CHANGES IN PULSE-WAVE PROPAGATION RATE IN CERTAIN ARRHYTHMIAE UNDER EXPERIMENTAL CONDITIONS

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In an experiment on dogs we studied the influence of the action of a number of factors on the elasticity of the aortic wall, which was evaluated from the pulse-wave propagation rate.

We simultaneously recorded an EKG (lead I) and a sphygmogram of the superior branch of the femoral artery. We determined the "lag" time of the pulse wave (t) from the interval between the beginning of the rise in the pulse curve. In healthy dogs $\underline{t}$ averaged 150 ±15 msec (137 experiments on 77 dogs). A decrease in $\underline{t}$ during the course of the experiment indicated an increase in the pulse-wave propagation rate, i.e., an increase in aortic elasticity, while an increase in $\underline{t}$ indicated a decrease in elasticity.

We detected a correlation between increased aortic elasticity and the appearance of extrasystole and ciliary arrythmia caused by the action of various factors [intravenous injection of adrenalin (3 μg/kg), ligation of a branch of the coronary artery, and prolonged feeding with cholesterol (0.5-2 g/kg) and methylthiouracil (1-3 g/kg)]. Extrasystole usually developed at a rather high aortic elasticity. Thus, adrenalin in a dose of 3 μg/kg (35 experiments on 13 dogs) generally caused a brief (1-2 min) increase in aortic elasticity. In experiments where the injection was accompanied by extrasystole this increase was greater in extent ($\underline{t}$ decreased by an average of 45 msec) than when the correct rhythm was maintained ($\underline{t}$ decreased by an average of 30 msec); the difference was statistically reliable ($P < 0.05$). The arrhythmia usually appeared at the instant when the maximum increase in aortic elasticity was reached.

Extrasystole was also conjoined with a relatively high aortic elasticity after transection of a branch of the coronary artery: $\underline{t}$ remained unaltered in six dogs with arrhythmia, while it increased by an average of 20 msec in seven dogs with normal rhythms, the difference being statistically reliable ($P < 0.05$).

When the animals were fed cholesterol and methylthiouracil extrasystole also appeared (in four of 13 animals) at the instant when the pulse-wave propagation rate increased ($\underline{t}$ decreased by 10-30 msec from its initial value before feeding began). One dog developed ciliary arrhythmia in conjunction with a marked rise in aortic elasticity ($\underline{t}$ decreased by 45 msec).

The pulse-wave propagation rate always dropped at the instant of extrasystole: $\underline{t}$ was greater after extrasystole than after the nomotopic impulse (34 experiments on 20 dogs). It was established in the experiments involving injection of adrenalin that the greater the decrease in $\underline{t}$ after the injection, the more substantially it increased at the instant of extrasystole (the correlation coefficiet was 0.735 and the correlation was statistically reliable, $P < 0.05$).

Extrasystole thus develops "against a background" of rather high aortic elasticity, while the elasticity drops at the instant of extrasystole, reverting to its initial level as it were. This is also noted in the presence of ciliary arrhythmia.

Certain authors [1-3] who detected a decrease in pulse-wave propagation rate in patients with extrasystole and ciliary arrhythmia attribute this phenomenon to a decrease in stroke volume resulting from curtailment of the pause between cardiac contractions. We cannot exclude this possibility, although the pause was frequently found to be pro-

longed in our experiments; on statistical processing no correlation was detected between the changes in $\underline{t}$ and those in the pause, either at the instant of extrasystole or in ciliary arrhythmia (the correlation coefficients were very low, 0.0252 and 0.0073 respectively).

The decrease in aortic elasticity in extrasystole may also have other causes. More specifically, it may be assumed that some role is played by the change in the character of myocardial contraction on ectopic excitation, which alters the dynamics of blood expulsion. However, we were convinced that in any case the character of the extrasystoles (auricular, nodal, or ventricular) does not materially affect the extent of the change in $\underline{t}$. The changes in aortic elasticity may also result from changes in arterial pressure. However, in a number of experiments involving direct recording of arterial pressure we did not detect any change in it at the instant of extrasystole. Considering the material presented above, we cannot deny the possibility that there is a direct action on the contractile elements of the aorta, reducing its elasticity in extrasystole. The very fact that aortic elasticity drops at the instant of extrasystole (having previously been elevated) may have positive value, reducing the expenditure of cardiac energy and promoting uniformity of blood flow.

LITERATURE CITED

1. É. D. Brovkovich, Kardiologiya, No. 4, p. 80 (1961).
2. F. Herzog and H. Meurer, Klin. Wschr., Bd. 18, S. 1150 (1939).
3. W. Weitz and C. Hartmann, Dtsch. Arch. klin. Med., Bd. 137, S. 91 (1921).
