# EXPERIMENTAL DYSTROPHY OF THE PARODONTIAL TISSUES

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As is known, various effects on the superior cervical sympathetic ganglia (their extirpation, injection of turpentine or croton oil into the ganglion) causes the development of a neurodystrophic process in the parodontium of dogs, rabbits, and cats [2-5, 7-9]. There is evidence that metabolism in the teeth-jaws system is regulated by trophic effects originating from the superior cervical sympathetic ganglion [7, 8].

The question of the possibility of a drug effect on the formation and course of dystrophic processes is of interest, however, one frequently is faced with the absence of an adequate model for experimental therapeutic investigations.

The task of this investigation entailed the reproduction of the neurodystrophic process in the parodontial tissues upon stimulation of the superior cervical ganglion in rats and the development of a quantitative method of evaluating this dystrophy.

## METHOD

In the study we used small laboratory animals, rats, since it was possible to obtain a model of parodontial dystrophy in them with other stimuli on the nervous system [1]. Furthermore, rats are convenient for experimental therapeutic investigations requiring the set up of experiments on large groups of animals with subsequent statistical analysis of the results. As is known when the superior cervical ganglion of rats is stimulated exophthalmos is observed as a result of the contraction of the m. orbitalis which is innervated by the superior cervical ganglion, and this can serve as a visual check of the efficacy of the stimulation.

In rats of optimal weight (150-200 g) [10, 11] for the operation we made an incision along the midline of the neck under aseptic conditions without anesthesia, moved aside the fibers of the sternocleidomastoideus and compressed with the branches of dissecting forceps the superior cervical ganglion situated above or up agains the place of bifurcation of the carotid artery. Some rats were subjected to bilateral stimulation of the superior cervical ganglia. Functional disturbances of the ganglion were judged by the development of exophthalmos which developed on the stimulated side 1-2 days after the operation. The animals were killed 2, 4, and 6 months postoperation. We examined the mucosa of the oral cavity with subsequent maceration of the soft tissues of the mandible. After this the mandibles were studied macro- and microscopically.

The dystrophic process in the parodontial tissues was visually demonstrated by the change in the color of the mucous membrane, by exposure of the roots of the teeth, and by the presence of pockets in the soft and hard dental tissues.

The degree of exposure of the dental roots was quantitatively determined by the value of K (relative exposure of roots) expressed in percent by the formula

$$K = \frac{\Delta l}{l} \cdot 100,$$

where $\Delta l$ is the distance from the margin of the dental alveolus to the lower largin of the crown, $l$ is the distance from the margin of the alveolus to the upper margin of the crown.

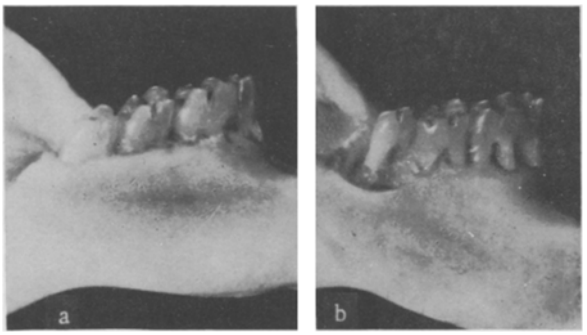

Fig. 1. Left half of the mandible of the intact rat (a) and experimental rat (b). a) Roots of teeth are very little exposed. Magnification 3.8 ×; b) roots of teeth are greatly exposed. Magnification 3.6 ×.

For the measurements we used the Mir-1 microscope (ocular 7 ×). The value of K was determined simultaneously for intact and experimental rats at various periods after stimulation. If the values of the relative exposure of the roots of the teeth for the experimental and intact animals are denoted respectively by $K_1$ and $K_2$, then the degree of development of the dystrophic process will be determined by the value $d = K_1 - K_2$.

For convenience of describing the results of the measurements the molars, as usual, were designated: tooth 1 (first tooth from incisor) tooth 2 (second tooth from incisor), tooth 3 (third tooth from incisor).

The investigations were carried out on 112 rats, in 70 of which the superior cervical ganglion was stimulated and the remaining were controls.

