gas composition. It is not practical or necessary, however, to obtain blood gases on all cases of respiratory disease referred to the board. It is a recommended practice for those cases which seem to have more functional impairment than can be ascribed on a mechanical basis. By assessing both the subjective effects of respiratory disease with effects that we can measure precisely, the degree of impairment can be separated into classes of impairment to driving.

Although the advisory board will not be directly involved in tests of respiratory function, certain background comments may be useful in interpretation of their results.

Tests of ventilatory function are not infallible, as there are areas of human performance that can affect the results. The tests require maximal voluntary effort on the part of the patient, who may be unable or reluctant to perform the tests as well as his true respiratory capacity would allow. For this reason, the tests should be repeated if a significant impairment is noted. A bronchodilator should be administered if the cause of the respiratory deficiency is suspected to be bronchial obstruction. If there is a 15 percent improvement in subsequent tests after this treatment, these values must be considered to be the true state of the individual's respiratory capacity.

The tests most likely to give a survey of the driver's ventilatory capacity are the one-second, forced expiratory volume (FEV₁₀), the forced vital capacity (VC), and the determination of the maximal voluntary ventilation (MVV). Most test subjects can easily understand the performance of these tests after a short explanation and encouragement to participate actively. Results of these tests should be expressed in terms of liters or liters per minute and also as a percentage of the predicted normal. The FEV₁₀ and FVC should each be administered three times, with the best test result determined as most representative of the patient's capacity. The MVV is a fatiguing test, requiring considerable muscular effort and for this reason the better of two attempts should be accepted. Impairment to driving caused by ventilatory deficiency may be grouped as follows:

**Group A**—Chest x-ray films are usually normal, but may show healed or inactive disease of the chest. Dyspnea, if it occurs, is consistent with the type and degree of physical exertion. Values obtained from at least two of the ventilatory function tests are no less than 85 percent of predicted normal values for patient's age, sex, and height. Blood gases are usually within the normal range.

**Group B**—Chest x-ray films are normal or abnormal. Dyspnea does not occur at rest and usually does not occur during the performance of usual daily activities. The subject can keep a normal pace with persons of his same age and body build on level ground without breathlessness, but not on hills or stairs. Values obtained from at least two ventilatory function tests are in the range of 70 to 85 percent of the predicted normal values. Blood gases usually are normal but the oxygen partial pressure present on a random sample of arterial blood may be diminished to 75 mm Hg.*

**Group C**—Chest x-ray films may be normal, but usually are not. Dyspnea does not occur at rest, but is present during performance of usual daily activities. The individual can walk one mile at his own pace without dyspnea, but is unable to keep up with his peers. Values of at least two ventilatory function tests are in the range of 55 to 70 percent of the predicted normal values. The blood gases are usually abnormal with the partial pressure of arterial oxygen no less than 70 mm Hg.

**Group D**—Chest x-ray films are usually abnormal. Dyspnea occurs climbing one flight of stairs, walking 100 yards on the level, or even at rest. Values obtained from at least two ventilatory function tests are below 55 percent of the predicted normal value. The partial pressure of arterial oxygen is less than 65 mm Hg.

* Numerical values may differ among laboratories and it should be noted that the following values are based on a lower limit of normal of 85 mm Hg.

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**Pulmonary Alveolar Proteinosis**

**Treatment of a Case Complicated by Tuberculosis**

S. Robert Latham, Jr., M.D.; Jay D. Williams, Jr., M.D.; Ross L. McLean, M.D.; and Jose Ramirez-R., M.D.

Observations over a ten-year period of a patient with pulmonary alveolar proteinosis complicated by pulmonary tuberculosis are presented. The mycobacterial infection responded well to chemotherapy, but the alveolar proteinosis rapidly progressed to the extent that the patient required continuous oxygen therapy. The progress of the disorder was not halted by six weeks of segmental irrigation with heparin and acetylcysteine. Dramatic restitution of a normal arterial oxygen saturation and complete resolution of acinar infiltrates was induced by a single washing of the lungs. The remission has persisted for four and one-half years.

The association of alveolar proteinosis and pulmonary tuberculosis has been well documented only once.*

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In that case tuberculosis and proteinosis remitted following pulmonary lavage despite inadequate antituberculosis chemotherapy. Because the alveolar material in patients with alveolar proteinosis supports the growth of mycobacteria, there is some question whether patients in whom these two diseases co-exist can be effectively treated for tuberculosis without adding the immediate risk of dissemination through pulmonary lavage.

