[2003 Fall A9] Decreased tissue pH caused by gastrocnemius muscle incision in the rat

Woo Y, Park S, Brennan T
University of Iowa Hospitals, Iowa City, IA, USA

Introduction: Postoperative incisional pain is a common form of acute pain. Most studies in postoperative pain management are focused on pharmacologic treatments. One approach to advancing acute pain management is to better understand the etiology of incision pain. We have developed and characterize a rat model for postoperative pain that includes plantar incision. In addition, we have examined the model of secondary hyperalgesia produced by incision to gastrocnemius muscle. The purpose of this study was to measure pH changes produced by an incision in the gastrocnemius muscle to determine if local decreases in tissue pH may be present in muscle incisions. In this study we measured tissue pH after gastrocnemius incision.
Methods: After approval by the Institutional Animal Care Unit, rats underwent single incision of the gastrocnemius muscle or a sham operation. Tissue pH was measured using a pH sensitive needle electrode (Diamond General, Ann Arbor MI). Gastrocnemius pH was measured after sham muscle incision or muscle incision. Muscle pH was measured for one hour on the day of incision, and on postoperative days 1, 4 and 8.
Results: Tissue pH prior to incision was 7.14 ± 0.7 in the sham operated group and 7.14 ± 0.4 in the incised group (n=6 per group). Five minutes after incision, tissue pH was 6.58 ± 0.11. In the sham operated group, tissue pH was 7.14 ± 0.17. Sixty minutes after incision, pH in the incised group was 6.76 ± 0.17 ($P<0.05$) and pH in the sham operated group was 7.14 ± 0.17. Tissue pH was also significantly decreased ($P<0.05$) on day 1 (6.96 versus 7.20) and day 4 (7.06 versus 7.18) after incision. Tissue pH was not different on postoperative day 8 (7.11 versus 7.15).
Conclusions: In conclusion, gastrocnemius incision decreased tissue pH beginning five minutes after incision and continuing through postoperative day 4. Decreased tissue pH after muscle incision corresponded to pain behavior, suggesting that tissue acidosis may contribute to nociceptor sensitization and subsequent pain behaviors like secondary hyperalgesia after muscle injury.

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