Endogenous inhibition of trigeminal nociception is impaired in persons with severe headaches
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Introduction
Research has shown that some chronic pain conditions, including headache disorders, are associated with a dysfunction of pain inhibitory systems. To date, however, few studies have examined this issue using a physiological measure of trigeminal nociceptive processing. The current study assessed diffuse noxious inhibitory controls (DNIC), which is a way of examining endogenous inhibition. DNIC involves the application of a tonic, noxious, conditioning stimulus that inhibits phasic pain evoked from a distant body site. Animal research suggests this inhibition is mediated by a spino-bulbo-trigeminal nerve spinal circuit that inhibits nociceptive processing as well as pain perception. The current study assessed whether endogenous inhibition, assessed from DNIC, of trigeminal nociception was disrupted in persons suffering from disabling headaches.

Participants
The severe headache group was comprised of 11 individuals with migraine and/or tension-type headache who were categorized as having moderate or severe disability according to the Migraine Disability Assessment Scale (MIDAS). Controls were 11 individuals with no headaches or with infrequent tension-type headache (minimal or mild disability according to MIDAS). Exclusion Criteria: ≥18 years of age, Current acute illness, Cardiovascular, neurological, circulatory, and/or hearing problems, Chronic pain condition (e.g., back pain), Current use of anxiolytic and/or anti-hypertensive medication, Recent psychological trauma.

Procedure
A reliable and objective nociceptive reaction to noxious stimulation of the head was used to assess pain sensitivity. The nociceptive blink reflex (nBR) was measured in response to an electrocutaneous stimulus delivered to the trigeminal nerve. Pain ratings were made following each stimulation.

Data Analysis
• Pain ratings and nBR were averaged across the 4 pre-ischemia (baseline) stimulations and across sets of 2 stimulations in the ischemia and post-ischemia phases. Data were standardized by calculating a percentage change from baseline.
• Severity Group: Controls vs. Severe Headache x 5 (DNIC Phase: Baseline, Isch1, Isch2, Postisch1, Postisch2) mixed model ANOVAs were conducted.
• Planned simple effects tests of DNIC phase were conducted for each group even in the absence of a significant interaction.

Pain Ratings
• No significant Main Effect of nBR (p=.27).
• Significant interaction of nBR and Severity (p=.033). DNIC-inhibition of nBR was seen in the Control Group but not the Headache Group.
• Simple Effect of DNIC within the Control Group (p=.004). The nBR was significantly larger during baseline when compared to post-ischemia magnitudes in the Control Group.

Conclusions
Results indicated DNIC-inhibition of pain ratings was similar in both groups. However, DNIC-inhibition of nBR was noted in the control group, but not the severe headache group. These findings suggest a dysfunction of endogenous inhibition of trigeminal nociception in persons suffering from disabling headaches, although replication with larger sample sizes may be needed. Further research is needed to determine whether inhibitory