Pulmonary Infection after Cardiac Transplantation: Clinical and Radiologic Correlations

Forty-one episodes of radiographically demonstrated pulmonary infection developed in 35 (30%) of 118 cardiac transplant recipients treated with cyclosporine. The most common pathogens were cytomegalovirus (CMV) (13 episodes), Pneumocystis carinii (12 episodes), and Aspergillus (five episodes). Appearance of CMV infection on radiographs was generalized and hazy \( n = 9 \) or limited to one lobe \( n = 4 \). All episodes of \( P \) carinii pneumonia, including six combined with CMV infection, appeared diffusely hazy. Aspergillus infection appeared either shaggy and nodular \( n = 3 \) or bibasilar and hazy \( n = 2 \). Aspergil-

Cardiac transplantation has proliferated since 1983, when cyclo-
sporine immunosuppression became generally available \( 1,2 \). For cardiac transplant recipients treated with cyclo-
sporine, 1-year survival is approximately 80%; 2-year survival, approximately 70%; and estimated 5-year survival, approximately 60% \( 1-5 \). The leading causes of morbidity and mortality in surgical survivors are cardiac rejection and infection, especially pneumonia \( 1-11 \). Radiologic descriptions of respiratory infections occurring after card-

PATIENTS AND METHODS

Use of cyclosporine as an agent for im-

Index terms: Drugs, effects, 60.251 • Heart,
transplantation, 51.459 • Lung, effects of drugs on, 60.251 • Lungs, infection, 60.2056, 60.2066, 60.2075

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Abbreviations: CMV = cytomegalovirus, HIV = human immunodeficiency virus.

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Table 1
Cardiac Transplantation: Mortality among 118 Survivors of the Operative Period

<table>
<thead>
<tr>
<th>Duration (mo) from Transplantation to Death</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cause of Death</td>
<td></td>
</tr>
<tr>
<td>Cardiac rejection</td>
<td></td>
</tr>
<tr>
<td>Pulmonary infection</td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td></td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
</tr>
<tr>
<td>&lt;4.0</td>
<td>17 (47)</td>
</tr>
<tr>
<td>4.1-12.0</td>
<td>10 (28)</td>
</tr>
<tr>
<td>12.1-24.0</td>
<td>6 (17)</td>
</tr>
<tr>
<td>24.1-36.0</td>
<td>1 (3)</td>
</tr>
<tr>
<td>&gt;36.0</td>
<td>2 (6)</td>
</tr>
</tbody>
</table>

Note.—Numbers in parentheses represent percentage of the 36 deaths; percentages may not total 100% due to rounding.

* Patient's blood tested positive for HIV.

† Patient was at risk for HIV.

RESULTS

Of the 118 patients, 82 (69%) were alive and 36 (31%) were dead as of September 30, 1988. For the 82 living patients, median follow-up time was 31 months (range, 15–66 months).

Causes of death versus duration of follow-up are shown in Table 1. Cardiac rejection was the leading cause of death and occurred throughout the first 24 months after transplantation. Pulmonary infection, which was the next most common cause of death, was fatal only in the first 4 months after transplantation (except for one patient at risk for the human immunodeficiency virus [HIV]). The miscellaneous causes of death were two unknown and one each of arrhythmia, slow tamponade producing cardiac arrest, mediastinitis and aortic rupture, pulmonary embolism, massive aspiration pneumonia, cerebral hemorrhage, and lymphoma.

Pulmonary infection after transplantation developed on 41 occasions in 35 (30%) of the 118 patients. Thirty-one (26%) of the 118 patients had a single episode of pulmonary infection, two had two separate episodes, and two had three separate episodes.

The likelihood of pulmonary infection was the same, regardless of sex. Thirty percent (30 of 101) of the male patients had pulmonary infection, compared with 29% (five of 17) of the female patients.

Ages of the patients with pulmonary infection ranged from 12 to 60 years (median, 44 years). The likelihood of pulmonary infection or pneumonia-associated death did not correlate with age.

