For a number of years, pulmonary involvement has been known to occur frequently in systemic lupus erythematosus and occasionally in rheumatoid arthritis. Although numerous clinical and pathologic descriptions of these conditions exist, information concerning their pulmonary pathophysiology has been scarce. This report presents 13 cases of rheumatoid disease and eight of systemic lupus erythematosus associated with pulmonary complications with special emphasis on abnormalities of pulmonary function.

Inasmuch as these two conditions may resemble each other clinically, especially in their early phases, it seems appropriate to consider the altered pulmonary function of both in the same report.

**Material and Methods**

The 13 patients with rheumatoid arthritis and the eight with lupus erythematosus, seen between 1950 and 1961, had pulmonary function studies because of abnormalities on roentgenograms of the thorax.

Of the 13 patients with rheumatoid arthritis, six were classified as having classic and seven as definite rheumatoid arthritis according to the revised (1958) diagnostic criteria of the American Rheumatism Association. On examination, all 13 had typical articular deformities, and nine had characteristic changes on roentgenograms of the joints. The flocculation test for the rheumatoid factor was reactive for the five patients tested. The ages of the 13 patients ranged from 45 to 69 years, and eight were men.

In the eight patients with lupus erythematosus, the diagnosis was made on the basis of: (1) a positive L. E. clot test in the seven patients so tested, and (2) a characteristic clinical course. All eight patients scored high enough to justify the diagnosis when the clinical criteria of Winslow and associates were applied. The ages ranged from 40 to 59 years, and the group included six men and two women.

Thus, we classified each of the patients into one of the two groups, although we realized that there are many intermediate forms in the continuum of collagen disease. A serologic overlap existed in that four of the 13 patients with rheumatoid arthritis had a positive result with the L. E. clot test. Patient 9 (Table 1), who had a positive L. E. clot test and rheumatoid factor, had a biopsy of the lung that showed interstitial fibrosis and lymphocytic infiltration and a biopsy of a subcutaneous nodule from the elbow that showed rheumatoid granulomatous changes. Patient 4, who also had positive tests for the L. E. cell and the rheumatoid factor, had a biopsy of synovial membrane that showed rheumatoid villous synovitis. In patient 13, who had a positive L. E. clot test, a flocculation test for rheumatoid factor was not done; but he had characteristic subcutaneous rheumatoid nodules.

The difficulties in serologic differentiation of these two conditions have been pointed out by others. Although we were arbitrary in establishing the two groups, we include both groups in one series because of the potential serologic overlap.

An attempt was made in every case to exclude other causes of the pulmonary dysfunction.
### Pulmonary Function Data for Rheumatoid Arthritis and SLE

