A 58-year-old man complained of substernal distress lasting from 10 to 20 minutes. The attacks recurred on several occasions during the past three months. There were periods during which he was free of pain. When the attacks did seize him, however, they were often, but not always, related to effort.

Physical examination showed marked obesity. Heart sounds were normal. There was left ventricular enlargement on fluoroscopic examination of the heart. Blood pressure levels were normal. Cholesterol and enzyme studies were within normal limits. The clinical impression was that the patient had coronary artery disease.

Repeated ECG studies were unequivocally normal. A carefully planned exercise test was considered to be justified. Again, the resting ECG was normal. After 11 ascents and descents the patient complained of substernal pain. The test was immediately stopped.

The sequence of ECG findings were as follows: immediately after the test, sharp S-T depressions were found in leads I, II, and most impressively in V2, V4 and V5. Three minutes later, the injury pattern was still present, but in the chest leads it was combined with T changes. Of considerable interest, moreover, was the appearance of bigeminal rhythm in the extremity leads.
A long strip makes this change even more conspicuous than the few cycles which can be shown in this presentation.

Ten minutes after the exercise, S-T segments have returned to normal and the T inversion remained as the only abnormality. Even in a long strip, ectopic beats were completely absent at this time. Fifteen minutes after the test, the ECG was again entirely normal.

This report does not pretend to present anything that is not commonly known. The clear demonstration of the sequence of events, however, was felt to be of interest: normal ECG, test, injury pattern first without rhythm disturbance, then with it, followed by ischemia pattern before return to normal. Premature systoles with and without coupling have been reported in previous Electrocardiogram of the Month by Elek.

The results of this test were suggestive of being of more than diagnostic value. It was found that this patient developed frequent premature contractions when myocardial injury occurred. Scherf has often stressed the point that it takes just one ectopic beat to set up a tachycardia; if it is of ventricular origin, then ventricular fibrillation may ensue. There was coupling already evident in this case. Further and more prolonged myocardial injury, therefore, might easily lead to irreversible conditions.

This patient is now on long-term anticoagulant therapy. Only one aspect of preventive treatment is dealt with by this measure. Since the changes developed after only a short exercise test, an attempt to suppress an irritable focus with quinidine appears justified, if not imperative.

———

RELATION OF SERUM PROTEIN LEVELS AND HYALINE MEMBRANE DISEASE

In the series of 54 treated infants and 119 untreated comparable control infants all with birth weights under 2500 mg., the following results were obtained: (1) serum protein levels are related to infant mortality, but no more closely than is birth weight of the patient; (2) the use of concentrated salt-poor human albumin in the first hour of life does not affect neonatal mortality or the incidence of hyaline membrane disease; (3) albumin given thus will cause a transient rise in serum protein levels and a more prolonged rise in total body protein levels; (4) the serum protein level of the cord blood of an infant bears no prognostic significance in relation to hyaline membrane development in the premature baby.


———

REGULATION OF PULMONARY VASCULAR BED

Pulmonary blood volume was estimated by means of rapidly successive injections of an indicator into the pulmonary artery and left atrium with sampling of blood from a systemic artery. In 40 patients with mitral valve disease and in four patients with mild aortic valve disease, this volume was found to range from 132 to 455 ml./M$^2$ with a mean of 263 ml./M$^2$.

Exercise, rapid intravenous infusion or methoxamine were found to cause passive pulmonary vaso-dilation manifested by a concordant rise in both pulmonary blood volume and intravascular distending pressure. Hexamethonium or vasovagal reactions induced passive narrowing, reflected by simultaneous reduction in both pulmonary blood volume and distending pressure. During acute hypoxia or angiotensin infusion, active constriction of the pulmonary vascular bed was evidenced by an increase in distending pressure, but a decrease or no change in the pulmonary blood volume. During acetylcholine infusion, active dilation of the pulmonary vascular bed was reflected by a decrease in distending pressure.