The probability of an episode of pulmonary infection developing was greatest in the early months after transplantation and decreased thereafter (Fig 1). Eighteen (44%) of the 41 infectious episodes occurred within 4 months after transplantation and 29 (70%) within 12 months. In the 2d year after transplantation, only five episodes (12%) developed in the first 6 months and only three (7%) in the next 6 months. Over the interval of 2–3 years after transplantation, three additional episodes of pulmonary infection occurred in three (5%) of 56 patients; two of these patients had already had a previous episode of pul-

Figure 1. Graph depicts probability (Kaplan-Meier) of an episode of pulmonary infection developing after cardiac transplantation, expressed as a function of time after transplantation. For the four (11%) of 35 patients who had more than one episode of infection, the first episode is represented. Δ = one death associated with pulmonary infection.

Table 2
Pulmonary Infection after Cardiac Transplantation: Causative Organisms

<table>
<thead>
<tr>
<th>Organism</th>
<th>No. of Episodes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cytomegalovirus</td>
<td>13 (32)</td>
</tr>
<tr>
<td>P carinii</td>
<td>12 (29)</td>
</tr>
<tr>
<td>Aspergilus</td>
<td>5 (12)</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>2 (5)</td>
</tr>
<tr>
<td>Herpes simplex</td>
<td>2 (5)</td>
</tr>
<tr>
<td>Mycobacterium tuberculosis</td>
<td>2 (5)</td>
</tr>
<tr>
<td>Nocardia</td>
<td>2 (5)</td>
</tr>
<tr>
<td>Enterobacter aerogenes</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Enterobacter cloacae</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Klebsiella</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>1 (2)</td>
</tr>
</tbody>
</table>

Note.—Numbers in parentheses indicate the percentage of the indicated organisms detected among 41 episodes of infection. Ten episodes had no organism detected.
Infections with Specific Organisms

Causative organisms were identified for 31 (76%) of the 41 episodes of pulmonary infection (Table 2), with multiple organisms found in ten of them (CMV plus P carinii in six and one each of Aspergillus plus M tuberculosis, Aspergillus plus E aerogenes, CMV plus P aeruginosa, Aspergillus plus E coli plus Klebsiella). Death occurred in four (40%) of ten episodes of multiple-organism infections and in four (19%) of 21 episodes of single-organism infections \( (P = .2) \).

CMV was the most common organism infecting the lungs in this series (13 episodes in 13 patients). CMV pneumonitis was first seen radiographically a median of 2.6 months after transplantation (range, 1.1–6.1 months) (this range excludes one fatal CMV infection that developed 39 months after transplantation in a homosexual patient with Kaposi sarcoma). Each of the 13 episodes of CMV-associated infection was either radiographically diffuse \( (n = 9, 69\%) \) (Fig 2a–2c) or limited to one lobe \( (n = 4, 31\%) \) (Fig 3). Six (46%) of the 13 infections were associated with a fatal outcome; four of these infections were diffuse and two limited to one lobe. In the six patients with CMV as the only organism, three different radiographic patterns were found (two patients had the same pattern for each category).
diffuse haze (with bilateral symmetry in one), dense single lobe with small pleural effusion, and focal subsegmental opacity. In the six patients who had both CMV and \textit{P. carinii} (Fig 2a, 2b), radiographs revealed a diffuse haze in the lungs (with bilateral symmetry in five, small pleural effusion in two, and dense single lobe in one). Neither the presence of CMV pulmonary infection nor specific radiographic subpatterns (generalized or focal parenchymal abnormality or presence of pleural effusion) showed any correlation with the pretransplantation diagnosis of cardiomyopathy \((n = 76)\) or noncardiomyopathy \((n = 42)\).

\textit{P. carinii} was the second most common organism in this series (12 episodes in 11 patients). \textit{P. carinii} pneumonia was first seen radiographically a median of 3.9 months after transplantation (range, 2.6–10.3 months) (this range excludes three episodes—two of \textit{P. carinii} pneumonia and one of \textit{P. carinii} pneumonia plus CMV infection—that developed later in two patients at risk for HIV). In all 12 episodes of \textit{P. carinii} infection (including those in the six patients with combined \textit{P. carinii} and CMV infection), the radiographic pattern was diffuse. \textit{P. carinii} was the sole infective organism in six episodes in five patients. None of these six episodes was fatal. Radiographs obtained during each episode revealed diffuse pulmonary involvement in all six (completely symmetrical in two and nearly symmetrical in four). Three radiographic subpatterns (manifested in two episodes apiece) were identified: hazy (with sparing of the apices in one), finely reticulonodular (two episodes in a man whose blood tested positive for HIV), or reticulonodular (Fig 2d).