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>X-ray Findings</th>
<th>Resp. (min.)</th>
<th>Vent.</th>
<th>VC</th>
<th>RV</th>
<th>TC</th>
<th>RV/TC Ratio</th>
<th>MBC</th>
<th>N. Index</th>
<th>% Rest</th>
<th>Exercise</th>
<th>Do</th>
<th>MMF</th>
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<tr>
<td>1</td>
<td>48</td>
<td>F</td>
<td>Mild, diffuse, hazy density with small scattered nodules</td>
<td>17</td>
<td>8.5</td>
<td>94</td>
<td>96</td>
<td>87</td>
<td>30</td>
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<td>5</td>
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<td>2</td>
<td>64</td>
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<td>Mild, reticular density in lower 5% of both lung fields</td>
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<td>7.8</td>
<td>71</td>
<td>13</td>
<td>50</td>
<td>40</td>
<td>82</td>
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<td>95</td>
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<td>1.82</td>
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<td>3</td>
<td>58</td>
<td>M</td>
<td>Mild, reticular, nodular density in both upper lung fields</td>
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<td>12</td>
<td>85</td>
<td>160</td>
<td>105</td>
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<td>4</td>
<td>54</td>
<td>M</td>
<td>Moderate, diffuse haziness in both lower lung fields</td>
<td>14</td>
<td>16</td>
<td>87</td>
<td>175</td>
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<td>40</td>
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<td>2.5</td>
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<td>—</td>
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<tr>
<td>5</td>
<td>53</td>
<td>F</td>
<td>Mild, reticular, haziness in both lower lung fields</td>
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<td>7.9</td>
<td>76</td>
<td>85</td>
<td>78</td>
<td>30</td>
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<td>95</td>
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<td>6</td>
<td>61</td>
<td>M</td>
<td>Moderate, diffuse, mottled haziness with nodularity</td>
<td>11</td>
<td>10</td>
<td>54</td>
<td>93</td>
<td>64</td>
<td>30</td>
<td>74</td>
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<td>55</td>
<td>F</td>
<td>Mild, basal-lateral haziness, greater on left</td>
<td>21</td>
<td>8.6</td>
<td>61</td>
<td>89</td>
<td>68</td>
<td>34</td>
<td>108</td>
<td>0.7</td>
<td>96</td>
<td>—</td>
<td>1.51</td>
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<td>8</td>
<td>53</td>
<td>M</td>
<td>Moderate, diffuse reticulonodular density with bullous emphysema of both upper lungs</td>
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<td>11</td>
<td>70</td>
<td>148</td>
<td>90</td>
<td>42</td>
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<td>1.7</td>
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<td>4.00</td>
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<tr>
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<td>47</td>
<td>M</td>
<td>Severe haziness with superimposed honey-combing</td>
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<td>17</td>
<td>64</td>
<td>110</td>
<td>70</td>
<td>36</td>
<td>124</td>
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<td>92</td>
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<td>6.87</td>
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<tr>
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<td>69</td>
<td>M</td>
<td>Severe, diffuse honey-combing</td>
<td>1959</td>
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<td>17</td>
<td>67</td>
<td>128</td>
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<td>39</td>
<td>76</td>
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<td>95</td>
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<td>45</td>
<td>F</td>
<td>Severe, diffuse, mottled haziness with small bilateral effusions</td>
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<td>9.6</td>
<td>39</td>
<td>119</td>
<td>60</td>
<td>53</td>
<td>59</td>
<td>1.2</td>
<td>96</td>
<td>94</td>
<td>(1.0)</td>
<td>2.50</td>
</tr>
<tr>
<td>12</td>
<td>59</td>
<td>F</td>
<td>Mild, basal, diffuse haziness</td>
<td>16</td>
<td>9.2</td>
<td>44</td>
<td>185</td>
<td>86</td>
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<td>69</td>
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<td>0.67</td>
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<td>M</td>
<td>Mild, diffuse, reticular density greater in lower half of each lung</td>
<td>16</td>
<td>9.6</td>
<td>83</td>
<td>166</td>
<td>104</td>
<td>40</td>
<td>78</td>
<td>3.0</td>
<td>95</td>
<td>92</td>
<td>(1.2)</td>
<td>1.25</td>
</tr>
</tbody>
</table>

**Rheumatoid Group**

**Lupus Group**

*Abbreviations: Resp.: respirations/minute; Vent.: ventilation (L/min.); VC: vital capacity (per cent of normal); RV: residual volume (per cent of normal); TC: total capacity (per cent of normal); MBC: maximum breathing capacity (per cent of normal); SaO₂: per cent saturation of hemoglobin with oxygen in arterial blood; Diff: diffusing capacity for carbon monoxide (ml./min./mm.Hg); and MMF: maximal midexpiratory flow (L/sec.).

†Normal is less than 2.5 per cent.
changes. Eighteen biopsies were performed in 11 cases. Examination of sputum for malignant cells and cultures of sputum, gastric contents, and tissues for tubercle bacilli and fungi were carried out in most cases.