This case demonstrates that pulmonary tuberculosis may come under antimicrobial control despite the presence of extensive and progressive alveolar proteinosis. It also illustrates that a complete remission may be rapidly induced in this disorder by a single washing of the lungs.

**CASE REPORT**

A 41-year-old Negro woman was admitted to Grady Memorial Hospital in September, 1961. In the preceding three years she had experienced four acute episodes of chills, fever and productive cough associated with pneumonic infiltrates. Clinical symptoms improved promptly with antibiotic therapy, and productive cough associated with pneumonic infiltrates. Five months after initiating antituberculosis therapy, (August, 1964), segmental "flooding" of the right lung three times daily was initiated. The patient received 100 ml of saline containing 50 to 100 units per ml of heparin and 1 percent acetylcysteine. Weekly chest films showed no resolution. At the end of six weeks of therapy, the oxygen tensions had improved only slightly. The patient was discharged from the hospital, but remained housebound.

On January 5, 1965, she was admitted in intense respiratory distress. The Po2 was 30 mm Hg (Table 1), and continuous oxygen therapy was required. After two weeks of hospitalization the patient was sent home, where she remained bedridden while receiving oxygen.

On March 11, 1965, she was readmitted for pulmonary lavaging by the method then available. Severe hypoxemia persisted (Table 1). Extensive bilateral pulmonary infiltrates had remained unchanged for the preceding three months (Fig 1A). The first pulmonary lavage was performed on March 17, 1965. The left lung was filled with 900 ml of normal saline containing 2 percent acetylcysteine and 75 mg of heparin. In the 24 hours following the lavage she expectorated 500 ml of heavy, cream-colored sputum. Six days after the lavage, extensive resolution of the infiltrates in the left lung was demonstrated. Eight days after the lavage, she was less hypopneic and required oxygen only intermittently while at rest.

The right lung was filled with 1600 ml of a similar solution on April 13, 1965. A chest film 24 hours after the lavage showed partial resolution of the right pulmonary infiltrates. Two weeks after the second lavage, the patient's sense of well being returned. She began to gain weight. After one year of incapacitating dyspnea she became fully ambulatory. During her convalescence she refused further arterial punctures. Six weeks after the second lavage there was extensive resolution of all pulmonary infiltrates. The oxygen saturation reached normal limits for the first time in six years of observation (Table 1). Nine weeks after the lavage, the pulmonary infiltrates had resolved completely (Fig 1B). When last seen in December, 1969, the patient had no positive showing 10 mm of induration. Two direct smears of the sputum showed acid-fast bacilli. Culture of the sputum subsequently grew Mycobacterium tuberculosis.

After one month of treatment with isoniazid, PAS and streptomycin, her sputum no longer harbored tubercle bacilli, but her dyspnea persisted. Five months after initiating antituberculosis therapy, (August, 1964), segmental "flooding" of the right lung three times daily was initiated. The patient received 100 ml of saline containing 50 to 100 units per ml of heparin and 1 percent acetylcysteine. Weekly chest films showed no resolution. At the end of six weeks of therapy, the oxygen tensions had improved only slightly. The patient was discharged from the hospital, but remained housebound.

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alveolar proteinosis continued to progress. In the only other case where the co-existence of these two diseases is well documented an organism resistant to drug therapy emerged one year after antimicrobial therapy was initiated. Consequently, it seems important that broncho-alveolar debridement be carried out soon after the tuberculosis infection is controlled.

The success of bronchopulmonary lavage after failure of segmental irrigation has been noted previously. In other cases of alveolar proteinosis intensive or repeated lavaging has been required to induce a remission. In this case, the therapeutic effectiveness of a single lavage of each lung is clearly demonstrated by rapid remission now lasting for four and one-half years.

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Pulmonary Cryptococcosis—A Case Diagnostically Confirmed by Transbronchial Brush Biopsy*

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This case demonstrates the value of transbronchial brush biopsy in the differential diagnosis of pulmonary lesions. Suspected pulmonary cryptococcosis in a 60-year-old woman was confirmed by using a technique of transbronchial brush biopsy. Examination of an immediate India ink preparation demonstrated the organism, Cryptococcus neoformans.

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