Pericardial and Myocardial Disease Associated with Serological Evidence of Infection by Agents of the Psittacosis–Lymphogranuloma Venereum Group (Chlamydiaceae)

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SUMMARY
Nine patients with acute pericarditis or myocarditis, or both, were found to have serological evidence of concurrent infection by a psittacosis agent. Three patients had conclusive epidemiological and clinical evidence of complicating pulmonary psittacosis and demonstrated that psittacosis agent does produce inflammatory heart disease in this geographic area.

Six patients had imperfect corroborative evidence of psittacosis and their clinical manifestations were atypical. Indeed, half of these patients were initially believed to have some form of idiopathic myocardial disease. Studies of these patients suggest that psittacotic infection may be causal in certain forms of heart disease commonly designated primary myocardial disease.

Additional Indexing Words:
Pericarditis, acute	Myocarditis, acute	Radioisotope precipitation test
Epidemiological studies	Idiopathic myocardial disease

Numerous infectious agents may cause perimyocarditis in human beings, but evidence of cardiac involvement by agents of the Chlamydiaceae family group (Bedsoniaceae, Miyagawanella, Psittacosis-lymphogranuloma) is rarely sought since these agents have not been isolated from the heart, and their causative relationship to human perimyocarditis is unproven. Nevertheless, many reports inferentially link pulmonary psittacosis or serological evidence of infection, or both, by a member of the Chlamydiaceae family† to perimyocardial disease.

Adamy in 1930 first commented on the development of postinfectious myocardial degeneration associated with psittacosis. This and other reports prompted Lyon in 1956 to include psittacosis in his monograph dealing with infectious agents considered capable of affecting the cardiovascular system. Subse-

†This family is referred to hereinafter as psittacosis.

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830
CARDIAC DISEASES AND PSITTACOSIS

sequently, psittacosis infection has been implicated in the production of perimyocardial
disease in reports from Germany,10 the United
States,11-18 Japan,14 and Scotland.15 Grist and
McLean15 stated that "enlargement and failure
of the heart is a recognized feature of psittac-
osis."

Therefore, historical precedent combined
with the chance detection of two patients
with apparent acute psittacosis pericarditis
prompted us to investigate the role of this
agent in acute pericardial and myocardial
disease. A complement fixation (CF) test for
psittacosis agent antibody was included in
the battery of CF tests performed on sera
from 599 patients suspected of having acute
pericarditis or myocarditis, or both. Serolog-
ical evidence implicating the psittacosis agent
was obtained in nine patients and comprises
the substance of this report.

Methods

Study Population

A series of 599 patients with suspected acute
perimyocarditis was developed from two sources:
in study group I, 400 patients suspected of hav-
ing acute viral disease involving the heart had one
or more specimens of serum submitted to the
Viral Laboratories of the State of Illinois during
the years 1959 through 1965; and in study group
II, 199 patients considered to have acute or
chronic primary myocardial disease (PMD)
were studied at Cook County Hospital, Chicago.

If CF antibody to psittacosis agent antigen was
found and if either a fourfold rise or fall in titer,
or two or more sequential titeres of 1:16 or great-
er were present, the patient was examined for
historical or physical evidence of inflammatory
pulmonary or heart disease, or both. The criteria
used to establish the diagnosis of pericarditis
and myocarditis16 were reviewed recently (see
table 3). The criteria used to establish the diag-
osis of primary myocardial disease associated
with alcoholism or pregnancy and to include or
exclude hypertensive, arteriosclerotic, rheumatic,
and other forms of heart disease, have been pub-
lished.17, 18

Patient Studies Included

History. Age, sex, race, places of residence
(rural versus urban), occupation(s), pregnancies,
contacts with birds or animals, prior disease (that
is, trachoma, cat-scratch disease, lymphgranu-
loma venereum (LGV), Hodgkins disease,19

syphilis, pneumonia, or carditis), and season,
date of onset, and details of the current illness
were obtained.

