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only 10 to 20 percent of normal antitrypsin concentration or activity to the serum. The heterozygous state (MM<sub>Duarte</sub>) has the same range of values for serum trypsin inhibitory capacity as the MZ phenotype (mean ± SD for MM<sub>Duarte</sub>, 0.73 ± 0.05 unit; n = 6). There are no procedures, other than perhaps liver biopsy, capable of distinguishing between the null variant and the MM<sub>Duarte</sub> variant. The null variant does not produce intrapulmonary globules,<sup>15,16</sup> whereas the MM<sub>Duarte</sub> and Z variants do. Diethylstilbestrol provocation or knowledge of the patient’s use of such medication can also be utilized to reveal the true antitrypsin state, since response to the estrogen would be limited (serum trypsin inhibitory capacity, less than 1.2 units in our laboratory). Four of the subjects in this familial study were receiving estrogens, and three were apparently antitrypsin-deficient, having values for serum trypsin inhibitory capacity of 0.92, 0.84, and 0.85 unit while receiving the medication. The third woman had a normal high level of 1.65 units while receiving estrogens.

This familial study is an example of how antitrypsin phenotyping by acid-starch electrophoresis alone may not reveal the existence of an inherited deficiency state; however, quantitative measurements of serum trypsin inhibitory capacity did reveal an intermediate deficiency. The proband was a smoker with alterations of pulmonary function suggestive of early pulmonary emphysema. The association of emphysema with an M phenotype has not been reported previously. Although a single case does not establish the existence of a significant relationship between the null gene and emphysema, this topic is worthy of further study.

REFERENCES

15. Seboue R, Martin JP, Rapozt C: The serum of a Pi<sup>−</sup>-subject contains a small quantity of α<sub>1</sub>-antitrypsin. Presented at the α<sub>1</sub>-Antitrypsin and Pi System Symposium, Rouen, Belgium, 1974

Unusual Electrocardiographic Changes in Spontaneous Pneumothorax

Paul Kuritzky, M.D.** and Allen L. Goldfarb, M.D., F.C.C.P.†

A young woman with spontaneous left pneumothorax had a phasic voltage alternation of her electrocardiogram that resolved with expansion of the lung. Likely explanations for this phenomenon are a respiratory dependent change in cardiac anatomy and a change in the volume conductor with respiration.

CASE REPORT

A 26-year-old woman was admitted to Millard Fillmore Hospital after sudden onset of stabbing infraclavicular pain radiating to the left axilla and arm and accompanied by severe dyspnea. Five weeks prior to admission, she had a laparotomy with removal of a right ovarian cyst. Findings from physical examination and a chest roentgenogram at the time revealed no pulmonary abnormalities. The patient

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smoked ten cigarettes per day.

The patient was thin, tachypneic, and in moderate distress. Her blood pressure was 116/88 mm Hg, and her pulse was 120 beats per minute. Auscultation of the lungs disclosed markedly diminished breath sounds over the left lung. Findings from the remainder of the physical examination were normal. A chest roentgenogram showed a left pneumothorax with complete collapse of the left lung. The results of routine laboratory studies were normal.

The electrocardiogram (Fig 1) showed sinus tachycardia and a marked phasic voltage alternation accompanied by shift of the P, QRS, and T axes. The phasic voltage alternation was seen in all leads and was best demonstrated in lead 2, where the QRS amplitude gradually shifted from a low of 5 mm to a high of 13 mm. The frontal QRS axis changed from $+75^\circ$ to $+90^\circ$ as the voltage diminished. Similarly, the QRS precordial transition zone shifted from leads V3 and V4 to V5 with the diminution in voltage.

A chest tube was inserted and connected to an underwater seal, and by the second day of hospitalization the left lung had expanded. A repeat ECG at that time (Fig 2) showed sinus tachycardia, increased precordial QRS amplitude, and low T waves in leads 2, 3, and V4 to V6. The phasic voltage alternation was gone. On the third day the chest tube was removed, and the pneumothorax recurred immediately. An ECG was not repeated at this time. The chest tube was reinserted; and following reexpansion of the left lung, the patient was discharged on the sixth day of hospitalization.

