In some people, headache is characterized by increased frequency crisis such as the usually termed Chronic Daily Headache (CDH). According to the ICHD-II criteria, it could be caused by either the Chronic migraine (CM) or the Medication overuse headache (MOH).

A disorder in the head region can provoke pain in the areas innervated by the trigeminal and upper cervical nerves due to convergence of the afferent fibers of the three superior cervical roots on the neurones of the trigeminal nerve spinal nucleus. The therapeutic effectiveness of greater occipital and supraorbital nerve blockade in chronic migraine patients with abuse of symptomatic migraine pain medication was investigated.

**OBJECTIVES**

In some people, headache is characterized by increased frequency crisis such as the usually termed Chronic Daily Headache (CDH). According to the ICHD-II criteria, it could be caused by either the Chronic migraine (CM) or the Medication overuse headache (MOH).

A disorder in the head region can provoke pain in the areas innervated by the trigeminal and upper cervical nerves due to convergence of the afferent fibers of the three superior cervical roots on the neurones of the trigeminal nerve spinal nucleus. The therapeutic effectiveness of greater occipital and supraorbital nerve blockade in chronic migraine patients with abuse of symptomatic migraine pain medication was investigated.

**METHODS**

N.* 63 patients (n.48 females and n.15 males) affected by chronic migraine according to the ICHD-II, were given repeated daily anesthetic blocks (once or twice a day for a five day administration). Perineural injections of 0.5 to 1.0 ml of 0.5% bupivacaine were given at the epicranial emergence points of the nerves in relation to the distribution of the cephalic pain only if nerves were conspicuously pain sensitive to pressure. Each patient makes a daily record of the frequency and severity of headache during the month preceding treatment and during at least one month after treatment. The efficacy of treatment was evaluated by the number of total migraine attacks per month, analgesic consumption per month, and the Pain Total Index (PTI), an integrated expression of the intensity and duration of the headache attacks over a month. Patients were considered responsive when the PTI decreased by ≥50% in the first month after treatment. Statistical analysis utilized the “one-tailed within subject t-test” with p ≥ 0.01 on the PTI, on the number of the severe migraine attacks, and the number of analgesic doses.

**RESULTS and DISCUSSION**

63 pazienti were screened, 55 included while 8 patients were excluded. Every patients showed a significant reduction in the PTI 1 month after therapy (from 563,14 to 147,19 P≥0,01). The number of total migraine attacks per month and the consumption of analgesics per month decreased significantly as well. The treatment was without side effects in all cases. Therapeutic blockade of greater occipital and supraorbital nerves may have resulted in inhibition of the constant trigeminal hyperexcitability characterizing headache not only by blocking the conduction of noxious stimuli, but also by blocking the antidromic flow of substance P and CGRP, mediators of the axonal reflexes that underpin perivascular neurogenic inflammation. The consequent vasodilatation and extravasation of these peptides, local reinforcing factors of the algogenic stimulation, may have been interrupted by the anesthetic, resulting in normalization of the response threshold to the nociceptive stimuli.

Inhibition of axonal transport by local anesthetic is well documented. Repeated anesthetic blocks could produce a long-lasting hypostimulation of the peripheral nociceptors, rebalancing their activation threshold and consequently arresting induction of the neuroplastic mechanism of central hypersensitization that may clinically produce chronic pain.

Repeted anesthetic nerve blockade, albeit not influencing the underlying cause, would interfere in the central pathogenic mechanism of formation and transmission of trigeminal nociceptive stimulation of the migraine crisis.

**CONCLUSION**

In conclusion, we consider our preliminary data supportive of the hypothesis that the technique described may be a new nonpharmacological treatment of chronic migraine. Since it does not have any negative side effects and is easily performed. We hope that this clinical experience, together with a correct neurophysiological study, may prove useful in clarifying the pathogenic aspects of migraine.

**REFERENCES**

