Necrotizing Sarcoid Granulomatosis*

Andrew Churg, M.D.; Charles B. Carrington, M.D.; and Raj Gupta, M.D., F.C.C.P.

Twelve cases of necrotizing sarcoid granulomatosis are presented as a retrospective study. The population of patients consisted of ten women and two men, with an average age of 50 years. Nine patients initially had a variety of pulmonary and nonpulmonary complaints, while three were asymptomatic. Chest roentgenograms demonstrated bilateral nodules in seven patients, solitary nodules in four, and a miliary pattern progressing to nodules in one. Enlarged hilar nodes were found in six. Histologically, all biopsies showed a combination of individual granulomas, confluent masses of granulomas which formed the nodular masses seen on the roentgenograms, a variable amount of necrosis of tissue and hyaline fibrosis, and a granulomatous vasculitis. Follow-up periods ranged from four months to 11 years. Eleven patients are alive and asymptomatic. The sole death occurred in a patient treated with an immunosuppressive agent (cyclophosphamide). We conclude that the clinical behavior of necrotizing sarcoid granulomatosis is not similar to that of the other angiocentric granulomatoses and that most patients with this disease can be left untreated or be treated with steroids alone. We suggest the possibility that necrotizing sarcoid granulomatosis may be the histologic counterpart of so-called nodular sarcoid.

In the 1973 J. Burns Amstrong lecture summarizing experience with pulmonary angiitis and granulomatosis, Liebow included a "provisional" description of 11 patients with a "condition" tentatively labeled "necrotizing sarcoid granulomatosis." The condition had the following three characteristics: (1) histologically, there was a background of sarcoid-like granulomata, a prominent and usually granulomatous vasculitis, and varying degrees of necrosis, the latter often superimposed upon a mass of confluent granulomas; (2) radiographically, there were usually pulmonary nodules but no enlarged hilar lymph nodes; and (3) clinically, the course was benign, even with minimal or no therapy. In particular, the prognosis, as judged from a three-year to 14-year followup of five patients, appeared to be far better than for patients with the other forms of angiocentric granulomatosis. Liebow speculated that necrotizing sarcoid granulomatosis might be yet another variant of angiocentric granulomatosis in which sarcoid-like features were unusually prominent or that it might instead be a variant of sarcoidosis. Recently, Saldana concluded from his experience with 24 cases that this was indeed a form of angiocentric granulomatosis. We report herein our experience with 12 cases.

Materials and Methods

A review of our files of consultations revealed 15 cases initially interpreted as necrotizing sarcoid granulomatosis because there were confluent masses of granulomas, necrosis of tissue within the granulomatous masses, and granulomatous vasculitis. Follow-up information, including radiographic findings, results of cultures, therapy, and course, were obtained from the referring physician. Whenever possible, the roentgenograms themselves were reviewed. Three of these cases were excluded because cultures of the specimens from biopsy yielded acid-fast bacilli or fungi; the remaining 12 comprise this report. Slides for all cases were stained with hematoxylin-eosin and for estain. At least one slide stained for acid-fast bacilli and one slide stained for fungi were available for review in ten of the cases.

Results

Clinical data are summarized in Table 1. Three of the 12 patients initially had no pulmonary signs or symptoms. Two of these were found to have abnormal chest roentgenograms during the course of routine medical examination. Pulmonary lesions were discovered in the third patient during evaluation for a pituitary adenoma. All except one patient had normal renal function; in the latter, a percutaneous renal biopsy revealed changes of chronic pyelonephritis but no glomerulonephritis or vasculitis. One patient also had diabetes insipidus, one patient had rheumatoid arthritis, and another had a high titer for rheumatoid factor without clinical evidence of rheumatoid arthritis.