Physical Examination. Symptoms and signs of
pulmonary (rales or dullness), pleural (friction
rubs or effusion), and pericardial inflammation
(friction rub, effusion, or positional pain-relief)
were sought. All evidence of heart failure (edema,
afficted venous pressure, audibility of the third
or fourth cardiac sound, cardiomegaly, tachycar-
dia) or other significant disease was recorded.

X-ray. Acute or chronic pulmonary infiltrates,
pulmonary congestion, pericardial or pleural ef-
fusion, or both, and abnormal cardiac contour
or cardiomegaly were noted from serial 2-m pos-
tero-anterior and lateral chest x-rays.

Electrocardiogram. Serial 12-lead electrocar-
diograms were recorded and analyzed according
to the criteria of the New York Heart Associa-
tion for: (1) disturbances of rhythm or conduction,
(2) changes of ventricular hypertrophy, damage,
myocarditis, or infarct, and (3) evidence of acute
pericarditis.

Ancillary Laboratory Procedures. These in-
cluded white blood cell count, antistreptolysin-O
(ASO) titers, and Kahn or Venerale Disease Re-
search Laboratories (VDRL) test in all cases.
Blood samples were drawn from all patients of
study group II (PMD) (but only irregularly
from study group I) for determination of fasting
and postprandial blood sugar values, cholesterol,
serum glutamic oxalacetic transaminase, cephalin
flocculation, thymol turbidity, total protein, and
for serum and hemoglobin electrophoretic stud-
ies. Additional studies included urinalysis, ven-
ous pressure, circulation time, and search for
lupus erythematosus cells by plasma clot method.

Serological Studies. Serial CF tests for antiviral
antibodies were done using appropriate serum
controls, initially by the macro technique, but
subsequently by the micro-titer method.20, 21 The
antigens included: mumps (soluble), poliomyelitis,
herpes simplex,22 adenovirus,23 Q-fever, Cox-
sackie B2,3,4,5, influenza A24 and B,25 para-
influenza 1, 2, and 3, and, when indicated, respi-
atory syncytial (RS).26 Unless otherwise stated, serolog-
tests for psittacosis were done with Psittacosis
Human Diagnostic Antigen* (purified element-
ary bodies from the allantoic fluid of hen's eggs
infectected with the DD34 strain of psittacosis agent).

Representative serum specimens were periodi-
cally checked for comparative purposes with two
other psittacosis antigens: Psittacosis Complement

*Markham Laboratories, Chicago, Illinois.
Fixation Antigen* (Cat. no. 75-0231), and Psittacosis Diagnostic Antigen† (the Hamilton strain propagated in chick embryo yolk sacs and harvested by heat and extraction).

Twelve samples of serum from four patients displaying CF antibody titers (>1:16) to psittacosis were also evaluated by the radioisotope precipitin test (RIP) for psittacosis group antibody. This test was performed by Dr. R. K. Gerloff.

Control Population

To serve as a frame of reference, specimens from three adult control populations were tested for complement-fixing antibody to psittacosis.

One thousand serum specimens were obtained from apparently healthy individuals in Freeport, Illinois, during the year 1961-1962.

Forty serum specimens were obtained from patients hospitalized at Cook County Hospital for miscellaneous noncardiac diseases. The patients in this group had similar ethnic and socioeconomic backgrounds, were from the same geographic area of northeastern Illinois, and were hospitalized during the same season of the same year (1963-1964) as the majority of patients in the study population.

Six thousand three hundred sixty-seven specimens from patients suspected of having viral respiratory disease were received by the Viral Laboratories of the State of Illinois between 1962 and 1965.

Results

Control Populations

Of 1,000 routine serum specimens from nonhospitalized residents of Freeport, Illinois, 4.6% contained antibody for psittacosis but no titer exceeded 1:8.