Studies of respiratory function consisted of determinations of vital capacity, total lung capacity, residual volume with our modification of the open-circuit technic of Darling, discussed by Miller and colleagues,1 maximal midexpiratory flow,1 maximum breathing capacity,1 and arterial oxygen saturation recorded by an ear oximeter2 during rest and during exercise (9-inch step, 15 cycles per minute). The pulmonary diffusing capacity for carbon monoxide was measured during exercise by a modification of a steady state method ("DcoIV") of Bates, Boucot and Dormer.8 Values for rate of carbon monoxide uptake and alveolar (end-tidal) tension were measured during the third minute of breathing 0.1 per cent carbon monoxide in air and of a standard exercise (9-inch step, 15 cycles per minute). Normal standards are based on studies done in our laboratory.10

RESULTS

Thirteen patients in the series showed a restrictive defect with diminished vital and total capacities and normal or only moderately decreased maximal breathing capacity (Table 2). Twelve patients demonstrated arterial oxygen saturation below 94 per cent: two while at rest and ten only with exercise. Four patients with rheumatoid arthritis were unable to exercise in the laboratory because of pain and deformity of the extremity. The carbon monoxide diffusing capacity was quite low for the three hypoxic patients so studied. Only four patients showed obstructive features, and all of these had associated major restrictive changes. Two of the four had a history of asthmatic bronchitis. The results of the pulmonary function studies of three patients (No. 2, 3 and 4, Table 1) were considered normal in spite of an increased residual volume since this change is often seen in older age groups. Each of the three had diffuse reticular densities on the roentgenogram of the thorax. Less than one third of the series had hyperventilation at rest.

The abnormalities on the roentgenogram of the thorax were quite variable and included both parenchymal and pleural changes (Table 1). The parenchymal abnormalities included increased peripheral markings with or without honeycombing and nodule formation, and the pleural involvement included thickening or effusion or both. Some patients showed both parenchymal and pleural lesions. The abnormalities were less extensive or less generalized in the four patients whose pulmonary volumes were in the predicted normal range than in those with definite defects of function.

Eleven patients had biopsy. In most of these, the findings were nonspecific and were useful only in eliminating other pathologic conditions. Four patients (No. 6, 9, 10 and 18) had lung biopsy. In patient 6, this showed marked dilatation of peripheral bronchi and nonspecific chronic inflammatory changes in the walls. In patient 9, the biopsy revealed marked interstitial edema, fibrosis with honeycombing, and focal lymphocytic infiltration. The biopsy findings in patients 10 and 18 are included in the illustrative case reports. Biopsies of scalene lymph nodes showed nonspecific inflammatory changes. A subcutaneous nodule showed granulomatous rheumatoid changes (patient 9). Synovial biopsy showed changes consistent with rheumatoid arthritis (patients 1 and 4) and moderate subacute synovitis (patient 14). Pleural and pericardial biopsies showed nonspecific inflammatory changes (patients 12 and 14).

All patients in the rheumatoid group had significant articular symptoms. In 11 of the 13, the arthritis preceded the pulmonary symptoms by two months to 45 years. In the two other patients, the onset of articular symptoms followed the pulmo-
nary symptoms by one and one-half and two years. All of the patients with abnormal pulmonary function had respiratory symptoms. Eleven complained of cough, which usually was nonproductive. Nine had dyspnea on exertion. Five had chest pain, which was pleuritic in four.

All eight patients with lupus erythematosus had febrile episodes, dyspnea on exertion, and pain in the chest. Four had cough, seven had articular symptoms, and two had a rash.

Eighteen of the 21 patients in the series had pulmonary abnormalities on physical examination; these consisted usually of basilar rales, but some patients with primarily pleural abnormalities had decreased breath sounds and diminished chest expansion.

Four patients with rheumatoid disease were treated with corticosteroids because of the pulmonary involvement. Patient 9 was treated for six months; this resulted in an increase in vital capacity of 33 per cent, in total lung capacity of 17 per cent, and in exercise period from 0.8 to 3.6 minutes with an arterial oxygen saturation of 84 per cent. The diffusion capacity for carbon monoxide increased from 6 to 10 ml./min./mm.Hg (23 to 35 ml. is normal). While receiving 1.5 mg. of dexamethasone daily, patient 12 showed no objective improvement in function despite a subjective decrease in pulmonary symptoms and patient 10 had worsening of pulmonary function and increased x-ray evidence of chest disease. Patient 11 was treated for two years, with resulting symptomatic improvement and some improvement in the appearance of the roentgenogram; the measurements of pulmonary function were not repeated.