Physical examination was usually unrevealing. Cutaneous tests for tuberculosis, histoplasmosis, and coccidioidomycosis were negative in all eight pa-

*From the Departments of Pathology, Stanford University, Stanford, Calif, and the University of California, San Francisco; and the Department of Medicine, Michael Reese Medical Center, Chicago. Supported in part by program project grant HL-19717 from the National Heart, Lung, and Blood Institute. Manuscript received October 6; revision accepted February 1. Reprint requests: Dr. Churg, Department of Pathology, University of California at San Francisco, San Francisco 94143.

406 CHURG, CARRINGTON, GUPTA

CHEST, 76: 4, OCTOBER, 1979
<table>
<thead>
<tr>
<th>Patient, Sex, Age (yr)</th>
<th>Enlarged Chest Roentgenogram*</th>
<th>Enlarged Hilary Lymph Nodes</th>
<th>Probable Clinical Diagnosis</th>
<th>Therapy</th>
<th>Status</th>
<th>Follow-Up Period, yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, F, 64</td>
<td>Cough; fever; anemia; renal insufficiency</td>
<td>Bilateral nodules (LML, LLL, RML, and RLL)</td>
<td>-</td>
<td>Wegener's granulomatosis</td>
<td>Roentgenogram cleared; asymptomatic</td>
<td>3</td>
</tr>
<tr>
<td>2, F, 59**</td>
<td>Dyspnea; fever</td>
<td>Bilateral nodules</td>
<td>-</td>
<td>Wegener's granulomatosis</td>
<td>Roentgenogram unchanged; asymptomatic</td>
<td>1.5</td>
</tr>
<tr>
<td>3, F, 62†</td>
<td>None</td>
<td>2-cm nodule (LLL)</td>
<td>-</td>
<td>Carcinoma</td>
<td>Wedge resection only</td>
<td>3</td>
</tr>
<tr>
<td>4, F, 65‡</td>
<td>Cough; dyspnea; pain in chest; cough</td>
<td>Multiple bilateral nodules (&lt;2 cm); increasing in size and number</td>
<td>+</td>
<td>Carcinoma; granulomatous disease</td>
<td>Roentgenogram unchanged</td>
<td>5</td>
</tr>
<tr>
<td>5, F, 55</td>
<td>Dyspnea; pain in chest; cough</td>
<td>Initially, LUL and RUL pneumonia infiltrates; partially resolved to show nodules</td>
<td>-</td>
<td>Unresolved pneumonia</td>
<td>Steroids</td>
<td>Roentgenogram cleared; asymptomatic</td>
</tr>
<tr>
<td>6, F, 23</td>
<td>Pleuritic pain (1 wk)</td>
<td>Multiple nodules (≤3 cm) (LUL, RUL, and RLL)</td>
<td>+</td>
<td>Steroids</td>
<td>Nodules shrinking; asymptomatic</td>
<td>0.5</td>
</tr>
<tr>
<td>7, F, 43</td>
<td>Hemothysis</td>
<td>Bilateral nodules</td>
<td>+</td>
<td>None</td>
<td>Nodules cleared over several weeks; asymptomatic</td>
<td>1</td>
</tr>
<tr>
<td>8, M, 60</td>
<td>Pain in chest; fever</td>
<td>Mass in RML for 4 yr before biopsy</td>
<td>-</td>
<td>None</td>
<td>Roentgenogram unchanged; asymptomatic</td>
<td>7</td>
</tr>
<tr>
<td>9, M, 68</td>
<td>None</td>
<td>Mass in LLL; upper lobe; RML for 4 yr before biopsy</td>
<td>-</td>
<td>Carcinoma</td>
<td>Lobectomy; then cyclophosphamide (Cytoxan)</td>
<td>Died of streptococal pneumonia 4 mo after lobectomy; no granulomatous lesions at autopsy</td>
</tr>
<tr>
<td>10, F, 45</td>
<td>Fever</td>
<td>Mass in RML</td>
<td>+</td>
<td>Brief antituberculosis therapy, then steroids</td>
<td>Roentgenogram unchanged; asymptomatic</td>
<td>11</td>
</tr>
<tr>
<td>11, F, 30‡</td>
<td>None</td>
<td>Miliary infiltrate (2 yr); slowly increased in size to 1-cm nodules</td>
<td>+</td>
<td>None</td>
<td>Roentgenogram unchanged 1 yr after biopsy; asymptomatic</td>
<td>3</td>
</tr>
<tr>
<td>12, F, 24</td>
<td>Cough; weakness; weight loss (3 wk)</td>
<td>Multiple bilateral nodules</td>
<td>+</td>
<td>Sarcoi; carcinoma</td>
<td>Steroids</td>
<td>Nodules cleared over 2 mo; asymptomatic</td>
</tr>
</tbody>
</table>