In the Cook County Hospital population antibody for psittacosis was found in three of 10 patients with liver disease, five of 18 patients with bacterial pneumonia, and six of 18 patients with traumatic injuries, but the highest titer recorded was 1:16.

Among the 6,367 specimens from patients with suspected noncardiac viral disease, 141 (2.2%) showed fourfold titer rises, or titers >1:32. In 35 of these 141 cases clinical data compatible with the diagnosis of lymphogranuloma venereum, cat-scratch disease, or febrile upper respiratory illness (URI) were obtained. Detailed information concerning these 35 patients is presented in table 1. Most patients had bird (parakeet, turkey, and pheasant) or animal (cat, dog, and rabbit) contact; the clinical diagnoses included lymphogranuloma venereum—one, cat-scratch disease—four, viremia or fever of undetermined origin (FUO)—six, upper respiratory illness (URI) or bronchitis—eight, and pneumonia—16. Sequential serum specimens were obtained in 32 patients; single specimens were obtained in three, but in each of these CF antibody titers exceeded 1:32. A fourfold or greater rise8 or fall in CF antibody titer was observed in 13 patients. Twenty patients had titers of 1:16 or greater. Significant evidence of infection by other agents was not found.

Table 1

Clinical and Serological Data on 35 Patients Displaying Evidence of Noncardiac Infection with Psittacosis Agent Studied by the Viral Laboratory, State of Illinois, During 1963 and 1964

<table>
<thead>
<tr>
<th>Age</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>15 yr</td>
<td>78 yr</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bird or animal contact</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Clinical diagnosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphogranuloma venereum</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Cat-scratch disease</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>FUO or viremia</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>URI or bronchitis</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Number of sera specimens per patient</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (CF titer &gt; 32)</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>3 or &gt;</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>CF antibody titer displayed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fourfold rise</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Fourfold fall</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Single (&gt; 32) or multiple 16 or &gt; titer</td>
<td>23</td>
<td></td>
</tr>
</tbody>
</table>

Study Population

Among the series of 599 patients investigated, nine had epidemiological, clinical, and laboratory evidence suggestive of acute infection by agents of the psittacosis group and

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*Communicable Disease Center, Diagnostic Reagents Division, Atlanta, Georgia.
†Lederle Laboratories, American Cyanamid Company, Pearl River, New York.
perimyocardial disease (table 2). Five were men and four were women. Five were Negro and four were Caucasian. Ages ranged from 32 to 60 years. The occurrence of illness was nonseasonal and no epidemiological factors suggested a common source of infection. In four patients cardiac disease other than acute pericarditis or myocarditis was also suggested by clinical data: arteriosclerotic heart disease with myocardial infarction, not recent in one; and primary myocardial disease associated with alcoholism in two and with pregnancy in one.

The nine patients were divided into subgroups A and B (table 2) on the basis of diagnostic criteria for infection by agents of the psittacosis group (table 3). Clinical data for these subgroups are listed in table 4 and the results of serological studies on sequential serum specimens are found in table 5.

Subgroup A

Three patients fulfilled the following criteria: (1) exposure to parakeets; (2) acute, radiologically documented pulmonary infiltrates; (3) concomitant fourfold or greater rise in CF antibody titer to routine psittacosis antigen; (4) complete clinical recovery; (5) favorable response to antibiotic therapy; and (6) no serological or historical evidence of trachoma, cat-scratch disease, lymphogranuloma venereum, or Hodgkin’s Disease.18

Two of these patients were men (one a physician), ages 33 and 45, and one was a woman, age 60 years. None had had antecedent cardiac or pulmonary disease (table 2). Each presented with evidence of acute

