THE EFFECT OF THE THYROID GLAND ON THE MORTALITY RATE IN SHOCK-HEMORRHAGIC CONDITIONS CAUSED BY STRONG SOUND STIMULI

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In a previous work [4], we described the methods used and the results effected by the action of a sound stimulus (electric bell) in the development of serious shock conditions in rats. The studies showed that after 15-18 minute action of a sound stimulus causing acute excitation in the animal, the animal began to manifest signs of a serious shock condition: disturbance of motor coordination, paresis of the extremities, nystagmus, etc. In 12.7% of the animals, the shock condition caused death. When the animals were dissected, cerebral hemorrhages were found, caused by the outward passage of the blood from the vascular bed due to diapedesis.

The leading factor in the development of shock-hemorrhagic conditions was the acutely stimulated state of the brain, caused by the action of the sound stimulus. The death of the animals was usually not observed so long as the extent of stimulation was limited by the restraining protective inhibition. Death occurred after the inhibitory-protective function of the nerve cells had weakened. In our experiments, this condition was obtained by means of a three-minute rest period after 15-minute interrupted sound stimulation. The use of the strong stimulus again, after the rest period, caused a sharp outburst of excitation no longer restrained by the protective inhibition, which had weakened during the three-minute interval, and this led to a serious shock condition ending in death.

The initial level of nervous system excitability in the experimental animals materially affects the mortality rate in nervous traumatism; due to a previous injection of caffeine (10-20 mg per 100 g of rat's weight), for example, the mortality rate increased considerably.

The internal secretion glands influence greatly the development of shock-hemorrhagic conditions. Twice as many males as females died as a result of the nervous trauma. Castration of the males decreased the percentage of death from shock-hemorrhagic conditions, but removal of the parathyroid glands considerably increased it.

This work presents the results we obtained when studying the role of the thyroid glands in the mortality rate of rats with shock-hemorrhagic conditions caused by the action of strong sound stimuli.

EXPERIMENTAL METHODS

The work was done on rats sensitive to a sound stimulus; the rats were, for the most part, males. The rats were subjected to the action of a continous sound stimulus for 1½ minutes and then to the action of an interrupted sound stimulus with 10-second sound exposures alternating with 10-second intervals for a period of 15 minutes. Then came a 3-minute rest period, after which the strong sound stimulus was used again.

The experiments were conducted using either hyperthyroidism or extirpation of the thyroid glands.

Thyroidin was administered to the rats (weighing from 150 to 200 g) per os in doses of from 0.025 to 0.1 g for a period of 10-14 days. After this, the animals were subjected to the traumatic action of the sound stimulus by the method already described. Rats of the same sex and age which had not received thyroidin were used as the control.

The thyroid and parathyroid glands were extirpated. In the control group of rats, only the parathyroid glands were extirpated. Both the experimental and control rats of this group were subjected to the action of the sound stimulus 10-15 days after the operation.

EXPERIMENTAL RESULTS

Table 1 shows the effect of hyperthyroidism on the mortality rate of the rats which were subjected to the traumatic action of the sound stimulus.

Table 1 shows the extraordinarily large influence of hyperthyroidism on death in the rats with the induced nervous trauma. The mortality of the hyperthyroid rats was 8 times higher than that of the control rats. One should also mention that the shock condition was far more serious in all the hyperthyroid animals than in the control both during the action of the sound and after it. From the first minutes of the sound stimulus action, signs of a serious shock condition developed in most of the hyperthyroid rats, rapidly terminating in the death of the animal. Macroscopic examination of the dead experimental and control rats showed no essential differences in the frequency of cerebral hemorrhages.

TABLE 1

The Effect of Hyperthyroidism on the Mortality Rate in Nervous Trauma

Group of Animals	No. rats in exp.	Hemorrhages found		
		in absolute No.	in %	
Hyperthyroid	103	69	66.6	78.6
Control.....	108	9	8.3	83.3

* Forty-two of the 69 dead hyperthyroid rats were dissected, and 6 of the 9 control rats were dissected.

TABLE 2

The Effect of Thyroidectomy on the Mortality Rate in Nervous Trauma

Type of Operation	No. of rats in exp.	of which died		
		in absolute No.	In %	Hemorrhages found
Thyroparathyroidectomy (experiment)	35	9	25.7	44.4
Parathyroidectomy (control)	42	18	42.9	75.0

* Nine rats were dissected in the experimental group, 16 in the control.

Table 2 shows how removal of the thyroid glands affected the mortality rate of the animals subjected to nervous trauma.

Table 2 shows that the number of deaths caused by nervous trauma was much smaller after extirpation of the thyroid and parathyroid glands than after parathyroidectomy alone. Statistical evaluation of the material obtained showed that the difference in the mortality rate of the animals in the indicated groups exceeded the probable error 2.31 times. Although the authenticated difference is not great, the data obtained allow the proposal that thyroidectomy has a tendency to decrease the possibility of death resulting from the shock conditions.

Macroscopically, fewer cerebral hemorrhages were found in the experimental animals than in the control. The data presented show that removal of the thyroid glands decreases the possibility of death from brain hemorrhages in animals as a result of a shock condition brought on by a strong sound stimulus.

One must mention that the decreased mortality effect of the thyroidectomy in the experiment described appeared on a background of heightened sensibility of the animals to shock conditions, which state was observed after parathyroidectomy.

Therefore, our experiments showed that change in thyroid gland activity materially affects the mortality rate in shock conditions: hyperthyroidism sharply increases the mortality rate, while a thyroid condition, on the other hand, somewhat lowers the mortality rate.

These effects are possibly connected with the influence of the thyroid gland hormone on the tonicity and permeability of the vascular wall and on the blood pressure level [2, 3, 4, 5, 6].

SUMMARY

In prolonged (15-18 minute) action of sound stimulant, a condition of shock is developed in rats after the stage of pronounced excitation. In 12-15% of cases it is terminated by death. Brain hemorrhages are found at autopsy, which are the result of diapedesis. The fatal outcome in shock-hemorrhagic conditions depends on the relationship of the processes of excitation and inhibition and the functional condition of the endocrine glands. The functional level of the thyroid gland has the most pronounced influence on the fatal outcome in shock-hemorrhagic conditions. In animals with hyperfunction of the thyroid gland mortality rate reaches 66.6% (with only 8.3% in control animals). In extirpation of the thyroid gland the mortality rate is reduced in comparison with control animals (it is 1.7 times higher in control animals).

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