EXPERIMENTAL METHODS FOR CLINICAL PRACTICE

Experimental Laser Destruction of Trigeminal Sensory Root

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The possibility of local laser destruction of the trigeminal sensory root by a puncture technique is demonstrated. A fibrous scar was formed on a limited area of the trigeminal root. There were no signs of axonal regeneration, posttraumatic neuroma, or considerable changes in the Meckel sinus.

Key Words: *high-power laser irradiation; trigeminal nerve root*

Trigeminal neuralgia (TN) is traditionally distinguished as a nosological entity within the group of pain syndromes [9]. The incidence of TN is approximately 5:100,000 people per year; this disease sharply reduces capacity for work and often results in disability.

There is no universal concept of TN pathogenesis, and hence no pathogenetically substantiated approachs to pain elimination were developed. It is generally accepted that in severe neuralgia resistant to conservative treatment only interruption of nociceptive pathways can provide pain relieve [4,11].

Surgical techniques applied for TN therapy, including transection of the sensory root via the transtemporal or transoccipital approaches, tractotomy, or compression of the gasserian ganglion and trigeminal root are not always effective. Moreover, these complex techniques are associated with serious complications and even lethal outcomes and do not guarantee against TN recurrence [6]. Alcoholization [9], hydrothermal destruction (injection of hot water into the Meckel sinus [11]), and phenol application to the trigeminal nerve root [1] are recognized as the most efficient techniques. However, these methods also produced only transient pain relieve: the recurrence of pain was observed in 24-30% patients. Electrocoagulation [11] showed practically no advantages over alcoholization. The effects of physical factors on trigeminal parenchyma and adjacent tissues and the volume of lesion are difficult to control because of leakage and diffusion of agents from the Meckel sinus. These and other factors may cause severe complications like keratitis, blindness, and damage to other cranial nerves [1,11].

All this necessitates the search for new methods of TN therapy combining high effectiveness of local destruction of the trigeminal sensory root with safety, simplicity, and technical feasiability.

The aim of the present study was to obtain experimental and morphological data substantiating the application of high-power laser radiation (HPLR) for TN therapy.

MATERIALS AND METHODS

The experiments were carried out on 37 outbred male and female dogs (10-15 kg) under intravenous Kalipsol anesthesia (1 mg/kg). A Raduga-1 Nd:YAG laser (λ=1.06-μ) was used. Laser beam (1 W, 60 sec) was delivered to the trigeminal ganglion and root through a monofiber quartz guide (1 mm in diameter) passed through a lumbar puncture needle. The needle was put upward sagittally into the soft palate and introduced into the oval foramen (at a depth of 2-3 cm). The localization of the needle was verified by reflex contractions of mandibular muscles due to stimulation of branch III of the trigeminal nerve). Then the needle was passed along the gasserian ganglion to the root (0.3-0.4 cm forward), and introduced into the Meckel sinus

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through its frontalwall. To prevent traumtization of the ganglion, the needle tip not reached the trigeminal impression on the frontal surface of the temporal bone pyramid, which was controlled by X-ray imaging. Then laser radiation with a power controlled by an IMO-2 apparatus was applied. After destruction the needle was extracted and the soft palate was sutured. Behavioral responses were restored immediately after cessation of narcosis and all dogs were in satisfactory conditions. The animals were sacrificed by intrapulmonar administration of 1 g sodium thiopental 1-90 days after surgery.

The following tissues were taken for histological examination: fragments of trigeminal ganglion with nerve roots (on ipsi- and contralateral site), adjacent tissues from the pons and medulla oblongata, fragments of dura mater (DM) forming the Meckel sinus, and bone fragments forming the oval foramen and pyramid frontal surface.

RESULTS

One day after HPLR, focal alterations such as loosening of the endo-, peri-, and epineurium, degeneration of myelin sheaths, fragmentation of axons, pyknosis, lysis, and rexis of cell nuclei were observed in all examined tissues. These degenerative changes were probably caused by thermal and cavitation effects of HPLR and aggravated by rapid evaporation of tissue fluids in the irradiation focus [2]. Morphological changes typical of circulatory dysfunction such as paresis of microcirculatory vessels, stasis, coagulation of blood protein, perivascular diapedesis, and swelling of vascular walls [2] were found at the periphery of damaged area. On day 10, the lesion focus usually localized in the initial segment of the trigeminal nerve root was clearly isolated from intact tissues. Macrophages (including siderophages), proliferating fibroblasts, endotheliocytes, lymphocytes, and solitary granulocytes were noted in epi-, peri-, and endoneurium. Focal demyelinization was observed in nerve fiber bundles. Proliferation of fibroblasts and endotheliocytes and the appearance of new capillaries and fuchsinophilic fibers were characteristic of later stages. Lump degradation of myelinated axons was observed outside the lesion focus. After 30 days, granulation tissue and considerable number of fuchsinophilic fibers appeared in the lesion area. Axonal degeneration became more pronounced. A cicatricial fibrous cord crossing the root was formed after 90 days. It merged with thick fibrous layer of the peri- and epineurium and ganglionar capsule. Silver impregnation revealed no axons and myelinated fibers in this zone. The number of fuchsinophilic fibers in the endoneurium increased. Proximal axonal segments resting against the scar had no terminal branches or spiral-like tendrils. In DM, small solitary clusters of proliferating fibroblasts and endotheliocytes were found after 20-30 days. At the final stages of observation, only minor and limited thickening of DM was observed. No structural changes were revealed in the temporal bone pyramid (the region corresponding to the projection of sinus puncture). Wallerian degeneration of the trigeminal root was accompanied by progressive degeneration of axons and myelin sheaths in fiber bundles located between trigeminal ganglion neurons, reflecting retrograde degeneration of central axonal segments. Moderate glyosis and fibrosis without significant decrease in neurocyte proportion were observed in the trigeminal ganglion. Neuronal degeneration occurred only near the initial segment of the trigeminal root, *i.e.* immediately in the lesion area, where all tissues showed the signs of focal destruction at the early stages and where the fibrous scar was formed at the later stages (after 30 days).

Reparation processes (proliferation of Shwann's cells, formation of Bungner's bands and axonal growth cones accompanied by pronounced macrophage response) usually start immediately after physical or chemical damage and proceed in parallel with degeneration [8]. Under favorable conditions the rate of neurofibrills growth is about 0.25-1 mm/day. Barriers (for instance, scars) and the absence of a tunnel formed by Schwann cells [5] disorganize their growth causing the formation of structures similar to amputation neuromas. No morphological signs of fiber regeneration were observed after exposure to HPLR. Since the recurrence of TN is usually associated with nerve regeneration [1,11], our results provide a favorable prognosis.

It is very important that the trigeminal ganglion remains intact, since interventions affecting the ganglion resulted in the development of severe postoperative complication, such as neuroparalytic keratitis [11, 12]. Preservation of neuronal bodies in the course of retrograde degeneration can be attributed to optimal parameters of HPLR producing no effects on neurons of the upper sensory and spinal tract nuclei.

Thus, HPLR of the trigeminal sensory root in outbred dogs produced a focal destruction of nerve fibers with the formation of a cross fibrous scar.

No signs of axonal regeneration on the stump side, traumatic neuroma, and significant changes in the Meckel sinus tissues were observed.

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