THE EFFECT OF THE COMBINED ACTION OF I^{131} AND NOISE ON THE CARDIAC ACTIVITY OF THE DOG

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In the literature of radiobiology the question of the effect of I^{131} on the cardiovascular system is little dealt with, in particular under chronic experimental conditions. Only in the paper of V. L. Shvedod [13] concerning animals subjected to the action of radioiodine, was a decreased P—Q interval detected on EKG and change in the T wave.

There are also separate investigations of the effect of noise on the state of the cardiovascular system [1-6]. However, we have not encountered data concerning the effect of the combined action of prolonged noise and ionizing radiation on the organism.

This paper present the result of experiments concerned with the effect of I^{131}, noise and the combination of these two factors on cardiac activity.

MATERIAL AND METHODS

The experiments were performed on 18 male dogs of weight 10-15 kg. The animals were divided into four groups: the first group five dogs, received I^{131}; the second group, five dogs, received I^{131} and were subjected to the action of noise; the third group, four dogs, were subjected to noise; and the fourth group, four dogs, served as control.

The animals of the first and second groups were daily administered I^{131} subcutaneously (4-5 microcuries/kg) for eight months. Animals of the second and third groups were subjected to noise (daily for three hours). The noise level registered on a MAG-V magnitofone in the dogs' quarters was 100-105 db. Frequency analysis of the administered noise showed that it could be regarded as low-, medium-, and high-frequency discharges of noise irritation; the maximum sound energy was approached at frequencies of 320-1280 Hertz.

Electrocardiograms were taken monthly with three standard leads using a 4PFD-7 instrument for functional diagnosis. To bring out early EKG changes a functional load was used—intravenous epinephrine (5-10 microgm/kg). The EKG was recorded before injection of epinephrine and continuously for five minutes after its injection. The indicated dose of epinephrine in control dogs did not provoke any toxic reactions.

RESULTS

In dogs which underwent the combined activity, EKG changes began after two months of the experiment and in animals subjected to the action of iodine or noise alone—after three or four months. In experimental dogs receiving the combined treatment, toward the 7-8th month of the experiment a decrease in the frequency of cardiac contraction was observed, amount to a mean value of 25 ± 6.3% compared to the initial data. In dogs subjected to the single action of I^{131} or noise, a decrease in the cardiac rhythm was expressed less strongly (14 ± 3.3—21 ± 2.3%). Atrioventricular conduction (Q—T) in the dogs of the second group, beginning with the second month of the experiment, increased an average of 0.015 sec, and after seven months this index reached a maximum value, exceeding the initial level by 0.028 sec.

In experimental animals of group I the P—Q interval after five months increased by 0.016 sec, and later fell again.

In distinction to the animals in Groups I and II, the dogs undergoing treatment with noise alone showed a decrease in the P–Q interval at the third month by an average of 0.015 seconds. It remained at that level to the sixth month of the experiment and then returned to the original level.

Ventricular systole (Q–T) in dogs undergoing combined treatment, became prolonged by the six month and after 7-8 months exceeded the original level by 0.03-0.05 sec. In dogs of Group I the Q–T interval was less prolonged. In distinction from the dogs of Groups I and II, the Q–T interval after 2-3 months in group III dogs decreased by 0.01-0.02 seconds and remained at that level for six months at the end of the experiment the Q–T interval was close to the initial value.

The length of the systolic index in animals from Groups I and III after six months of the experiment had not changed from normal. After 7-8 months of the experiment the systolic index in animals receiving I^{131} fell to 32 ± 3.2% and in animals treated with noise to 34 ± 3.8% of the starting value; in control dogs (Group IV) at that time the systolic index was 39 ± 0.6%.

In dogs receiving the combined treatment the systolic index fell to 33 ± 2.6% somewhat earlier than in dogs of Groups I and III (after 6 months); after eight months the systolic index in this group fell to 28 ± 1.2%.

The most distinct deceleration of systole during the experiment was noted in dogs which underwent combined treatment with I^{131} and noise.

The P wave in dogs of Groups II and III at three months after the start of the experiments were 0.1 mv on the average, and in animals of Group I increased by 0.01-0.02 mv subsequently decreasing by 0.04 mv.

At one to two month after the start of the experiment the R wave in Group II dogs had increased by 0.15-0.25 mv; after 7-8 months it decreased by 0.2-0.22 mv. In four dogs in Group II, receiving combined treatment, at four months the S–T interval fell beneath the isoelectric line. In addition, these dogs showed bi-phasic or negative T waves.

For animals in Groups I and III, displacement of the S–T interval below the isoelectric line was noted only in two instances (after five months of the experiment).

The use of adrenalin evoked functional changes in cardiac activity at much earlier times of observation. If, before the appearance of effects of an epinephrine dose of 5-10 μg/kg, toxic reactions (extrasystoles, arrhythmias, blockage, etc.) did not appear, then after one month of the experiment for Group II dogs and 2-3 months for Group I and III dogs, the injection of epinephrine evoked distinct toxic reactions. The greatest changes in cardiac sensitivity to epinephrine were in dogs from Group II (three months after the start of the experiment). From this time on, increase in epinephrine-induced bradycardia was noted and a less distinct speeding up of the cardiac rhythm after vagus inhibition. For five or more minutes the systolic rate remained below the initial level. In animals of Groups I and III the type of changes in systolic rate were the same, but the wave of quickened rhythm after epinephrine bradycardia was more marked. Injection of epinephrine was accompanied by changes in the EKG pattern.

In dogs of Group II, after two months of the experiment, the P–Q and Q–T intervals, in response to epinephrine injection, increased by a mean of 0.03 and 0.04 sec, respectively. At the end of the experiment they exceeded the initial level by 0.05 and 0.06 sec. The S–T interval fell below the isoelectric line by 2.5 to 3 mm in all animals, in a subsequent period of observation the P, R and especially T waves underwent marked changes. In experimental animals from Groups I and III these changes were not a pronounced as those in dogs which were subjected to the combined treatment with noise and I^{131}.

Thus, the action of I^{131} and noise on the organism evokes a number of changes in cardiac activity which appear on the EKG. Considering the work of several authors [8, 10, 12] we suggest that the displacement of the S–T below the isoelectric line may be considered the result of coronary trophic disturbance. In our opinion, deceleration of the cardiac rhythm, observed at the 7-8 month of the experiment, evidently, evokes an increased parasympathetic effect on the heat. The decrease in arterial pressure and changes in the capillary permeability of the skin, in these animals, as we observed earlier, may also reflect the latter effect.

Concerning the stimulation of the parasympathetic nervous system, other evidence is given by the greater epinephrine-induced bradycardia and less marked quickening of the rhythm after vagal inhibition which was observed in these experiments. The definitive role in the mechanism of these changes from the point of view of the cardiac activity, evidently, is played by the disturbance of neuro-endocrine inter-relationships, in particular the function of

the thyroid gland. Ya. M. Kabak [7] in experiments on ducks after I^{131} destruction of the thyroid gland detected deceleration in systole and disruption of thermoregulation.

After the treatment with noise alone, changes in the cardiovascular system may be explained by the central nervous system effects of this factor [9, 11, 14, 15]. A number of authors [2-4, 11] detected definite changes in cardiac activity such as decrease in the systolic index, deceleration of systole, lengthening of electrical systole Q–T), decrease in height of T waves and in certain cases the appearance of Negative T waves.

The results of this study show that the use of the combination of noise and I^{131} evokes earlier and more marked changes in cardiac activity that the isolated chronic administration of I^{131} or of noise.

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