**LUL, left upper lobe; LML, left middle lobe; LLL, left lower lobe; RUL, right upper lobe; RML, right middle lobe; and RLL, right lower lobe.

**Also has rheumatoid arthritis.

†History of allergy to nitrofurantoin.

‡Also has chronic obstruction of airflow and positive test for rheumatoid arthritis.

§Antituberculosis chemotherapy was given after biopsy was performed.

|| Radiologically thought to be highly suspicious, but unequivocal reading of enlarged nodes could not be rendered because of presence of multiple parenchymal nodules.

††Pituitary adenoma.

Table 1—Data on Patients with Necrotizing Sarcoi Granulomatosis

CHEST, 76: 4, OCTOBER, 1979

NECROTIZING SARCOID GRANULOMATOSIS 407
patients tested. Control testing for anergy was mentioned in only one patient, who failed to react to streptokinase-streptodornase. Arterial blood gas levels were examined in only five patients. Four demonstrated moderate hypoxemia and hypocapnia, and one had normal values. The serum level of angiotensin-converting enzyme was measured in only one patient and only after spontaneous regression. At that time the value was normal.

Initial chest roentgenograms in ten patients revealed pulmonary nodules and in one patient revealed a miliary pattern, which later became nodular. In the 12th patient the x-ray films on admission had bilateral infiltrates thought to be pneumonia. During antibiotic therapy, these partially resolved, leaving nodular residua. The process was bilateral in eight patients and the lesions were distributed in all lobes (Fig 1). In the four patients who had unilateral disease, the nodules were solitary. Sizes ranged from less than 1 to 4 cm. No cavities were seen. Unequivocal bilateral enlargement of the hilar lymph nodes was present in four patients and was strongly suspected by the reviewing radiologist in two others. In four patients the pulmonary nodules were followed radiographically for 1.5 to 4 years before biopsy. In three of these (patients 1, 8, and 10), the nodules slowly increased in size and number. In patient 11, both the hilar lymph nodes and the nodular infiltrates (which had increased up to 1 cm at the time of biopsy) became progressively more prominent over a period of two years. Prior to thoracotomy, a needle biopsy of the liver demonstrated caseating granulomas without visible organisms in case 7, and a transbronchial biopsy revealed noncaseating granulomas in case 8.

The tissue resected at thoracotomy consisted of one specimen from lobectomy, one wedge resection, and ten biopsies. Cultures of the resected tissue were negative in all 12 cases. Grossly, the nodules were generally not sharply defined. They were 0.1 to 4.5 cm in diameter and gray-white to yellow, and they often appeared necrotic. In the specimens from two patients (patients 3 and 9) with radiographically solitary nodules, only the single nodules were found. In six other cases, including one with a radiographically solitary nodule, multiple nodules measuring 1 to 5 mm were observed, in addition to those visible on roentgenograms. Thus, in most cases, the total number of nodules was considerably greater than seen radiographically.

Histologically, all specimens had granulomas, necrosis, and angiitis. Within the nodules, the pulmonary parenchyma was completely effaced by confluent granulomas, which were not sharply demarcated from the surrounding pulmonary tissue but tended to blend into an infiltrate of lymphocytes and histiocytes and individual granulomas slightly separated from the main mass (Fig 2). The individual granulomas were composed of giant cells and epithelioid histiocytes and were interspersed to a variable degree with chronic inflammatory cells and

**Figure 1.** Multiple bilateral pulmonary nodules and probably enlarged hilar lymph nodes are evident on this roentgenogram. Patient was treated with steroids, and nodules disappeared over several months (case 12).

