THE PATHOGENESIS OF NEURODYSTROPHY

(EXPERIMENTAL MODEL OF PARODONTOSIS)

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Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 55, No. 3, pp. 117-121, March, 1963
Original article submitted May 3, 1962

The pathogenesis of neurodystrophy is still an important problem. The work of A. D. Speranskii's school laid the foundations of the study of dystrophic processes observed clinically and experimentally. Several workers [7-10, 12-14, 16] have paid particular attention to the experimental reproduction of these processes.

Previous studies [1-4] have shown that after injury to the second and third branches of the trigeminal nerve in some animals a reversible neurodystrophy develops in the parodontium. The effect of conditions such as a change in allergic reactivity and chronic stimulation of the cerebral cortex on its course has been determined.

The importance of the functional state of the higher divisions of the central nervous system to the development of parodontosis has been demonstrated by clinical observations [5, 11]. Changes have been described in the parodontium of rats during depression of the functional state of the cerebral cortex [6].

When seeking factors which would lead to the development of a pathological process in the parodontium of experimental animals closely resembling parondontosis in man, we tested the effect of a functional disturbance of the higher divisions of the central nervous system, which was combined in some animals with injury to the branches of the trigeminal nerve. Experiments were carried out on animals showing normal and allergic reactivity.

EXPERIMENTAL METHOD

The investigations were conducted on 83 male rats weighing 120-150 g. The functional disturbance of the highe divisions of the central nervous system was produced in an electrode chamber by the action of an electric current and a sound stimulus by the method described by M. O. Raushenbakh and co-workers [15].

When rats were treated daily in this manner, after 4-5 weeks the conditioned reflexes to the bell were extinguished, there was no reaction to an unconditioned stimulus, and the animals were slow to gain in weight. At this stage the branches of the trigeminal nerve were injured in some of the animals. The second branch was exposed as it left the infraorbital foramen, and the third where it left the submental foramen. The exposed nerve was ligated with a silk thread soaked in formalin. Centrally to the ligature, the nerve was crushed, and a drop of formalin was applied to the crushed area. This method provided prolonged stimulation of the injured nerve. The operation was performed under ether anesthesia. In one series of experiments the rats were sensitized by three subcutaneous injections of horse serum; reacting factor was injury to the trigeminal nerve, carried out 3 weeks later [3].

The animals were sacrificed after intervals of between 2 weeks and 4 months. The jaws with the teeth were examined histologically. Sections were stained with hematoxylin-eosin and by Mallory's method, and the nerves were impregnated by the methods of Bielschowsky and Campos. Altogether five series of experiments were carried out (see table).

EXPERIMENTAL RESULTS

Injury to the second and third branches of the trigeminal nerve in rats (the first series of experiments) led to degenerative changes in many nerve fibers and in their endings in the parodontium, and also to destructive and inflammatory changes in the parodontal tissues. In nearly all the animals of this series of experiments, 14 days after operation the gingival pockets around the upper and lower molars appeared friable, especially on the side of the op-

Distribution of Experiments by Series

Series of experiments	Character of experiment	No. of animals
First	Injury to branches of the trigeminal nerve	15
Second	Functional injury to central nervous system	16
Third	Functional injury to central nervous system and injury to branches of the trigeminal nerve	26
Fourth	Functional injury to central nervous system, sensitization, and injury to branches of the trigeminal nerve	16
Fifth	Control (intact rats)	10

eration. The epithelium lining the pathological gingival pockets grew down into the inflamed connective tissue. The changes subsequently grew in intensity. The interdental papillae were frequently eroded and coated with a purulent deposit, the gingival pockets were deepened and full of pus, and tartar was deposited on the exposed roots.

Meanwhile the alveolar process of the jaw underwent absorption. This began at the crests of the interdental and interradicular septa and extended to the whole alveolar process. The marrow in the region of the resorbing bone was replaced by connective tissue rich in cells. Diffuse infiltration of the marrow and periodontium with polymorphonuclear leukocytes and plasma cells was often observed.

The changes described above were intensified for a period of 2 months, after which repair of the parodontal tissues began to take place, while degeneration of a large proportion of the nerve fibers continued. Hence, after injury to branches of the trigeminal nerve of the rat, a comparatively intensive but reversible neurodystrophy developed in the parodontium of its molars. The destructive and inflammatory changes were much less intensive in the parodontium of the incisors, presumably on account of the structural differences between the teeth, with constant growth.

After functional injury to the central nervous system in rats (second series of experiments) a large proportion of the medullated and nonmedullated nerve fibers and their endings in the parodontium showed obvious signs of irritation (varicose thickenings, hyperimpregnation of axis cylinders, local swellings of the axis cylinders with a "saw-tooth" appearance, hypertrophy of the endings).

Destruction and inflammation developed very slowly and sluggishly in the parodontium by comparison with what was observed in the animals in the first series of experiments. In its morphological manifestations this process resembled the initial stages of parodontosis in man. A particularly noteworthy feature was that the process became irreversible in character, without signs of repair of the parodontal tissues in the course of observation for 4 months.

Fig. 1. Nerve trunk in the marrow of a rat's jaw. Degeneration of a large proportion of the nerve fibers. Objective 40x, ocular 7x. Impregnation by Campos's method.

