

## CLINICAL CORRESPONDENCE

# Occipital nerve block rapidly eliminates allodynia far from the site of headache: a case report

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Seventy to 80% of persons with migraine develop allodynia during the course of a severe attack (1). During a migraine attack, allodynia spreads topographically to extratrigeminal territory (1, 2). Dynamic mechanical allodynia, otherwise known as brush allodynia (BA), is a subtype of allodynia that is easily tested.

Ashkenazi & Young (3, 4) recently reported on the immediate benefits of greater occipital nerve (GON) block on brush allodynia and pain in migraine and in cluster headache. In these studies, testing was performed at fixed sites in the trigeminal and cervical distributions. Allodynia in thoracic dermatomes was not studied.

### Case report

A 47-year-old woman with severe, left-sided menstrual migraine and chronic, left (more than right)-sided posterior neck pain, was evaluated. Her last severe menstrual migraine lasted three days, ending seven days prior to presentation, at which time her neck pain was at its baseline. On examination, she had moderate cervical paraspinal tenderness and left-sided allodynia from C2 to T5, including her left arm. Brush allodynia was tested by stroking the subject's skin repetitively with a folded 2 × 2 inch gauze pad at 2 Hz until she experienced an unpleasant sensation or eight brushes were completed.

A left GON block and bilateral tender point injections at C2 and left C5 paraspinal and trapezius muscles were given. A total of 5cc of 2% lignocaine and 10 mg of triamcinolone were used. One minute after achieving GON anaesthesia the allodynia was reduced in intensity and all allodynia and neck pain had resolved after five minutes.

### Discussion

In this report we show that GON block with paraspinal tender point injection can eliminate allodynia caudal to the site of injection. Previous reports of allodynia responding to GON block were consistent with an effect via convergent inputs to second order trigeminal nucleus caudalis neurons with expanded receptive fields. In order for such a monosynaptic process to be involved in this case, axons would need to enter the dorsal root entry zone at T5 and synapse with neurons near the cervicomedullary junction, a distance of approximately 0.3 meters, which is unlikely.

We therefore offer an alternative explanation, and suggest that a more generalized process is involved. We propose that a diffuse antinoceptive process is initiated by GON/tender point anaesthesia resulting in the turn-off of BA.

Diffuse noxious inhibitory controls (DNIC) occur when the response to a noxious stimulus is inhibited by a second, spatially remote, noxious stimulus (5). This phenomenon has been extensively studied in animal models of pain (6) and in man (7, 8). A possible explanation for our findings is therefore that the injections themselves initiated DNIC, independent of an anaesthetic effect. The DNIC effect has been shown to start as early as one minute after the application of the noxious stimulus (8). This is in accordance with our observation in this patient. A saline injection could show that the technique was either due to DNIC or a placebo effect.

Further observations are needed to clarify whether it is the nociceptive element of injection or

the anaesthetic effect of GON/TP injection that turn off allodynia after GON block.

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