REPORT OF ILLUSTRATIVE CASES

CASE 10

A 69-year-old cattle salesman was first seen at the Mayo Clinic in July, 1959; rheumatoid arthritis was diagnosed. He recalled having had an attack of polyarthritis involving most of his joints in 1914 when he was 24 years old which confined him to bed for three months. He had noted a moderate cough with sputum in 1952, and by 1958 the cough had become worse and mild dyspnea on exertion became apparent. He had smoked cigars moderately for many years. His arthritis remained quiescent from 1914 until three months before he was seen in 1959. The flare-up involved his hands and wrists, and the patient had responded promptly to corticosteroid

![Figure 1: (Case 10) Thorax showing disseminated reticular density with a honeycombed appearance.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21410/)
therapy. He described a mild, nonspecific pain in the right anterior portion of the chest.

Examination revealed rather typical, but mild, rheumatoid involvement of the hands, wrists, ankles, and knees, but no subcutaneous nodules. Clubbing of the fingers was present. Scattered, crackling rales were heard throughout both lungs.

Hemoglobin concentration was 13.2 gm. per 100 ml. of blood. The leukocytes numbered 9700 per cubic millimeter, and the erythrocyte sedimentation rate was 94 mm. in one hour (Westergren method). There was mild proteinuria. The serologic test (VDRL) for syphilis was not reactive, and L. E. clot test was negative. Repeated smears and cultures of the sputum revealed no fungi or tubercle bacilli. The roentgenogram of the thorax showed a disseminated reticular density with a honeycombed appearance involving both lungs (Fig. 1). The roentgenographic appearance of the hands and wrists was normal. Pulmonary function studies revealed a restrictive defect with a decrease of arterial oxygen saturation to 90 per cent after 1.2 minutes of exercise. A biopsy of a scalene node showed inflammatory reaction, and a biopsy of the lung showed marked interstitial fibrosis associated with foci of organizing pneumonitis and pronounced thickening of the walls of the blood vessels with an organizing thrombus in one small artery (Fig. 2). Cultures of the lymph node and the lung for tubercle bacilli, fungi, and Brucella organisms revealed no growth.

The patient continued to take 0.75 mg. of dexamethasone twice daily until he returned two years later because of progressive dyspnea. Symptoms involving the joints remained quiescent. The roentgenogram of the thorax showed more pulmonary involvement. The flocculation test for the rheumatoid factor was reactive, and the result of the L. E. clot test remained negative. The pulmonary volume had decreased further, and the arterial oxygen saturation decreased to 85 per cent after 0.5 minute of exercise. The carbon monoxide diffusing capacity was 3.3 ml./min./mm. Hg (normal >15).

Case 18

A 40-year-old rancher from Wyoming registered at the Mayo Clinic in August, 1961. He had been in excellent health until May, 1961, when he noted sudden pain involving all peripheral joints. Two weeks later, left lateral pleuritic pain and severe dyspnea appeared on exertion. He had a low-grade fever and lost 15 pounds during the next two months. During this time, roentgenograms of the thorax revealed progressive opacification of the midportions of both lungs.

Physical examination revealed a cyanotic, chronically ill man with moist rales in the lower half of each lung. The joints were normal.

The hemoglobin concentration was 16.2 gm., and the leukocyte count was 5800 per cubic millimeter with a normal differential leukocyte count. The erythrocyte sedimentation rate was 35 mm. in one hour (Westergren method). The urine was normal. The serologic test for syphilis was not reactive. The results of the L. E. clot test were positive on four occasions. The flocculation test for the rheumatoid factor was nonreactive. The serum protein electrophoretic fractions were normal, except for an albumin value of 2.82 gm. per 100 ml. Roentgenograms of the thorax showed bilateral, diffuse, patchy infiltration, which was more evident in the central

Figure 2: (Case 10) Specimen from lung showing marked interstitial fibrosis and foci of organizing pneumonitis. There is pronounced thickening of the walls of the blood vessels with an organizing thrombus in one small artery (hematoxylin and eosin; x27).
PULMONARY DYSFUNCTION IN RHEUMATOID ARTHRITIS AND SLE

Since Ellman and Ball first described widespread pulmonary lesions in rheumatoid arthritis, many reports of such cases have appeared in the literature. These authors aptly suggested the term "rheumatoid disease" because of the widespread nature of this mesodermal condition, and since the respiratory tract contains considerable amounts of connective tissue, one would logically expect pleural and pulmonary involvement. Although Aronoff and associates expressed doubt that such an entity as rheumatoid disease of the lung existed, most authors agree that both specific necrobiotic pulmonary inflammatory lesions and, more commonly, nonspecific ones may be associated with rheumatoid arthritis.