The third most common organism was \textit{Aspergillus} (five episodes in five patients) (Fig 4). These infections were first observed radiographically a median of 1.8 months after transplantation (range, 0.2–2.5 months). There were two major radiographic patterns: round, focal, shaggy opacities in three patients and bibasilar haze in two patients. One patient had four focal opacities; another, three; and the last patient, one. The focal opacities varied in maximum diameter from 0.9 to 8.0 cm (median, 3.8 cm) (Fig 4) and none was cavitary. Seven (88\%) of the eight opacities were in an upper lobe. Two of the three patients with focal opacities had no associated symptoms or signs. The two patients with bibasilar hazi-

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure45.png}
\caption{(4) \textit{Aspergillus} pulmonary lesions in a 60-year-old man 11 weeks after cardiac transplantation. Frontal radiograph, detail view, shows two poorly marginated, rounded, peripheral opacities (arrows) in right upper lobe. (5) \textit{E. cloacae} pneumonia in a 50-year-old febrile woman 11 months after cardiac transplantation. Frontal radiograph, detail view, shows irregular peripheral opacities in right upper lobe.}
\end{figure}

ness each had additional pulmonary disease: One had superinfected bilateral pulmonary infarcts; the other had bilateral pleural effusions, lung abscess (\textit{E. coli} plus \textit{Aspergillus}), and empyema (\textit{E. coli} plus Klebsiella).

Eight other proved organisms (Table 2), found in 12 episodes of pulmonary infection in 11 of the patients, were each limited to a single lobe or lung zone, except for in one patient with \textit{Nocardia} infection. Evidence of \textit{Nocardia} was seen radiographically in two lobes in one patient 1.0 month after transplantation as three shaggy focal opacities with maximum diameters of 2, 5, and 5 cm; these opacities cavitated as they decreased in size during a course of antibiotic therapy. The other patient with \textit{Nocardia} had two neighboring, noncavitary, well-demarcated, small (1.2 and 1.5 cm maximum diameters) nodules 6.9 months after transplantation. Neither of these patients had signs or symptoms of pulmonary infection.

Herpes simplex virus was found in two patients, each with transient pneumonitis and transient radiographic findings (onset, 4.2 and 10.7 months after transplantation). In each, a small discrete peripheral opacity was evident; one also had a small ipsilateral pleural effusion.

Each of the eight bacterial pulmonary infections, caused by various proved organisms, developed within the 1st year (0.3–11.0 months; median, 2.2 months) after transplantation. There were two episodes of \textit{E. coli} infection: One appeared radiographically as a focally dense, round opacity; the other, as a focal, ill-defined opacity in a lower lobe and an associated pleural effusion. In the two episodes with \textit{M. tuberculosis}, the infection appeared radiographically as patchy opacities in the left lower lung and a left pleural effusion (in a patient with tuberculous meningitis) and as a nodule in an upper lobe (the latter case was associated with \textit{Aspergillus}). One episode of \textit{E. aerogenes} infection (accompanied by \textit{Aspergillus}) was found by means of bronchoalveolar lavage performed 2 days before death. The one episode of \textit{E. cloacae} infection, manifested as opacities in a single lobe (Fig 5), responded dramatically to a course of cefotaxime. The one episode of \textit{P. aeruginosa} infection (associated with CMV) was seen as a focal opacity, approximately 6 cm in diameter, and containing air bronchograms in the right lower lobe. At necropsy the opacity proved to be an abscess.

\begin{table}
\centering
\caption{Infections with No Specific Organism}
\begin{tabular}{|c|c|}
\hline
\textbf{Infection} & \textbf{Organism} \\
\hline
Pericardial effusion & \textit{E. coli} \\
\hline
Parotid gland & \textit{E. coli} \\
\hline
Hepatic abscess & \textit{E. coli} \\
\hline
Intestinal perforation & \textit{E. coli} \\
\hline
Abdominal abscess & \textit{E. coli} \\
\hline
\end{tabular}
\end{table}

Infections with No Specific Organism

The ten episodes of clinically diagnosed pulmonary infection in which no specific organism was identified developed 19.7 months ± 10.9 (mean ± standard deviation) after transplantation, compared with a mean of 3.8 months ± 3.4 \((P < 0.001)\) in 28 episodes in which an organism was found. (Three episodes that occurred later in two patients at risk for HIV were excluded from this comparison.) On radiographs the infection
appeared focal and limited to a single segment in five episodes, patchy and limited to two or three basal segments in three episodes, as a general-ized haze in one episode, and as a left middle zone reticulonodular pattern in one episode. One death occurred in this group. 1.9 months after transplant.

