The Human Electrocardiogram in Anaphylactic Shock*
Report of a Case

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Allergic reactions may profoundly affect the cardiovascular system. Widespread vasculitis often occurs and extensive focal degeneration of heart muscle has long been recognized.1,2 Both in experimental animals and in humans, varying electrocardiographic changes in antigen-antibody reactions have been described.3-4 Electrocardiographic observations of near fatal anaphylactic reactions in the human are sparse.

In anaphylactic shock, the most dread of all allergic manifestations, widespread histamine release results in marked bronchospasm with attendant hypoxia, systemic

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3-13-64
5:40 p.m. Standard and Goldberger leads

Strips of Lead II

5:45 p.m. Full ECG

FIGURE 1

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hypotension, widespread focal capillary damage, change in cellular permeability, and exudation. Each of these factors contributes to the electrocardiographic alterations observed. In the face of this catastrophe, powerful cardio-tonic drugs are utilized and further modifications may occur.

In non-fatal animal experiments, changes are usually limited to the ST-T segments with transient ST segment shifts and reversal of T wave polarity. Fatal experiments frequently demonstrate varying grades of heart block.5

Microscopic review of heart muscle reveals focal myocardial necrosis in the subendocardial layers.3

CASE REPORT
S. D., a 36-year-old housewife, was seen by her family physician complaining of: increased nerv-
ous tension, hot flashes, menstrual irregularity. Previous medical history revealed known allergies to wool and tomatoes. There was no history of asthma; however, chronic rhinitis had been a long-standing problem. Two years before, she had experienced an acute allergic reaction manifested by bronchospasm and subsequent urticaria following an injection of penicillin in oil.

Prompt response to administration of epinephrine and penicillinase injectable (Neutrapen) followed by ACTH resulted. After physical examination disclosed no abnormality, a diagnosis of menopausal syndrome was entertained and 20,000 units estrogenic substance in sesame oil was injected intramuscularly by an office nurse. One minute after injection, she complained of

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**Figure 3**

- 3-15-64
- 3-18-64
breathlessness and apprehension. She immediately collapsed and blood pressure was unobtainable. Marked over-distension of the lungs was noted and totally ineffective respirations characterized by marked prolongation of the expiration phase were observed. Intravenous epinephrine and dexamethasone (Decadron) were immediately administered and nasal oxygen initiated. She was transported to the hospital by ambulance arriving approximately 15 minutes after her collapse. In the emergency room, blood pressure was 55/45, heart tones were inaudible, and the pulse at the wrist was barely obtainable. Cyanosis was marked. Breathing sounds were coarse with extreme prolongation of expiration. Positive pressure oxygen, hydrocortisone 21-sodium succinate (Solucortef), metaraminol (Aramine), and lidocaine (Xylocaine) were administered during the next four hours. Continuous support with Aramine and Solucortef was necessary for 16 hours. These drugs were slowly withdrawn and the patient made an uneventful recovery. At no time did friction rub appear. A third sound gallop was audible throughout the second day.

Serial electrocardiograms are reproduced.

**DISCUSSION**

In non-fatal animal experiments, electrocardiographic changes in anaphylaxis may be evanescent. This human case demonstrates evidence of myocardial damage extending over a period of several days. Myocardial cell injury is attested to by the appearance of slowly evolving ST-T changes resulting from a current of injury and marked lengthening of the corrected QT duration.

Evidence of selective chamber enlargements appears related to the profound bronchospasm with hypoxia, and increased right ventricular work.

Rate and rhythm changes may reflect the response both to hypotension and vasopressor administration.

**REFERENCES**