Only a few authors have referred to pulmonary function studies in rheumatoid and lupus erythematosus pulmonary disease. Andersen and Moersch reported briefly on two patients with rheumatoid arthritis, one having a restrictive defect and the other an obstructive change with arterial desaturation with exercise. The same au-

slight clearing. On dismissal, the patient was following a schedule of gradually decreasing doses of corticosteroids to be continued under medical supervision.

**COMMENT**

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Diseases of the Chest

Newcomer, Miller, Hepper and Carter

...tors commented on a lupus erythematosus patient showing decreased volumes and expiratory slowing. Brinkman and Chai... presented a case of rheumatoid arthritis involving decreased vital capacity and arterial oxygen saturation. Ognibene... reported on two patients with rheumatoid disease with restrictive defects, one of whom had severe cor pulmonale. Morgan,9 Cud-......lowicz and associates,9 and Doctor and Snider10 reported single cases of rheumatoid disease with brief mention of pulmon-......ary function. The last two reports included data suggesting a diffusion defect.

The two groups in the present study showed similar changes in pulmonary function. The differences were only in degree with the abnormalities among cases of lupus erythematosus being more pronounced. In individual patients, no consistent relationship was found between the severity of restrictive changes and the degree of hypoxemia. Thus, in some instances greatly reduced vital capacity was associated with normal saturation, while in others, moderately reduced vital capacity was associated with definite hypoxemia.

The changes in lung volumes can be explained readily by restriction following pleural effusion, pleural thickening, thickening and rigidity of the costoarticular par-enchymal structures, or any combination of these. That the medium and major airways are largely spared is documented by the only occasional and, even then, mild reduction in estimates of maximal expiratory flow. These mild reductions may be attributable to muscular weakness.

Hypoxemia probably resulted both from thickening of considerable portions of the diffusing surface by interstitial fibrosis and from edema. Additional factors contributing to hypoxemia could include reduction in diffusing surface area by nodules and limitation of ventilation-perfusion adjustment with exercise.

The pathologic changes in rheumatoid lung disease that produce these changes in function are both specific and nonspecific. Rubin11 found that the most frequent abnormality in rheumatoid disease of the lung was a pleuritis appearing as a small serous effusion. Rickards and Barrett,12 in reviewing the literature on pathologic changes in rheumatoid lungs, found reports on 11 patients showing nonspecific fibrosing pneumonitis and 26 patients showing necrobiotic foci of the rheumatoid type, the majority of this latter group having associated pneumoconiosis. The typical rheumatoid nodule contains a core of hyalinized collagen that is surrounded by inflammatory cells and fibroblasts which may show a palisade arrangement.

The biopsy of the lung for three patients in the rheumatoid group showed only nonspecific fibrosing pneumonitis and no typical rheumatoid nodules. The walls of the blood vessels in patient 9 showed marked thickening. This particular process may become so severe that pulmonary hypertension accompanied by oblitative disease of the small pulmonary arteries and arterioles may occur.11

Baggenstoss14 observed that when distinctive lesions are found in systemic lupus erythematosus, they are most commonly in the kidneys or heart, and that no pathologic lesions are found in the lungs. Purnell and co-workers15 reviewed 54 cases of lupus erythematosus at necropsy and found that the pulmonary changes included terminal bronchopneumonia, hemorrhage, pleural effusions, pulmonary edema, inflammatory exudates within alveolar walls, acute fibrinous pleuritis, plural fibrosis, and thickening, in the order of decreasing frequency.