**DISCUSSION**

The major findings of this study concern organisms, timing of pulmonary infection after transplantation, and radiographic patterns.

**Organisms**

Opportunistic pathogens are the most commonly detected organisms in pulmonary infections in cardiac transplant recipients treated with cyclosporine (6,7,9,10,13,15–20). Our data confirm this observation.

The most frequent organism in this series was CMV (Table 2), appearing in 13 (42%) of 31 episodes of post-transplantation pulmonary infection with a documented organism. Overall, the frequency of CMV pneumonitis in our series was 11% (13 of 118 patients), which is comparable to the frequency of 9% reported by Dum-mer et al (9) for cardiac transplant recipients receiving cyclosporine. CMV also has been described as the most common organism found in infected lungs in renal transplant recipients who were treated with cyclosporine (20). The considerable susceptibility of transplant recipients to CMV infection appears to be related to viremia and suppressed T cell–mediated immunity, but mechanisms of this susceptibility are not understood in depth (7,9,10,15,21). The virulence of CMV is a matter of debate (9,10,21,22). In our series, clinical criteria strongly implicated CMV pneumoni-tis as a major contributor to mortality in six (15%) of the 41 episodes of pulmonary infection (ie, in 5% of the total 118 patients). (Two of our patients with CMV pneumonitis have been previously described in detail [16].)

Early in the era of treating transplant recipients with cyclosporine, a report from Dummer et al (9) suggested that idiopathic cardiomyopathy might be a risk factor for post-transplantation CMV infection; how-ever, we found no significant difference in prevalence of CMV pulmonary infection between the patients with cardiomyopathy and those without (12% [nine of 76] vs 10% [four of 42], respectively). Dosage levels of cyclosporine employed in the early study (9) were far higher than those in the current study, so the two series are not strictly comparable. Our data indicate no excess risk of CMV pneumonitis developing in patients who have cardiomyopa-thy (presumably viral) before transplantation.

*P carinii* was the second most frequent organism in this series, appearing in 12 (38%) of 31 episodes of pulmonary infection with a documented organism. This frequency is higher than the 3%–20% reported by others after cardiac transplantation (9,10,23,24). Two contributing factors to this high frequency can be identified: In our series three episodes of *P carinii* pneumonia occurred in two patients at risk for HIV, and our series had a longer follow-up (15–66 months; median, 31 months), compared with shorter observation times in other series. The fact that varied frequencies of *P carinii* pneumonia have been reported among renal transplant recipients (20,25) also sug-gests that the frequency of this infection in cardiac transplant recipients may vary among institutions and geographic regions.

*P carinii* pneumonia, when unac- companied by other organisms, was always associated in this series with a favorable clinical outcome (no deaths and complete radiographic resolution). The only deaths associated with *P carinii* pneumonia among our 118 patients occurred in two patients who had coexistent CMV pneumoni-tis (one of these patients had Kaposi sarcoma, and presumably acquired immunodeficiency syndrome [AIDS]), but refused HIV testing; the other had disseminated CMV infection). These findings confirm previous observations that the course of *P carinii* pneumonia in the posttransplanta-tion population tends to be less severe than in patients with AIDS (18).

The third most common pathogen was *Aspergillus*, found in five (16%) of the 31 documented episodes. This finding agrees with those of Hofflin et al (10), who reported an 11% fre- quency of *Aspergillus* infection among 72 patients treated with cyclosporine (10). The series of Hofflin et al included one patient with *Candida* pneumonia; none was observed in our series. In most series, the likel-ihood of fungal infections in recipients of various transplanted organs has decreased since the advent of cy-closporine immunosuppression (6,10,20,21).

