breath holding for durations similar to that of the activity, and subsequently they complained of dyspnea similar to that following the activity.

**Discussion**

This study shows that during some common activities, there appears to be shallow, irregular breathing involving both chest and diaphragmatic movement, and this is followed by a period of increased ventilation. It is possible that during the activity, there is some carbon dioxide retention and possibly a fall in arterial oxygen tension. Hyperventilation after the activity is needed to return the blood gases towards the previous resting level. This increased breathing following the activity is appreciated as dyspnea in patients with obstructive lung disease. In normal subjects, compensation for the shallow, irregular breathing is achieved by a few deep breaths which are easily performed without subjective discomfort.

Patients become fearful when they find themselves short of breath after such minor exertion as doing their hair, and explanation that this is due to a breathing pattern, which they can personally observe, can do much to reassure them and eliminate unnecessary fear of their disease.

**S. Tangri, M.D. and C. R. Woolf, M.D., F.C.C.P.*
Toronto, Ontario**

*Associate Professor, Department of Medicine, University of Toronto.

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The Electrocardiogram as a Diagnostic Aid in Pneumothorax

**To the Editor:**

The electrocardiographic changes of spontaneous pneumothorax are not widely appreciated, although they may be of considerable diagnostic aid. Careful attention to the electrocardiogram, which is usually readily available in the emergency room, office or on the hospital ward, may provide rapid confirmation of a diagnosis of pneumothorax. Physical examination may be unreliable, especially in patients with obstructive lung disease. Even the chest roentgenogram may be misleading—if the patient has visible blebs or bullae—or may be technically unsatisfactory resulting in an undesirable delay in treatment.

**Case 1**

A 37-year-old white man who smoked suddenly developed left anterior chest pain which began while he was at rest. When he was first seen six hours later the pain radiated through to the back and was associated with dyspnea and lightheadedness. He had no previous pulmonary symptoms and worked as an orthopedic appliance fitter. Physical examination: blood pressure-120/70 mm Hg, respiration-28 and pulse-96 per minute. Vocal and tactile fremitus were decreased, the percussion note was increased and breath sounds were absent over the left hemithorax. The heart tones were markedly diminished. A 50 percent collapse of the left lung was seen on the chest roentgenogram. The electrocardiogram (Fig 1-upper) showed poor progression of R wave amplitude across the left precordial leads with a decrease in the amplitude of the QRS complexes in all precordial leads. Following insertion of a chest tube and reexpansion of the lung, the electrocardiogram showed recovery of the R wave amplitude and normal progression of the QRS complexes (Fig 1-lower).

**Case 2**

A 67-year-old black man was hospitalized because of the rapid onset of dyspnea and a tight sensation in the right chest. His history included 20 years' employment as an underground hard coal miner, 50 pack years of cigarette smoking and pulmonary tuberculosis. Physical examination:

**Figure 1.** Upper tracing: ECG during partial lung collapse. Lower tracing: immediately following decompression.
he was cyanotic, blood pressure-90/60 mm Hg, respiratory rate-40 and pulse-150 per minute. He appeared to be in marked respiratory distress. The breath sounds were absent and resonance was increased over the right hemithorax. A third heart sound was audible. The chest roentgenogram revealed a 30 percent right-sided pneumothorax and bilateral fibronodular infiltrates. The electrocardiogram showed marked right axis deviation, some increase in the amplitude of the R waves in the right precordial leads and tall, peaked P waves in leads 2, 3 and aVF. A chest tube was inserted and the lung re-expanded with prompt clinical improvement. Thereafter, his electrocardiogram showed an abrupt change in axis, a decrease in the QRS complex duration and resolution of both the P-pulmonale and incomplete right bundle branch block pattern.

The severity of the clinical picture in the presence of only moderate lung collapse plus prompt improvement following decompression of the right hemithorax suggest that this patient had a marked loss of pulmonary reserve. Otherwise, he would have tolerated this modest degree of lung collapse with minimal symptoms.

**Comment**

The first electrocardiogram of case 1 shows a loss of voltage in the left precordial leads. This is typical of left side pneumothorax and is thought to be due to both a change in heart position and to interference with transmission of cardiac potentials to the chest wall by the air interposed between heart muscle and electrode.

Copeland and Omenn reported a patient whose electrocardiogram mimicked anterior lateral wall myocardial infarction with absent R waves in the right precordial leads and a loss of R wave amplitude in the left precordial leads. When the ECG was taken in the sitting position the R wave amplitudes returned to near normal. The change of position presumably eliminated much of the air that gravitated between the sternum and heart when the patient was recumbent.

In Case 2, the P wave configuration and marked right axis shift seen in the initial electrocardiogram and their rapid resolution following decompression of the thorax suggest acute cor pulmonale. Although we have no direct measurements of cardiac function, he undoubtedly had acute ventricular dilatation.

An awareness of the changes in the electrocardiogram which accompany spontaneous pneumothorax is helpful in alerting the physician to this diagnostic possibility. Although the electrocardiographic changes of pneumothorax are rather characteristic, they often go unrecognized.

**Roland S. Summers, M.D.**

*Chicago*

**References**

1. Master AM: The electrocardiographic changes in pneumothorax in which the heart has been rotated: The similarity of some of these changes to those indicating myocardial involvement. Am Heart J 3:472, 1928


**Dilemmas in Drug Nomenclature**

_To the Editor:_

I have compiled a list of drugs whose names look alike or sound alike. When a pharmacist takes a prescription over the telephone or attempts to decipher a physician's handwriting, a drug product not intended by the prescriber might be dispensed. Such an error might be the result of a sound-alike or look-alike drug.

The following is a list of drugs used in relieving vasodilation, mucosal edema and bronchospasm, such drugs having striking similarities. Physicians are urged to exercise great care when telephoning or writing prescriptions.

- **Asminyl**—**Esimil**
- **Asminyl**—**Asmolin**
- **Brondecon**—**Bronkotabs**
- **Isuprel**—**Isomel**
- **Isuprel**—**Isordil**
- **Isuprel**—**Ilomel**
- **Isuprel**—**Ismelin**
- **Marax**—**Maalox**
- **Marax**—**Atarax**
- **Mudrane**—**Modane**
- **Myambutol**—**Nembutal**
- **Pas-C**—**Pasca**
- **Quadrinal**—**Quatrasal**
- **Rifadin**—**Ritalin**
- **Sudafed**—**Sudolin**
- **Tedral**—**Teldrin**

*Benjamin Teplitsky*

*Brooklyn*

*Resident in Internal Medicine, Chicago Wesley Memorial Hospital.*

*Chief, Pharmacy Service, Veterans Administration Hospital.*

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