**Figure 2.** In this view of outer rim of large nodular lesion, there is extensive central necrosis (at lower right) surrounded by dense band of confluent granulomas. This was most frequently observed pattern of necrosis. Note individual granulomas (at top) (case 6; hematoxylin-eosin, original magnification x 90).
pneumonic foci. CHEST, 76: 4, OCTOBER, 1979

60
Some vascular structures were entirely replaced by necrosis. In
other instances, only a portion of the wall of a vessel was
involved. In these vessels, the granulomas were transmural,
but granulomas confined to the adventitia and outer media or
to the intima were not infrequent. In most vessels, the granulomas
were found only within a portion of the circumference; but
occasional vessels cut longitudinally showed multiple sites
of involvement. In most instances the granulomas in the
vessels formed discrete masses similar to the granulomas in
the remaining parenchyma, but in a few vessels a more diffuse
proliferation of giant cells and epithelioid histiocytes was
found. This pattern of granulomatous vasculitis was found
both in large confluent masses and frequently also away from
the nodular masses. A second type of vascular lesion was
usually found only within confluent nodules of granulomas (Fig
5). This concerned small vessels and was characterized more
by compression of the vessels by surrounding granulomas than
by infiltration of the wall of the vessel itself. Elastic stains

proliferating fibroblasts (Fig 3). In most cases, numerous
individual foreign-body giant cells were also scattered through
the nodular masses. Occa-

sional giant cells contained asteroid bodies. An inter-

stitial infiltrate of lymphocytes and histiocytes was also present
around the larger masses. In all cases in

which it was possible to examine tissue away from
the main lesion, smaller masses of confluent gran-

ulomas and often individual granulomas were
observed. These small masses and individual granu-

lomas tended to occur along lymphatic routes, i.e.,
along the interlobular septa and bronchovascular
bundles. Occasional granulomas were found in
bronchial walls, and rarely they eroded bronchial
and bronchiolar mucosa.

The extent of necrosis in the granulomatous
masses varied considerably from case to case. In
some instances, the entire central portion of a nodule
was destroyed, leaving only a rim of viable cells
(Fig 2). In other instances, there were single or
multiple small punctate foci of necrosis. Usually,
there was no residual structure in the necrotic de-
bris, although elastic stains sometimes demonstrated
vascular remnants. In two cases the central portions
of the masses were largely hyalinized, and only small
foci of necrosis remained. Necrosis was sometimes
present within the smaller confluent masses of

granulomas but was rarely seen in the individual
granulomas.

A prominent arteritis and phlebitis occurred in
three different histologic patterns. The most
common was characterized by destruction of a por-
tion of the wall of a large vessel by intramural
granulomas (Fig 4). Usually, these granulomas
were transmural, but granulomas confined to the
adventitia and outer media or to the intima were not
infrequent. In most vessels, the granulomas involved
only a portion of the circumference; but occasional
vessels cut longitudinally showed multiple sites
of involvement. In most instances the granulomas in
the vessels formed discrete masses similar to the
granulomas in the remaining parenchyma, but in a
few vessels a more diffuse proliferation of giant cells
and epithelioid histiocytes was found. This pattern
of granulomatous vasculitis was found both in large
confluent masses and frequently also away from
the nodular masses. A second type of vascular lesion was
usually found only within confluent nodules of granulomas (Fig 5). This concerned small vessels and was characterized more by compression of the vessels by surrounding granulomas than by infiltration of the wall of the vessel itself. Elastic stains...
revealed the wall of the vessel clearly distorted by the granuloma while retaining its normal elastic structure; however, sometimes, the elastic stain revealed destruction of the wall of the vessel, and occasionally, true granulomas protruded into the vessel’s lumen. Many of these small vessels were largely occluded by intimal fibrosis. This type of vasculitis was more frequent in nodules where the confluent granulomas were not separated by a prominent inflammatory and fibroblastic proliferation. The third type of angiitis involving both large and small arteries and veins was seen only within the large nodular masses, almost always near or within foci of necrosis (Fig 6). In this pattern the wall of the vessel was destroyed by an infiltrate of lymphocytes, plasma cells, and histiocytes. Occasionally, giant cells were also present but not discrete granulomas.