Seven days after discharge, the patient was again admitted with a spontaneous pneumothorax. A chest roentgenogram revealed a 50 percent-collapse of the left lung. An ECG (Fig 3) showed sinus tachycardia, an RSR' pattern in leads V1 to V3, and marked diminution in precordial QRS voltage with the QRS amplitude in leads V4 to V6, being less than 5 mm. The patient was again treated with a chest tube connected to an underwater seal. The left lung did not fully reexpand with conservative treatment, and a thoracotomy was performed on the seventh day of hospitalization, with resection of emphysematous blebs and pleurodesis. Five days after surgery, the patient's ECG was normal.
DISCUSSION

Electrocardiographic changes in spontaneous left pneumothorax have recently been reviewed, and their importance in the differential diagnosis of acute chest pain has been stressed by Walston and associates. They studied 7 patients with spontaneous left pneumothorax and surveyed the literature concerning the ECG in pneumothorax, finding the following four relatively consistent electrocardiographic changes: (1) a rightward shift in the mean frontal QRS axis; (2) diminution in precordial R voltage; (3) diminution in QRS amplitude; and (4) inversion of precordial T waves.

To our knowledge, the phasic voltage alternation seen on the ECG during our patient’s original pneumothorax has not previously been described. We speculate that these voltage changes are an accentuation of respiratory effects on the ECG and are due to two basic mechanisms. The first is a respiratory dependent change in cardiac anatomy, and the second is a change in the volume conductor with respiration.

A large pneumothorax might well cause a large mediastinal shift with each respiration. This shift could produce changes in cardiac position, changes in intracardiac anatomy with partial obstruction to flow, and changes in the relation of right- to left-sided cardiac output. Any combination of these could be responsible for the phasic voltage variation. The voltage alternation of total electrical alternans appears similar to the phasic voltage variation in the present case. There is good evidence that total electrical alternans is produced by anatomic oscillations in heart position, and this has been demonstrated by echocardiographic and cineangiographic studies.

The concept of an enlarged retrosternal air mass affecting the volume conductor was advanced by Littmann. He studied 2 patients with spontaneous pneumothorax and mediastinal emphysema. With a tilting table, he demonstrated that when his patients were supine, they had T-wave inversion and low R voltage in leads CFv and CFt (comparable to unipolar precordial leads) and on chest roentgenogram had a large retrosternal air space. When the patients were upright, the ECG was normal, and the retrosternal air space was much smaller. Similar changes in the retrosternal air mass and, thus, in electrical conductivity might have occurred with each respiration in our patient, giving rise to the voltage changes seen.

The explanation of the phasic electrocardiographic voltage variation is not clear and must await its reobservation with collection of appropriate hemodynamic and anatomic (roentgenographic or echocardiographic) data. Nevertheless, we believe that this finding may be useful as an electrocardiographic sign of pneumothorax.

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REFERENCES


Endobronchial Tuberculosis
Progressing to Bronchial Stenosis*

Fiberoptic Bronchoscopic Manifestations

Richard K. Albert, M.D., and Thomas L. Petty, M.D., F.C.C.P.

Severe stenosis of main-stem and segmental bronchi occurred in a 79-year-old woman with previously and adequately treated endobronchial tuberculosis. Symptoms and physiologic abnormalities did not occur until more than three years after diagnosis and initiation of therapy.

Tuberculous infection of the tracheobronchial tree generally resolves as the primary disease becomes controlled; however, when inflammation extends more deeply into the bronchial mucosa, causing ulceration and necrosis, healing can occur with fibrosis and result in bronchial stenosis.

A patient with endobronchial tuberculosis and no evidence of parenchymal involvement was diagnosed using the fiberoptic bronchoscope. She initially responded well to treatment but during follow-up developed cough and dyspnea. Repeated bronchoscopic examination demonstrated marked endobronchial stenosis.

CASE REPORT

A 79-year-old white woman was admitted for evaluation of increasing cough, dyspnea, and weakness. With the exception of diet-controlled adult onset diabetes mellitus and essential hypertension, the patient had been healthy. She had never smoked cigarettes. Her cough began in 1969, and, despite consultation of multiple physicians, went undiagnosed until August 1972, when fiberoptic bronchoscopic examination revealed endobronchial tuberculosis. While discrete ulcerations were not visible, the bronchial mucosa was diffusely erythematous and covered with minute whitish plaques. Smears and cultures of sputum were positive for Mycobacterium tuberculosis, and no parenchymal focus could be found, despite extensive evaluation.

The patient was treated with 300 mg of isoniazid (INH), 1,600 mg (25 mg/kg of body weight) of ethambutol, and 600

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