Degree of Evidence of the Dystrophic Process in the Parodontium Upon Stimulation of the Rat Superior Cervical Ganglion

| Time postoperation (in months) | Degree of dystrophy (in %) with indication of confidence limits | | | | | |
|---|---|---|---|---|---|---|
| | left half of jaw | | | right half of jaw | | |
| | tooth 1 | tooth 2 | tooth 3 | tooth 1 | tooth 2 | tooth 3 |
| 2 | 10±3,7 | 9±4,4 | 7±4,8 | 10±3,5 | 8±4,2 | 5±4,8 |
| 4 | 18±5,1 | 19±5,6 | 18±7,5 | 19±4,8 | 18±6,4 | 15±6,6 |
| 6 | 15±5,6 | 15±7,2 | 9±7,1 | 15±4,2 | 13±6,8 | 6±6,3 |

There were five series of experiments. In the experiments of the I series we used 20 rats, in the experiments of the II and III series 15 rats in each, in the IV series 20, and in the experiments of the V series 42 rats (control).

In the I, II, and III series of experiments the left superior cervical ganglion was stimulated once with subsequent observation for 2, 4, and 6 months respectively. In the IV series bilateral stimulation of the superior cervical ganglia was performed once with subsequent observation for 2 months.

## RESULTS

On the second day after the operation we observered exophthalmos in most rats, which, as is known, is the result of functional disorders of the ganglion. In certain animals we noted ptosis, which was considered to be the result of the death of the ganglion cells [3].

Stimulation of the superior cervical sympathetic ganglion of the rats caused the development of neurodystrophic changes in the parodontial tissues, which in all rats was manifested in the exposure of the dental roots, the formation of bony pockets, and in some animals the formation of gingival pockets and loosening of the teeth (Fig. 1). The average values of the relative exposure of the dental roots of the left half of the jaw at various postoperative periods are shown in Fig. 2. As we see from Fig. 2, the relative exposure of the roots after stimulation of the superior cervical ganglion increases, this increase becoming greater with time.

As our investigations showed, the difference in the degree of development of the dystrophic process with uni- and bilateral stimulation of the superior cervical ganglion is statistically unreliable, which permitted us to combine the data obtained with these stimulations. The degree of dystrophy (d) is shown in the table with an indication of the confidence limits ($P = 0.05$) for each period of investigation.

It follows from the data of the table that the dystrophic process for each tooth progresses with time after the operation, reaching a maximal value between 2 and 4 months, which is statistically reliable. Even in the first experiments we noted dystrophy in both halves of the jaws with unilateral stimulation of the superior cervical ganglion.

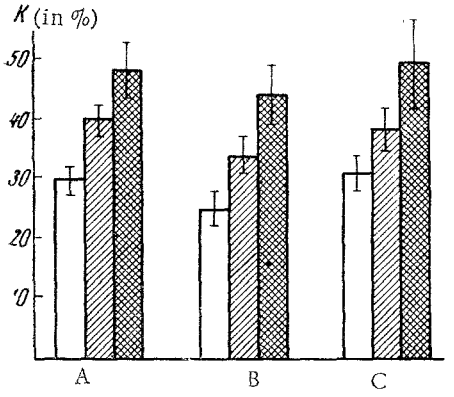


Fig. 2. Relative exposure of dental roots of the left half of the jaw at various postoperative periods. Average values (confidence limits were calculated for P = 0.05). Unhatched columns) intact rats; oblique hatching) 2 months postoperation; double hatching) 4 months postoperation. A) Tooth 1; B) tooth 2; C) tooth 3.

It is apparent from the table that the dystrophic process develops symmetrically in the left and right halves of the jaw, the differences in the degree of dystrophy being statistically unreliable. The differences in the degree of dystrophy 4 and 6 months after the operation are also statistically unreliable.

Thus the investigations showed that uni- and bilateral mechanical stimulation of the superior cervical ganglion of rats leads to the development of a neurodystrophic process in the parodontial tissues. The dystrophic process in unilateral stimulation of the superior cervical ganglion develops identically in both halves of the lower jaw; bilateral stimulation of the superior cervical ganglia does not increase the degree of dystrophy.

The dystrophic process in the parodontium develops gradually, reaching a maximum between 2 and 4 months after stimulation; its increase stops later. But during the observation period (up to 6 months) a statistically reliable restoration of the hard tissues of the parodontium was not noted.

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