### Table 2

**Comparative Clinical Data of Patients with Acute Perimyocarditis by Subgroups**

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>A</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Age (yr):</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minimum</td>
<td>33</td>
<td>32</td>
</tr>
<tr>
<td>Maximum</td>
<td>60</td>
<td>50</td>
</tr>
<tr>
<td>Average</td>
<td>38</td>
<td>41</td>
</tr>
<tr>
<td>Sex:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Female</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Race:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Nonwhite</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Coexisting cardiac disease:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary myocardial disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post partum</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Alcoholic</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Arteriosclerotic heart disease with myocardial infarction</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

### Table 3

**Epidemiological, Clinical and Laboratory Evidence Indicative of Infection by Psittacosis Agent in Two Subgroups of Patients**

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>A</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Fourfold or greater rise in CF antibody titer</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Parakeet exposure</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Complete clinical recovery</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Leukocytosis</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Response to antibiotics</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Fourfold fall in antibody titer</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Multiple antibody titers of 1:16 or greater</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Other Chlamydiaceae disease*</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Chronic illness</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Acute perimyocarditis</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>

*Other disease due to Chlamydiaceae agents: Lymphogranuloma venereum, trachoma, inclusion blemorrhoea and Hodgkin’s disease.19
### Table 5

**Serial Changes in Reciprocal of Complement Fixation (CF) Antibody Titer to Psittacosis Antigen* in Each of Two Subgroups of Patients**

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Complicating CV disease†</th>
<th>Serological observation (wk)</th>
<th>Time and sequence of CF tests expressed as months after clinical onset of disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subgroup</td>
<td></td>
<td></td>
<td></td>
<td>1/2 1 2 3 4 5 6 7-12 13-24 25-60</td>
</tr>
<tr>
<td>1</td>
<td>45</td>
<td>None</td>
<td>4</td>
<td>8 64</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>None</td>
<td>11</td>
<td>0 32 16 16</td>
</tr>
<tr>
<td>3</td>
<td>33</td>
<td>None</td>
<td>3</td>
<td>32 256</td>
</tr>
<tr>
<td>4</td>
<td>46</td>
<td>PMD-PP</td>
<td>13</td>
<td>0 256 32</td>
</tr>
<tr>
<td>5</td>
<td>43</td>
<td>PMD-AL</td>
<td>145</td>
<td>0 32 32 16 16-64 0-32 32-16</td>
</tr>
<tr>
<td>6</td>
<td>32</td>
<td>ASHD</td>
<td>5</td>
<td>8 32 256</td>
</tr>
<tr>
<td>7</td>
<td>50</td>
<td>PMD-AL</td>
<td>148</td>
<td>8 0-32 32-32 0-32 64-16-0-32</td>
</tr>
<tr>
<td>8</td>
<td>38</td>
<td>PMD-AL</td>
<td>148</td>
<td>8 32-32</td>
</tr>
<tr>
<td>9</td>
<td>35</td>
<td>None</td>
<td>5</td>
<td>0-32 32-32</td>
</tr>
</tbody>
</table>

*Psittacosis Human Diagnostic Antigen, Markham Laboratories, Chicago, Illinois.
†Key: CV = cardiovascular disease; PMD-AL = primary myocardial disease, post partum; ASHD = Arteriosclerotic heart disease with myocardial infarction.

None of the remaining six patients fulfilled all of these criteria. Three of these patients were men and three women. Ages ranged from 23 to 59 years. Two had pneumonitis, and one patient had parakeet exposure (table 4). A fourfold or greater rise in CF antibody titer to psittacosis antigen was detected in all cases (table 5). In one case (case 5, table 5) sera cross-reacted with psittacosis control antigen in the radioimmunoprecipitation test (table 6), and in another case (case 6, table 5) sera cross-reacted with psittacosis control antigen in the radioimmunoprecipitation test (table 6). The results of this test are reported as a percentage of radioactive precipitate. Eleven sera from patients (cases 4, 5, 7, and 9) were subjected to the radioimmunoprecipitation test (table 6). The results of this test are reported as a percentage of radioactive precipitate. Eleven sera from patients (cases 4, 5, 7, and 9) were subjected to the radioimmunoprecipitation test (table 6).
CARDIAC DISEASES AND PSITTACOSIS