After a combination of functional injury to the central nervous system and injury to the branches of the trigeminal nerve (third series of experiments), most of the nerve fibers and endings in the parodontium degenerated (Fig. 1). During the first 6 weeks after the operation, the character of the changes in the nerve fibers was the same as after injury to the branches of the trigeminal nerve alone. However, at the end of the second month, nearly all the surviving and regenerating nerve fibers showed signs of irritation, such as are characteristically found in rats after injury to the nervous system. These signs of irritation persisted throughout the period of observation.

Similar findings were observed in the parodontal tissues: exactly the same manifestations of neurodystrophy developed here as after injury to the branches of the trigeminal nerve alone, and then during the second month after the operation the process became increasingly severe, with no visible signs of repair of the tissues. The pathological gingival pockets became deepened, and the roots of the teeth were correspondingly exposed (Fig. 2). The resulting morphological picture was like that observed in advanced parodontosis in man.

Fig. 2. Exposure of the root of a rat's molar with the formation of a pathological gingival pocket and absorption of the bony alveolus. Objective 8x, ocular 7x. Stained with hematoxylin-eosin.

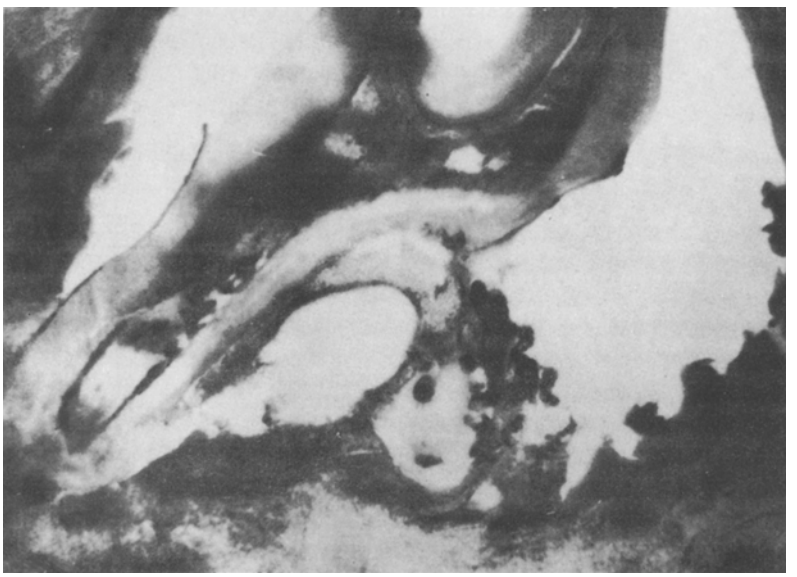

Fig. 3. Considerable exposure of the roots of a rat's molar with resorption of the cement and dentine. The pulp of the tooth is necrotic, with multiple petrificates. Objective: Reichert 3 without the frontal lens, ocular 7. Stained with hematoxylin-eosin.

In some of the rats the pathological gingival pockets reached the apex of the roots and the teeth were shed, after which the destruction and inflammation in the soft tissues and bone persisted, as also in parodontosis in man [5]. The degree of resorption of the bone varied: in some cases it was restricted to the alveolar process, while in others it spread to the body of the jaw. The marrow was often replaced by connective tissue, rich in cells, which in some cases resembled granulation tissue. The tissues of the tooth also underwent changes. Denticles and petrifications appeared in the pulp, followed by necrosis and complete death of the tooth. Areas of the cement, and sometimes of the root dentine, were resorbed (Fig. 3).

Hence, after a combination of functional injury to the central nervous system and injury to the branches of the trigeminal nerve, an irreversible neurodystrophy was reproduced, leading to loosening and shedding of some of the molars in rats.

It may be concluded from a comparison of the first and third series of experiments that the changes in the higher nervous activity and in the trophic functions of the central nervous system grossly impaired the compensatory mechanisms and the regulation of the reparative processes.

In the fourth series of experiments the functional injury to the central nervous system was inflicted in sensitized animals. In these conditions a more rapid and intensive degeneration was observed, followed by regeneration of the nerve fibers. Typical allergic changes – coarsening of the nerve elements and hyperimpregnation – were also observed. Hypemeurotization frequently developed [3].

The allergic inflammation of the parodontal tissues was manifested as vasculitis, with hypertrophy of the endothelium and serous infiltration of the vessel walls, and also by perivascular infiltration. Three rats developed parodontal abscesses which complicated the course of the process. Otherwise, the changes in the parodontium were of the same character as in the preceding series of experiments.

Hence, the combination of functional trauma to the central nervous system and injury to the branches of the trigeminal nerve led to the development of an intensive irreversible neurodystrophy in the parodontium of albino rats, thereby creating an experimental model of parodontosis.

SUMMARY

In injury of the II and III trigeminal nerve branches a reversible neuro-dystrophic process develops in the parodontium of the molars in albino rats. Changes in the higher nervous activity and of the CNS trophic functions markedly disturb the compensatory mechanisms and the regulation of restorative processes. A combination of the CNS functional traumatization and injury of the trigeminal nerve branches leads to the development of an irreversible neurodystrophic process in the parodontium of rat molars, which is an experimentally reproduced parodontosis model.

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