We were interested in determining whether therapy could reverse the observed changes in function. Reports in the literature indicate that there has been a variable response to corticosteroid therapy for rheumatoid disease of the lung; also in our four patients so treated, the results were variable. Although subjective improvement was noted in three of four patients, only one had an increase in lung volume and some improvement in diffusion.
The dangers of undesirable side effects of corticosteroid administration, particularly in the patients with rheumatoid arthritis, cannot be overemphasized. Such therapy should be undertaken only when the pulmonary symptoms demand or when it is evident that the progression of the pulmonary disease will lead to significant impairment of function.

**Summary**

Pulmonary function was studied in 13 cases of rheumatoid disease and eight of systemic lupus erythematosus associated with roentgenographic evidence of pulmonary involvement. The most common abnormality noted was a reduction of lung volumes associated frequently with arterial oxygen unsaturation. These changes possibly were due to pleural effusion, restrictive pleuritis, parenchymal involvement, or any combination of these. Corticosteroid administration was attended by variable results.

**Resumen**

Se estudió la función pulmonar en 13 casos de artritis reumatoide y en ocho de lupus eritematoso generalizado, en los que había evidencia radiográfica de compromiso pulmonar.

La anormalidad más común fue la reducción de los volúmenes del pulmón a los que se asoció frecuentemente falta de saturación arterial de oxígeno. Estos cambios posiblemente se debieron a derrame pleural, pleuritis restrictiva o retráctil, invasión del parénquima pulmonar o las combinaciones de estas condiciones. La administración de corticosteroides fue seguida de resultados diversos.

**Résumé**

La fonction pulmonaire a été étudiée dans 13 cas d’affections rhumatismales et 8 cas de lupus érythémateux disséminé associé à la preuve radiographique d’atteinte pulmonaire. L’anomalie notée le plus communément fut la réduction des volumes pulmonaires fréquemment associée à la désaturation oxygénée artérielle. Ces altérations sont imputables à l’épanchement pleural, à l’atteinte parenchymateuse ou à toute association de ces altérations. L’administration de corticoïdes fut suivie de résultats variables.

**Zusammenfassung**


**References**


For reprints, please write Section of Publications, Mayo Clinic, Rochester, Minnesota 55901.

ESOPHAGEAL OBSTRUCTION

It would seem that endoscopically inserted intraluminal tubes are a useful adjunct to the other techniques available for the management of esophageal carcinoma. It is possible in this relatively simple and safe manner to provide a patient passage for food and to eliminate regurgitation. Such tubes are useful for lesions at any level and of any length. The Soutar tube can be used in conjunction with x-ray therapy. Most of our patients with lesions considered incurable by virtue of local extension were treated with radiation following insertion of the tube. Since the tube can become obstructed by particles of solid food, it is important to maintain the patient entirely on liquids and soft foods following insertion.


RESULTS OF DOMICILIARY TREATMENT IN AN URBAN TUBERCULOSIS CLINIC

In an urban clinic with average facilities, 78.4 per cent of the patients can be made noninfective in 12 months. Of the total cases, 71.6 per cent can be made to take regular treatment, the treatment being made to continue for at least 12 months in 77 per cent of the cases.

Theoretically speaking, 98.2 per cent of patients can be converted in 12 months, if with improved supervision all patients are made to take treatment with regularity for at least 12 months. Looking at the cost involved in providing a larger supervisory staff and realizing some weakness of human nature, one may for the present be content with the above figures, regularity and adequacy in a community as a whole, though we should aim at 100 per cent results, especially in individual cases. Conversion rates increase with the increase in the standard of regularity of treatment. To achieve conversation rate at 80 per cent or above, regularity in medicine administration should be at least of the order of 80 per cent. It means body condones irregularity of the order of 20 per cent to the maximum.


HEART BLOCK

Data on 61 children with complete heart block are presented. In at least half these patients, it may be assumed, with certainty, that the conduction disturbance was congenital in origin. In only seven are we certain that the complete heart block developed after birth. Approximately 50 per cent of the patients surely had associated congenital heart disease, and 16 per cent certainly did not. The atrial and the ventricular rates both decreased significantly with age. The duration of the QRS complexes of seven patients, three of them now dead, was 0.10 second or longer. The increase in ventricular rate averaged just below 20 per cent after exercise and atropine whereas isoproterenol caused an average increase in ventricular rate of about 50 per cent.