Table 6

Comparison of Complement Fixing Antibody Titer to Psittacosis Antigen* and Radioisotope Precipitation (RIP) Test for Chlamydia Group Antibody in Four Patients

<table>
<thead>
<tr>
<th>Case</th>
<th>No. sera tested</th>
<th>CF titer (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Sub-</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>group</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>B</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>4</td>
<td>35</td>
</tr>
</tbody>
</table>

*Psittacosis Human Diagnostic Antigen, Markham Laboratories, Chicago, Illinois.
†Corrected P values = Percentage of radioactivity of meningopneumonitis particles labeled with \( P_{32} \) precipitated by test serum. Normal limits: \(-20\) through \(+20\).

Two patients (cases 4 and 9) displayed both a fourfold rise and fall in CF titer and in three patients (cases 5, 6, and 8) persistent high titers have been detected for about 3 years. These three patients (cases 5, 6, and 8) are considered to have primary myocardial disease.

Five patients recovered and are in apparent good health. One alcoholic patient (case 8) with primary myocardial disease has had recurring evidence of congestive failure for 4 years. Histological examination of his pericardium and myocardium obtained by biopsy 2 months after the onset of disease and after tetracycline therapy disclosed an active, chronic nonspecific inflammatory process without inclusion bodies; cultures of myocardium, blood, stool, and sputum for infectious agents were negative.

No patient had historical or serological evidence of syphilis; none had lymphogranuloma venereum, trachoma, cat-scratch disease, or Hodgkin's disease.

Discrimination

Agents of the psittacosis family cause widespread disease in birds, animals, and man. Diseases recognized in human beings are psittacosis, ornithosis, lymphogranuloma venereum, cat-scratch disease, trachoma, and inclusion blennorrhea. These infectious agents can be identified by light microscopy, a method used in diagnosis of trachoma and inclusion blennorrhea. Isolation and identification of the agent from sputa in pulmonary psittacosis and ornithosis are the most accurate means of diagnosis but are successful in only five to 10% of cases. The entire psittacosis-LGV group share a common antigen, and an increasing concentration of CF antibody to this antigen during disease is the basis for serological diagnosis. In psittacosis characterized by atypical pneumonia and bird exposure, a fourfold rise in CF antibody titer to 1:64 or greater develops by the third week of the disease. The titer subsides slowly over several months. Antibiotic therapy may suppress or delay antibody response or hasten subsidence. Latent or chronic infection may develop, and chronic pulmonary infection of 8 years' duration has been documented by Meyer.

Subgroup A

Subgroup A patients present epidemiological, clinical, and serological evidence of psittacotic infection and complicating perimycarditis. Psittacosis perimycarditis has been described. Indeed, Levinthal discovered the inclusion bodies bearing his name in the pericardial fluid of infected birds and the disease in turkeys is predominantly a myocarditis. Two reports furnish histological or cultural confirmation of myocardial involvement in man by psittacosis infection. Jammach described psittacosis myocarditis in a human infant and demonstrated characteristic inclusion bodies in the heart, lungs, and liver identical with those demonstrated in a bird contact. Sheldon and associates isolated the
agent from a supraclavicular node in a man with pericarditis and systemic lymphogranuloma venereum.

**Subgroup B**

The validity of relating the acute perimyocarditis of these patients to infection by psittacosis family agents depends chiefly upon serological evidence. Corroborative epidemiological and clinical evidence of recent past or intercurrent psittacosis infection was lacking; but we consider it unlikely that these serological changes are fortuitous for the following reasons:

1. The serological response of subgroup B patients of the study group was similar to that of the 35 patients in the third control group.

2. The sera of 1,042 controls (1,000 apparently healthy persons and 42 patients hospitalized with nonviral noncardiac disorders) displayed no CF titer to psittacosis greater than 1:16; all subgroup B patients displayed higher titers.

