THE MECHANISM OF THE ELECTROCARDIOGRAPHIC DISORDERS IN ANAPHYLACTIC SHOCK

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The essential participation of the sympathetic nervous system in the pathogenesis of anaphylactic shock has been proved by the work of several authors [15 and others].


In the present investigation we studied the part played by the sympathetic nervous system in the changes in the activity of the heart during anaphylactic shock and the influence of ether anesthesia on these changes.

EXPERIMENTAL METHOD

The part played by the sympathetic nervous system in the disorders of cardiac activity in shock was investigated in 27 guinea pigs (males) weighing 300-350 g. The animals were sensitized by a single subcutaneous injection of 0.05 ml of normal horse serum, without preservative, 15-17 days before the assaulting injection of 1 ml of the same serum into the external jugular vein.

In the first series of experiments the animals received injections of ephedrine in doses of 1-4 mg/kg into the jugular vein 5-10 min before the assaulting dose; in the second series nine guinea pigs received intravenous injections of atropine in doses of 0.1-5 mg/kg 5-25 min before the assaulting dose; in 12 animals in the third series of experiments, bilateral vagotomy was carried out 5-25 rain before the assaulting injection. The action of anesthesia was studied in 28 guinea pigs sensitized in the manner described above. An assaulting dose (0.2-3.5 ml of the same serum) was injected into the jugular vein of the animals on the 15th-17th day of sensitization 5-10min after the induction of deep ether anesthesia. In order to provide a comparison with the usual course of shock, the results of 56 experiments carried out earlier were used.

In the course of each experiment simultaneous recordings were made of the electrocardiogram in three standard leads and of the respiration by means of a 4-channel ink-recording apparatus of type 4-PFD-7. In some animals an effective sign of respiration—the movement of air in the trachea—was recorded by a photocell. Simultaneously, the respiratory movements, the blood pressure in the carotid artery (by means of a Hörthle manometer) and the general motor reaction were recorded on a kymograph.

EXPERIMENTAL RESULTS

In the experiments using ephedrine and atropine, and also after vagotomy, the injection of an assaulting dose of serum into the sensitized animals caused fatal anaphylactic shock. In the period of injection of the antigen, the changes in the blood pressure and the ECG (Fig. 1b) were the same as in ordinary sensitized guinea pigs. Those observed included a slowing of the rhythm, lengthening of the QT (more rarely, the PQ) interval, left axis deviation of the T wave and an increase in its amplitude with a less marked right axis deviation of the QRS (R), demonstrating that the disorder of function mainly affected the right ventricle [5]. Similar changes in the ECG were observed during stimulation of the vagus nerves with an alternating current. In all the experiments, during the development of the shock, the T wave was displaced to the right of the axis and inverted in the standard leads; right axis deviation of the T wave from 90° (−T1) was noted in three cases, from +150° (−T1,2) in six and beyond−150° (−T1,2,3) in 17 cases. At the same time there was an increase in the amplitude of the T wave and a sloping negative displacement of the ST interval. These changes, reflecting a disturbance of the functions mainly of the left ventricle, were intensified in the period of respiratory-motor disturbances of shock. During the
development of shock in the sensitized animals, inversion of the T wave was observed, as a rule, in only one of the standard leads, but the magnitude of the T wave and of the discordant displacement of the ST interval was less strongly and less constantly altered.

Ventricular extrasystoles, which are customary in shock, were much reduced or absent in these particular experiments. Another characteristic feature was the acute development of terminal changes in the ECG, occurring simultaneously with the terminal disorders and cessation of the respiratory movements, but with no definite connection with the reduction of the effective sign of respiration (Fig. 1c). These changes took the form of an increasing widening of the PQ interval, an accelerated left axis deviation of the T wave and the vector of the deflection of the ST interval (with suc-

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Fig. 1. Anaphylactic shock on a background of the action of ephedrine. Significance of the curves (from above, down): for a: respiratory movements; arterial pressure; general motor reaction; marker of injection of drugs and stimulation of the peripheral end of the right vagus nerve; time marker (3 sec); for b and c: marker of injection of serum; I, II, III standard ECG leads; respiration (tracing from a piezoelectric pickup); time marker (0.25 sec). The signs $\rightarrow$ in Figs. a and c designate the same moments of the state of shock.