Transpleural extension of the granulomatous process was apparent in cases 3 and 10. In the latter, hilar lymph nodes were also involved by direct extension. An additional biopsy of a hilar node was performed only in case 9, and this node was unremarkable.

Eleven of the 12 patients, treated or not, are alive and asymptomatic six months to 11 years after initial examination (Table 1). The 12th patient died from infection following therapy with cyclophosphamide (Cytoxan). In seven patients the chest roentgenogram cleared (in two by surgical removal of the only visible lesion), and in five patients the radiographic lesions persisted essentially unchanged. As noted in Table 1, two patients briefly received therapy with antituberculosis drugs after the biopsy. Inasmuch as cultures of all biopsies were sterile, this treatment was thought to be irrelevant.

Five patients received no treatment after surgery. Radiographic infiltrates persisted unchanged in three, vanished in one, and have not reappeared after surgical removal of a solitary nodule in one. In the four patients treated with steroids alone, the nodules shrank or disappeared in three and persisted unchanged in one. In the two surviving patients treated with cyclophosphamide (Cytoxan) or cyclophosphamide plus steroids, the infiltrates vanished in one and persisted unchanged in one. The only fatality was an asymptomatic man whose solitary lesion was entirely removed surgically. He was then treated with cyclophosphamide (Cytoxan), and he died four months later of streptococcal pneumonia. At autopsy, there were no granulomatous lesions or renal lesions.
DISCUSSION

The disease in these 12 patients appears both clinically and histologically similar to that originally described in 11 patients by Liebow, although some differences do exist between the two series. In both series the majority of patients initially had nonspecific symptoms often suggesting infection, but a few patients were asymptomatic. Chest roentgenograms usually demonstrated multiple bilateral nodules without preferential location. Six of our patients had enlarged hilar lymph nodes in addition to the parenchymal infiltrates, whereas only one patient had transient enlargement of the hilar nodes in Liebow's original group. Both of these series are at variance with that described by Saldana et al., two-thirds of his patients were asymptomatic, and the lesions were localized, usually in the upper lobe, in 88 percent (21 patients).

Histologically, the findings in our series and in Liebow's original group were very similar, since the combination of multiple granulomas, necrosis of tissue, and vasculitis are the features which define the lesion. All of our patients had large nodular masses composed of confluent granulomas, and necrosis was usually confined to the confluent masses. Since Liebow speculated that necrotizing sarcoid granulomatosis might be either a variant of angiocentric granulomatosis or a variant of sarcoidosis, it is interesting that in our cases the number and distribution of epithelioid-cell granulomas, the hyalization of granulomas, and the predominant type of vasculitis were strongly suggestive of sarcoidosis. The extent of vasculitis and necrosis were more reminiscent of the angiocentric granulomatoses. There were always numerous and confluent sarcoid-type granulomas in the parenchyma, as in sarcoidosis itself; whereas such granulomas are absent or sparse and never confluent in the angiocentric granulomatoses. When adequate tissue was available for examination, the distribution of the small masses and individual granulomas was noted to follow lymphatic pathways along the bronchovascular bundles and interlobular septa, just as in sarcoidosis. This is in contrast to both forms of Wegener's granulomatosis and lymphomatoid granulomatosis, in which the individual lesions, although angiocentric, tend to be randomly distributed, rather than in the string-of-pearls so typical of sarcoidosis. In several of the biopsies, there were hyalized regions. This feature was also noted by Liebow, and hyalization is also common in sarcoidosis. Of particular interest is the fact that in two of our cases with extensive hyalization, nodules have persisted for 2 and 11 years, the latter in spite of steroid therapy. The major types of angiitis originally described were found. Vasculitis characterized by intramural, sarcoid-like epithelioid-cell granulomas was common in our cases but is rarely seen in the angiocentric granulomatoses. Such angiitis is also common in ordinary sarcoidosis. However, in sarcoidosis the angiitis is usually less severe, and it is less extensive than in necrotizing sarcoid granulomatosis and the angiocentric granulomatoses by an order of magnitude. Vasculitis characterized by a heavy and diffuse cellular infiltrate without discrete granulomas was relatively infrequent and was associated with foci of necrosis in the large nodular masses. In contrast, in the classic and limited forms of Wegener's granulomatosis, this form of vasculitis predominates and is frequently seen also in isolated foci well away from the necrotic masses. The extent of necrosis, of course, is also an outstanding difference between ordinary sarcoidosis and necrotizing sarcoid granulomatosis; the pathologic definition of sarcoidosis usually includes the adjective, nonnecrotizing or noncaseating. The necrosis encountered in some otherwise typical cases of sarcoidosis usually consists of minute foci containing a few necrotic cells or granular cosinophilic debris. The extent of necrosis seen in the angiocentric granulomatoses is similar to that of necrotizing sarcoid granulomatosis.

