The ability to inhibit pain signals is disrupted 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, no known study has 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-spinal circuit that inhibits nociceptive processing as well as pain perception. The current study assessed whether endogenous inhibition (assessed from diffuse noxious inhibitory controls, DNIC) of trigeminal nociception was disrupted in persons suffering from disabling headaches.

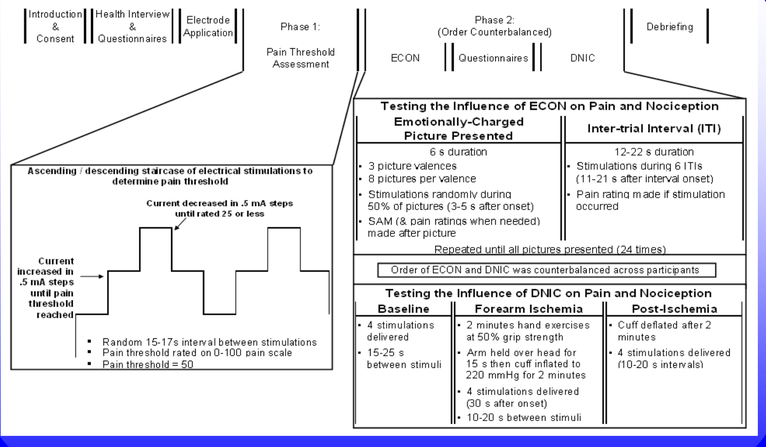
Objective

It was hypothesized that headache sufferers with the greatest amount of disability would evidence the greatest disruption of endogenous inhibition.

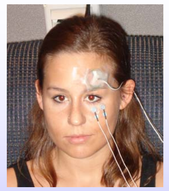
Participants

- 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)
  - Recent use of analgesic medication
  - Current use of anxiolytic and/or antihypertensive medication
  - Recent psychological trauma

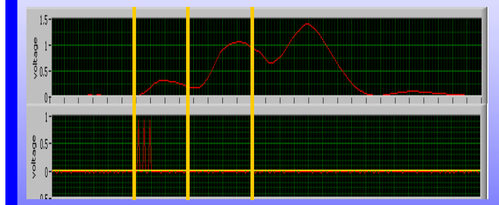
Procedure



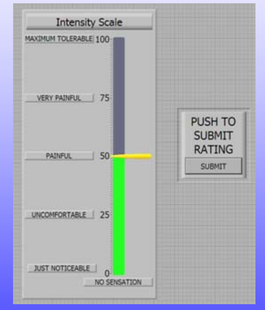
Nociceptive Blink Reflex (NBR) Magnitude



- **Concentric stimulating electrode**: left trigeminal nerve
- **NBR recording electrodes**—left orbicularis oculi muscle

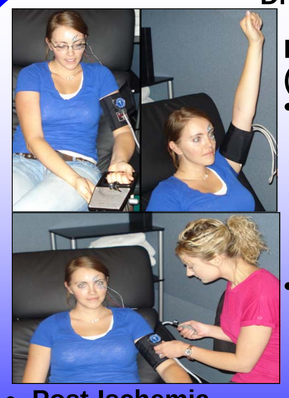


- **NBR:** a reliable and objective nociceptive reaction to noxious stimulation of the head
- **NBR magnitude** = mean of orbicularis oculi EMG in 27-87ms post-stimulus interval minus mean of 60ms pre-stimulus interval



- **Pain ratings:** made following each stimulation

DNIC Procedure



Pre-Ischemia (Baseline)

- 4 electrocutaneous stimulations delivered to trigeminal nerve (120% pain threshold): 15-25 s interval between stimulations
- Pain ratings made following each

Ischemia

- 2 minutes of hand exercises (50% maximum grip strength) followed by 15 s of arm elevation then blood pressure cuff inflated to 220 mm/Hg
- 4 electrocutaneous stimulations delivered to trigeminal nerve (120% pain threshold): 15-25 s interval between stimulations

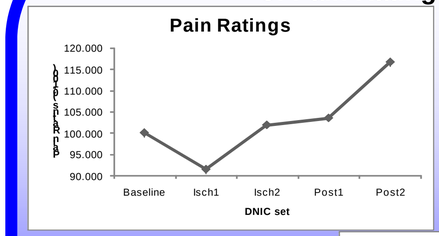
Post-Ischemia

- Blood pressure cuff deflated after 2 minutes
- 4 electrocutaneous stimulations delivered to trigeminal nerve (120% pain threshold):15-25 s interval between stimulations
- Pain ratings made following each stimulation

Data Analysis

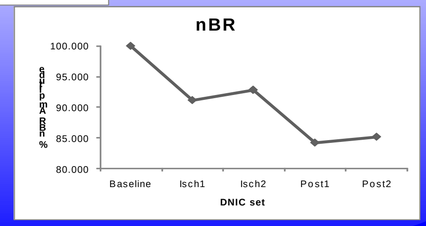
- Pain ratings and nBR were averaged across the 4 pre-ischemia (baseline) stimulations and across sets of 2 stimulations in the Ischemia and Post-Ischemia phases
- 2 (Severity Group: Controls vs. Severe Headache) x 5 (DNIC Phase: Baseline, Isch1, Isch2, PostIsch1, Postisch2) mixed model ANOVAs were conducted
- Planned simple effects tests of DNIC phase were conducted for each group even in the absence of a significant interaction

Results: Endogenous Inhibition of Pain Ratings and nBR



- **Main Effect of Pain Ratings (*p*<.01) was significant.** Pain ratings were significantly higher during the final post-ischemic set, relative to both ischemic sets and the first post-ischemic set.

- **No significant Main Effect of nBR (*p*=.09).**



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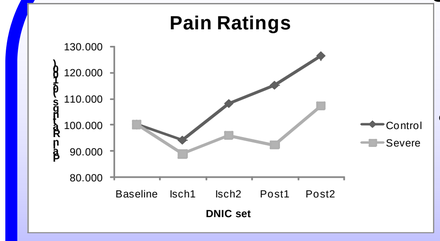
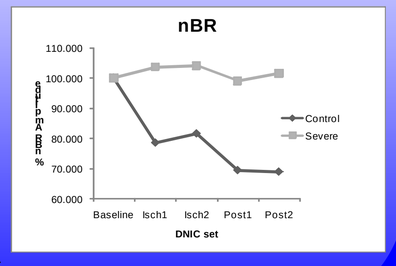


- **No significant interaction of Pain Ratings and Severity**

- **Significant interaction of DNIC Phase and Severity Group (*p*<.05).** DNIC inhibition of nBR was seen in the Control Group during both post-ischemic sets but not the Severe Headache Group.
- **Simple Effect of DNIC phase within the Control Group was significant (*p*<.01).** The nBR reflex was significantly larger during baseline when compared to both post-ischemia set magnitudes in the Control Group.



Conclusion

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. Further research is needed to determine whether inhibitory dysfunction is a precursor or a consequence of the headaches.