3. Eleven of 12 specimens of serum from four patients, of study subgroup B (cases 4, 5, 6, 8) examined by the radioisotope precipitation (RIP) test had abnormally high levels of antibody. This test is a very sensitive, group-specific detector of antibody. It is two to 32 times more sensitive than CF, probably detects antibody qualitatively different from that detected by CF, and detects antibody in many sera negative by CF test. This may explain why one of 12 sera showed no correspondence between CF and RIP tests. Since the antigen employed in the RIP test is the Cal. 10 strain of meningopneumonitis agent cultivated on mouse fibroblasts, the problem of cross reactivity with hen's egg yoke was encountered in the CF test is eliminated.

Three additional factors contribute to the difficulties encountered in assessing the significance of rising CF antibody titers in the absence of bird exposure, typical clinical findings, or recovery of the agent. They are as follows:

1. **Enhancement of CF titer to psittacosis** antigen has been reported with other diseases such as brucellosis, Q fever, and hepatitis. Such antibody titers are usually low and subside rapidly.

2. Since two of these patients had primary myocardial disease and alcoholism, the antibody titers to routine psittacosis antigen might be attributed to an altered immune mechanism associated with liver disease. However, no patient had clinical or laboratory evidence of hepatic involvement.

3. Demonstration of a serological response to psittacosis agents does not necessarily indicate this agent to be causal in perimyocarditis despite coincident appearance. The agent may be merely a passenger and some other agent or factor may have caused the cardiac involvement. Thus, our current failure to demonstrate the agent in secretions, blood, pericardial fluid, or myocardium detracts from our thesis that psittacosis agent caused the heart disease in subgroup B patients. Yet, the failure to isolate the agent from biopsied myocardium or pericardial fluid, attempted in two patients, may well have been due to delay in obtaining specimens, and use of HeLa cell culture media.

In spite of these limitations, we believe psittacosis agent is responsible for heart disease in the nine patients described. Increasing knowledge has modified the necessary criteria for diagnosis. Bird contact is not essential. Kemmerer and associates have commented on the difficulty in obtaining admission of bird contact; since patients may be afraid of losing pets. Furthermore, this agent can be transmitted from man to man, and from domestic animals to man. Clinical manifestations are not always in the classic mold; psittacosis infection produces a spectrum of illness, subclinical to severe. Gerlach isolated the agent from five asymptomatic individuals and concluded that human psittacosis is not invariably accompanied by recognizable clinical signs and symptoms, that human beings are subject to subclinical latent psittacosis and are carriers, and that man-to-man infections occur frequently. Ward and associates described several asymptomatic
patients with high CF titers among families with other members who had respiratory illness and significant CF titers. Harding\textsuperscript{31} noted demonstrable antibody in 41\% of 275 parakeet owners with no history of acute febrile respiratory illness. Fraser and co-workers\textsuperscript{45} detected serological evidence of infection in 11\% of 2,175 naval recruits without bird contact; 41\% of these recruits had no history of respiratory illness. Thus, attention must be given to the possibility of chronic psittacosis infection, and to the presumption that a titer of 1:32 or higher sustained for 2 or more months indicates recent and continued activity of the agent. Such titers are not found in normal persons; such titers are associated with typical clinical cases, and persistent high titers occur with recrudescent LGV or with chronic pulmonary psittacosis.\textsuperscript{32}

Thus, prior observations and our experience suggest that human infection by psittacosis agents is rather frequent and produces a spectrum of disease. Subgroup A patients had acute perimyocarditis and typical epidemiological, clinical, and serological evidence of psittacosis. This group of patients is manifest evidence of the existence of psittacosis heart disease in this geographic area. The subgroup B patients may have even greater significance. Studies of these patients suggest that psittacosis infection may be causal in some forms of myocardial disease previously designated idiopathic.

Acknowledgment

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References


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