosis is associated with a decreased diffusing capacity, hypoxemia, and residual restrictive disease.[95] In nonmiliary, pulmonary tuberculosis, declines in DLco and to a lesser extent in VC correlate well with radiolographic demonstration of involvement.[96] PFT studies in patients with chronic pulmonary tuberculosis have revealed proportional reductions in TLC, VC, and RV.[97] When the RV/TLC ratio was increased in one study of 30 patients, chronic bronchitis was frequently present.[97]

Pulmonary infection with atypical mycobacteria occurs in older patients with coexisting lung disease who are not immunocompromised.[97] In a series of 232 patients with *M. kansasii* infection and 120 patients with *M. avium-intracellulare* (compared with an equal age-matched group of patients with *M. tuberculosis*), the authors found obstructive airways disease to be more common in patients with *M. kansasii* infection (69% versus 57%) and restrictive lung disease to be more common in those with *M. avium-intracellulare* infection (68% versus 58%), but these ventilatory abnormalities did not correlate with radiolographic findings, as they did with *M. tuberculosis* lung disease.[98] Atypical mycobacterial infection responds poorly to treatment, but the disease is usually indolent and rarely disseminated.[99] However, in the AIDS population, disseminated *M. avium-intracellulare* infection is extremely common, reported in 17% to 28% of patients before death and in over 50% at autopsy.[100] Although *M. avium-intracellulare* is often recovered from respiratory secretions, its role as a pulmonary pathogen in AIDS is not clear.[100,101] The inflammatory response to *M. avium-intracellulare* is limited and organ function may be unaffected. Although 10% to 25% of patients with *P. carinii* pneumonia may also have *M. avium-intracellulare* infection, there is little evidence that this coinfection causes significant additional impairment in pulmonary function.[101] However, modest reductions in VC (73% of predicted) and more severe reductions in DLco (51% of predicted) have been reported with *M. avium-intracellulare* infection in AIDS.[69]

Pulmonary Mycoses

The occurrence of deep pathogenic mycoses (including *Nocardia* infection) is well described in immunosuppressed patients.[102] They are increasingly encountered in the AIDS population, in whom extrapulmonic dissemination is frequent.[33,42,103] The clinical and radiographic presentations are protean. Both obstructive and restrictive ventilatory defects have been described in acute disease.[104,105] Mild reductions in VC and TLC have been seen in one of four, and reductions in DLco in two of four patients with acute histoplasmosis.[104] In 19 patients with acute blastomycosis, four showed mild decreases in VC, one a decrease in TLC, five a decrease in resting oxygen saturation, eight a decrease in exercise oxygen saturation, and 16 of 19 had an FEV_1/FVC ratio less than 75% (but many patients had coexisting pulmonary disease and histories of cigarette smoking).[105] Importantly, histoplasmosis and blastomycosis have been associated with an ARDS-like picture in the AIDS patient.[106] Histoplasmosis may also cause a severe fibrosing mediastinitis, with pulmonary hypertension becoming a prominent feature.[107]

Opportunistic fungal infections cause serious disease in the immunosuppressed host, particularly in the neutropenic patient.[102,103] Invasive candidiasis is the most commonly encountered fungal infection in the neutropenic host, but clinically significant candidal pneumonia is uncommon.[103] In the AIDS patient, tracheoesophageal candidiasis occurs, but invasive bronchopulmonary disease is unusual.[42] Invasive bronchopulmonary aspergillosis and mucormycosis, however, may present with progressive pulmonary infiltrates in the immunosuppressed host,[42,103] at times with a vasculitic picture resembling acute pulmonary infarction.[108] Reports on PFT studies in these diseases are rare and probably limited to monitoring acute gas exchange abnormalities and to evaluating lung function recovery, both of which probably will correlate with chest radiographic findings.