

*Letters to the editor*

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**Successful use of nerve-block therapy with 12% tetracaine for trigeminal neuralgia caused by arteriovenous malformation****Susumu Tamakawa<sup>1</sup>, Yoshikatsu Iwanami<sup>2</sup>, Junko Tsujimoto<sup>1</sup>, Akemi Iharada<sup>1</sup>, and Hidemichi Ogawa<sup>2</sup>**<sup>1</sup> Department of Anesthesiology, Rumoi City Hospital, 1-choume, Kotobukichou, Rumoi, Hokkaido 077, Japan<sup>2</sup> Department of Anesthesiology and Critical Care Medicine, Asahikawa Medical College, 4-5-3-11, Nishikagura, Asahikawa, 078, Japan

*To the editor:* We report a case of trigeminal neuralgia, in which a nerve-block therapy with a 12% tetracaine and 0.5% bupivacaine solution relieved pain for 3 months without prolonged sensory loss.

A 35-year-old man consulted our hospital because of intermittent pain in the right forehead and cheek. The pain was always sudden in onset and persisted for a few seconds, and was triggered by pressing the right infraorbital foramen. He was diagnosed as having trigeminal neuralgia. Magnetic resonance imaging and angiography revealed an arteriovenous malformation in the cerebellum. Some part of the nidus had been embolized, and the next embolization procedure was scheduled by a neurosurgeon for 3 months later. Treatment with carbamazepine, 400 mg per day, did not reduce the patient's pain. We performed nerve-block therapy. A 22-gauge block needle was inserted into the infraorbital foramen, then 40 mg tetracaine dissolved in 0.3 ml of 0.5% bupivacaine solution (i.e., the tetracaine concentration was 12%) was injected. The pain subsided immediately, and there was an area of sensory loss corresponding with the distribution of the infraorbital nerve. Sensory loss persisted for 3 days; however, the pain did not return for 3 months.

Takenaka et al. [1] have reported that a thoracic, intrathecal block with 10% tetracaine for patients with post-herpetic neuralgia resulted in a prolonged analgesia. This report did not, however, explain why such a high concentration of tetracaine was used. In our case, we used a 12% tetracaine and 0.5% bupivacaine solution to verify the neurolytic effects.

A patient with trigeminal neuralgia has abnormal large fibers of the trigeminal nerve [2]. These fibers are enormously hypertrophic, with an axis cylinder containing degenerated Schwann-cell cytoplasm, which transmit the pain signals of trigeminal neuralgia [2]. The large fibers are easily destroyed because of their abnormal structure and the degenerated Schwann-cell cytoplasm [2].

A potential action of tetracaine to degenerate nerve fibers has been reported to be proportional to its concentration [3] in rabbits. One percent tetracaine produces a subperineural edema and a Wallerian degeneration of nerve fibers caused by endoneural ischemia [4]. On the other hand, intrathecal 2% tetracaine produces small foci of degeneration characterized by swelling of axons and myelin sheaths in the nerve roots and superficial white matter, in contrast to 0.5% or 1% tetracaine which do not degenerate fibers [3]. It may appear that 12% tetracaine selectively destroys a greater number of large fibers than does 1% tetracaine, because 12% tetracaine could induce more significant edemas and ischemic effects than does 1% tetracaine.

Recovery from an edematous state or the reproduction of nerve fibers subsequently allows for recovery from sensory loss. However, the large fibers seem to grow slowly because of abnormalities in the Schwann-cell cytoplasm. We believe that the decrease in the large fibers induces analgesia without sensory loss.

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