The frequency of bacterial infection after cardiac transplantation in patients treated with cyclosporine has also decreased. Others (10,21) have reported that aerobic gram-negative rods are the predominant bacter-i(al organisms, and our experience confirms this observation. Of eight bacterial infections in this series, six (75%) were caused by a range of five enteric bacteria and two (25%) by *M. tuberculosis*. Freeman et al (11) report-ed pneumococcal pneumonia as a late complication in five (25%) of 20 card-iac transplant recipients, but no such instances were found in our se-ries.

*Nocardia* is another organism for which frequency of infection among transplant recipients has decreased with the use of cyclosporine (10,13,20,21,24). *Nocardia* was found in only two (2%) of the 118 patients in this series, a finding also in accord with the 4% prevalence recently re-ported by Hofflin et al (10). The her-pes simplex virus and CMV are mem-bers of the herpes virus group (7); two (2%) nonfatal focal pulmonary infections with herpes simplex virus were found in our series. We found no episodes of *Legionella* infection, even though it has been reported as a complication of cardiac transplanta-tion, both with (10) and without (14) cyclosporine treatment. The epidemi-ologic patterns of infection with *Legionella* appear to vary among institu-tions and geographic regions, and perhaps in time as well.

**Timing**

The period of 0.8–4.0 months after transplantation was the time of high-est incidence of pulmonary infection (Fig 1) and highest mortality from pulmonary infections (Table 1), simi-lar to the experience with renal transplant recipients (15,20). The only other death from pulmonary in-fection (39 months after transplantation) was probably related to AIDS. Except for this latter patient, our data indicate that survival beyond 3 years after cardiac transplantation (n = 27 patients) carried no increased risk for pulmonary infection. Indeed, with the exception of the two patients at risk for HIV, each patient who had no known pulmonary infection in the first 26 months after transplantation (n = 38) remained free thereafter of pulmonary infection (Fig 1).

In transplantation populations generally, *Aspergillus* and CMV have...
been described as the infecting agents found most frequently between 1 and 4 months after transplantation (15,20). Hofflin et al (10) described eight heart transplant recipients with Aspergillus infections (four episodes involving the lungs) that were characterized by early time of onset (12-45 days after transplantation; median, 23 days). Our data confirm that experience, although our median onset (1.8 months after transplantation) and outer range (2.5 months) were somewhat later. The particularly early appearance of Aspergillus pulmonary infections probably relates to the nadir of T cell-mediated immunity in the first few weeks after transplantation (15). In a series of eight transplant recipients (four, heart only; four, heart and lungs) treated with cyclosporine, Dummer et al (9) reported the onset time of CMV pneumonia to be 1.1-6.5 months (mean 3.8 months), which is remarkably similar to the 1.1-6.1-month range we found in a similar group of 12 patients (excluding the patient at risk for HIV).

*P. carinii* pneumonia has been reported to develop most frequently within 2-6 months after transplantation in renal transplant recipients (15,20). In our series of heart transplant recipients, *P. carinii* pneumonia occurred between 2 and 11 months after transplantation. Why the range of onset for *P. carinii* pneumonia is somewhat later than those of Aspergillus and CMV is not known. One possible explanation is that the relatively high levels of corticosteroids given in the first months after transplantation may attenuate the inflammatory response to infection by *P. carinii* (17,26,27).

*Nocardia* infection was described by Hofflin et al (10) as a “late onset” (5-8 months after transplantation) phenomenon in three heart transplant recipients treated with cyclosporine. *Nocardia* pulmonary infection in our two patients developed 1.0 and 6.9 months after transplantation; thus, *Nocardia* infection may have a somewhat wider range of onset than previously appreciated. Herpes simplex pneumonitis also had a somewhat late onset in our series (4.2 and 10.7 months after transplantation). For all proved bacterial pulmonary infections in our series (n = 8), the entire 1st year (0.3-11.0 months) after transplantation was a period of risk.

One unanticipated finding in this series was the late onset (19.7 months ± 10.9 [mean ± standard deviation]) of ten episodes of clinically diagnosed pulmonary infection in which no organism was demonstrated. We tentatively suspect these episodes were predominantly bacterial in origin. Limitation of the infection to a single segment (n = 5) or lung zone (n = 4) in nine (90%) of ten instances and the favorable response of the infections to a course of empiric antimicrobial therapy (except in the one patient in this group who died) appear to support this speculation.