More important than the histologic analysis is the fact that in terms of clinical disease and prognosis, the patients with necrotizing sarcoid granulomatosis are much more like patients with ordinary sarcoidosis than patients with the other angiocentric granulomatoses. Eleven of our 12 patients with necrotizing sarcoid granulomatosis have survived for six months to 11 years (average, 3.5 years), and Liebow noted similarly long survivals. All of the survivors are essentially asymptomatic, and in most the pulmonary disease has either stabilized or regressed, either without therapy or with steroids alone. The role of immunosuppressive agents in treating this disease is questionable, and the only death in our series was caused by bacterial infection in a patient treated with cyclophosphamide. In contrast, the five-year survival in allergic angiitis and granulomatosis treated with steroids and immunosuppressive agents is 50 percent. In a large series of patients with lymphomatoid granulomatosis, the five-year survival was only 20 to 30 percent with steroid or immunosuppressive therapy. Patients with true Wegener's granulomatosis are usually quite ill and have either upper respiratory or renal involvement; the disease in patients with limited Wegener's granulomatosis may also be dramatic and include painful, necrotizing cutaneous lesions. Most patients with either form respond well to immunosuppressive therapy, but their survival without therapy or with
steroids alone is poor. The recently described entity of benign lymphocytic angiitis and granulomatosis\(^{10,11}\) is somewhat similar to necrotizing sarcoid granulomatosis in that lesions may have granulomas and the course is fairly benign; however, there is no granulomatous angiitis, and a good clinical response requires immunosuppressive therapy.

Because of the histologic features and course, we suggest that necrotizing sarcoid granulomatosis is most likely a variant of sarcoidosis or at least closely related. A clear understanding of this relationship may have to await a better understanding of sarcoidosis itself. In the interim, it is worth noting that patients with necrotizing sarcoid granulomatosis have not yet been tested with Kveim’s antigen. The only measurement of the serum level of angiotensin-converting enzyme was made during remission, so the normal value is not helpful.\(^{12}\) It is also interesting to speculate that necrotizing sarcoid granulomatosis, which has been defined histologically, may be fundamentally the same or similar to “nodular sarcoid,” which has been defined clinically. In large series, 2 to 4 percent of the patients are ultimately considered to have sarcoid present with nodules radiographically, frequently in the absence of enlarged hilar lymph nodes.\(^{13}\) The course of nodular sarcoid has not been well studied, and the histologic appearance and exact relation to ordinary sarcoid are uncertain. Sharma et al\(^{14}\) and Onal et al\(^{15}\) described 11 such patients, five of whom had enlarged hilar nodes and three of whom had Kveim tests (all positive). A minority demonstrated arthralgias and uveitis. None of them was tested for angiotensin-converting enzyme. Biopsies from several patients showed confluent masses of granulomas, and one biopsy showed necrotizing granulomas. The radiographic nodules cleared spontaneously in three of the patients and disappeared during steroid therapy in three others. In the remaining five, the nodules persisted with or without steroid therapy for up to five years. Of particular interest are two recent reports of cavitation in nodular sarcoid.\(^{16,17}\) Although necrosis of tissue was not described, the cavitation implied that necrosis had occurred. Biopsy at the proper time and site might document necrosis more frequently. In our biopsies of necrotizing sarcoid granulomatosis, the necrosis ranged from massive to that present in only a portion of one of many sections.