**Radiographic Patterns**

Pulmonary infection in immuno-suppressed hosts generally has one of three radiographic patterns: (a) diffuse, bilateral haziness, as in CMV or *P. carinii* infection; (b) patchy segmental or subsegmental opacity, as in bacterial infection; and (c) a focal nodule or nodules, sometimes cavitary, as in fungal infection or septic emboli (12,15,18,20,28,29). Our results confirm this general formulation, although certain atypical features were noted.

A diffuse, bilateral haze was the most common pattern in this series, characterizing nine (69%) of 13 episodes of CMV pneumonitis and all 12 (100%) episodes of *P. carinii* pneumonitis. CMV pneumonitis has been previously described as usually diffuse on radiographs (20,22). The hazy patterns of diffuse pulmonary involvement with CMV appeared indistinguishable from those of *P. carinii* pneumonitis (Fig 2). The rate of progression of radiographic changes in each of the patients with diffuse CMV pneumonitis or *P. carinii* pneumonitis was acute or subacute, which confirms previous descriptions (15,30).

*P. carinii* pneumonia was once regarded as commonly peripheral, sparing the periphery in its early phases (31). That pattern was not observed in this series. Patchy consolidation in the upper lobes, simulating the radiographic appearance of post-primary tuberculosis, has also been described for *P. carinii* pneumonia in immunocompromised patients (32), but this pattern was also not seen in this series. Although atypical patterns of *P. carinii* pneumonia have been commonly found in immunocompromised patients who have undergone transplantation (56% in a National Institutes of Health series [33]), only the typical pattern of diffuse haziness was seen in this series. Bibasilar haziness was found in two of the five patients with *Aspergillus* infection.

For this organism, a diffuse pattern is less common than focal disease (15,29).

Focal disease occurred with each organism in this series except *P. carinii* (in four episodes with CMV, three with Aspergillus, two with *Nocardia*, two with herpes simplex virus, and nine with various bacteria). As use of bronchoalveolar lavage increases, the detection of localized CMV pneumonitis (Fig 3) may possibly increase (16). In the three patients with focal Aspergillus pulmonary infection, seven (88%) of eight lesions were round and somewhat shaggy (Fig 4) in an upper lobe and were subacute (15,30) or chronic in their rates of radiographic change. These patterns have been previously described (29) and may be secondary to ventilation-perfusion inequality in the upper lung zones (34).

Herpes simplex virus pneumonia after bone marrow transplantation has been described as more commonly focal than generalized (35). The two patients with herpes simplex virus pulmonary infection in this series also had radiographically focal, acute disease. In general, focal viral infection in the lung is thought to represent contiguous spread of infection within the respiratory tract, whereas generalized viral pneumonitis is thought to represent a manifestation of hematogenous dissemination of the virus (9,35). In the seven patients with bacterial infections, as in the two with herpes simplex virus infection, acute disease was limited to a single lobe (Fig 5) (15,30). Because the patients with unidentified organisms causing pulmonary infection tended to have similarly focal and acute patterns of disease on radiographs, we suspect their infections were likewise predominantly bacterial.

Differential diagnosis of a focal pulmonary parenchymal opacity in the patient who has undergone transplantation includes postbronchoscopic bleeding. In one series of cardiac transplant recipients who underwent transbronchoscopic biopsy for suspect pulmonary infection, the rate of moderate (25-100 mL) hemorrhage caused by the procedure was 10% (17). Residual fluid may also remain in the lung after bronchoalveolar lavage (36).

The issue of the frequency of ordering chest radiographic examinations after cardiac transplantation was addressed in this study by a deliberate policy of frequent examinations (as described in Methods). The
data resulting from our series support this policy. Because of the high prevalence of lethal pulmonary infection in the first 4 months after transplantation, frequent examinations are particularly appropriate during this time. Asymptomatic pulmonary infection occurred as late as 6.9 months after transplantation; thus, frequent examinations remain indicated in this time frame. Because the likelihood of new pulmonary infection declined over the first 26 months after transplantation, gradual decrease in the frequency of follow-up chest radiographic examinations during this period appears to be justified. Any symptoms or signs suggestive of pulmonary infection should always lead to prompt radiographic examination in this population.

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References