No matter how one tentatively classifies these cases, our experience indicates that it is important for prognostic and therapeutic reasons to separate necrotizing sarcoid granulomatosis from the other types of angiocentric granulomatosis. It should also be remembered that infection is the primary diagnosis to consider when necrotizing granulomas are encountered, and appropriate stains and cultures must be performed in every case. Three cases which were appropriate for this series from histologic, clinical, and radiographic criteria were excluded because culture of the specimen from biopsy proved an infectious cause.

**Addendum**

Since this manuscript was completed, we have had occasion to review lung biopsies on another nine patients with necrotizing sarcoid granulomatosis. All had pulmonary lesions and six had clinical features similar to those described above. However, three patients had extrapulmonary involvement resembling that seen in ordinary sarcoidosis: two patients had uveitis (in one uveitis was the presenting complaint, and the pulmonary lesions appeared several years later); and one patient developed hypothalamic insufficiency several years after biopsy of her lung nodule. One of the patients with uveitis also had non-caseating granulomas in her hilar lymph nodes. These findings also strengthen our belief that necrotizing sarcoid granulomatosis is a variant of sarcoidosis.

**Acknowledgments:** We thank the following physicians, who gave us the privilege of seeing slides and roentgenograms on these cases and also provided clinical and follow-up data: case 1, A. Haghighat, Bridgeport, Conn; case 2, H. Battifora, Chicago; case 3, J. Ottis, Atlanta; case 4, R. Redding and J. Cunningham, Pawtucket, Rl; case 5, J. Christman, E. Sermier, and A. Nabi, Schenectady, NY; case 6, W. Burger, Tallahassee, Fl; and case 7, W. Recant, Chicago; case 8, B. Panburg, Boston: case 9, D. Troxel, Concord, Calif; and case 10, G. Baer, Brockton, Mass. We also thank Norman Blank, M.D., of Stanford University, Stanford, Calif, for help in interpretation of the chest roentgenograms.

**References**


412 CHURG, CARRINGTON, GUPTA

CHEST, 76: 4, OCTOBER, 1979

Supercourse V

The Supercourse V postgraduate program on lung disease will be held November 27-December 1 at the Hyatt Regency Hotel, New Orleans, sponsored by the American Lung Association of Louisiana, Inc. and its medical section. The program consists of the 16th Annual Pulmonary Function in Health and Disease Course, the 12th Annual Respiratory Disease Course, and the 9th Annual Pediatric Pulmonary Course. The three programs run concurrently. Program information is available from Dr. John B. Bobear, American Lung Association of Louisiana, Suite 500, 333 St. Charles Avenue, New Orleans 70130.

International Congress on Respiratory Diseases

The International Congress on Respiratory Diseases will be held in Algarve, Portugal, December 5-7, under sponsorship of the Sociedade Portuguesa de Patologia Respiratoria. Official languages will be Portuguese, English and French with simultaneous translation. For information, write the Sociedade at Rua de S. Sebastiao de Pedreira 82-1, 1000 Lisbon, Portugal.

Echocardiography Courses

The Division of Diagnostic Ultrasound of Thomas Jefferson University Hospital, Philadelphia, will present a course on Basic Echocardiography, November 26-30, and a course, Advanced Echocardiography, December 3-7. For information, contact Ms. Lynn Lindengrass, Education Secretary, Division of Diagnostic Ultrasound, 1015 Walnut Street, Philadelphia 19107.