POTASSIUM IONTOPHORESIS AS AN EXPERIMENTAL PAIN STIMULUS: ITS PSYCHOPHYSICAL CHARACTERISTICS AND ITS UTILITY FOR INVESTIGATING THE SPINAL MODULATION OF PAIN

A Thesis presented in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Psychology at Massey University

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1995

ACKNOWLEDGEMENTS

I would like to thank my first superviser Professor Nigel Long for his helpful assistance, practical advice and patience; Malcolm Johnson for his help and useful ideas; Dr John Podd for believing in good experimental design; and Dr. Alan Winton for his constant support, and for being enthusiastic about psychology.

I would also like to thank the people who gladly volunteered for the experimental studies.

A special thanks to Andrea who proof-read the draft. Thank you for helping me at the important final stage.

And, finally, to my wife Sharon and family who provided love and understanding throughout the research. Your support has helped me bring this thesis to fruition.

ABSTRACT

The present study investigated the psychophysical characteristics of potassium iontophoresis and its suitability as an experimental pain stimulus. Experiment One investigated the optimal duration of the pain stimulus for reliable reporting across repeated trials, and whether the relationship between stimulus and subject response was linear, logarithmic or a power function. Experiment Two determined the optimal inter-stimulus interval (ISI) for reliable pain reporting and evaluated stimulus history effects, both in terms of session effects and the effects of immediately preceding stimuli. Experiment Three compared potassium iontophoresis with a sodium iontophoresis control.

Linear functions described the stimulus-pain relationship best. No significant differences in the goodness-of-fit coefficients of determination, correlations, or coefficients of variation, were found for the stimulus durations of 1, 2 and 4 seconds. Significant stimulus history effects were found across a session, with adaptation and enhancement of responding for low and moderate intensity stimuli respectively. The effects of the immediately preceding stimuli were suppression or enhancement of pain response depending on the ISI, the preceding stimulus intensity and the present stimulus intensity. Potassium iontophoresis was a significantly more effective pain stimulus than sodium iontophoresis.

It was concluded that potassium iontophoresis is a convenient and reliable experimental pain stimulus. It can be presented rapidly and repeatedly with minimal loss in consistency of subject pain report. Potassium iontophoresis provides a technique for investigating the neural modulation of pain in the relative absence of inflammation processes and tissue damage.

The properties of potassium iontophoresis determined in Experiments One, Two and Three indicated that it could be an ideal nociceptive stimulus for a quantitative analysis of some of the spinal modulation mechanisms predicted by the gate control theory of pain. Clinical and experimental support for the gate control theory of pain was overviewed.

According to the gate control theory of pain a peripheral stimulus that activated both small and large-diameter afferent fibres would be perceived as painful, though there would be some reduction in the intensity of the pain due to the inhibitory action of the large fibre activity.

The present study investigated a prediction of gate control theory that there would be a transient increase in pain above that of the background level - a pulse of pain - as the pain stimulus was being ramped off due to the large fibre activity at the spinal level falling away more quickly due to the different peripheral conduction velocities of large and small fibres. A further prediction was that the more distant the peripheral stimulus was from the spine the greater the pain pulse would be for any given ramp-off rate. Supraspinal pain modulatory mechanisms were overviewed but excluded as possibly obscuring the predicted pain pulse generated through the ramping off of the peripheral nociceptive stimulus.

Fourteen subjects had the experimental pain stimulus of iontophoretically applied potassium ions (K^+) applied to an upper and a lower site on the dominant arm. Each stimulus trial consisted of four seconds of constant pain followed by the stimulus being ramped off. In a threshold detection task a double random staircase method was used to adjust the ramp-off rate. Subjects were asked to indicate if they could detect a brief pulse of additional pain during this ramp-off phase. Subjects were clearly able to detect a pulse of pain at both sites. The average rate of stimulus ramp-off in order to detect a pain pulse was statistically greater for the upper-arm site (14.3 μg K^+/s), than for the lower-arm site (9.4 μg K^+/s). The average ramp-off time required to generate a detectable pain pulse was 192 ms and 261 ms for the upper and lower arm sites respectively.

These results were consistent with the predictions of gate control theory and our ramp-off model. Alternative explanations for the results, including intrinsic differences in nociceptive responding for different dermatomes and anode break excitation, were considered.

It was concluded that the detection of a pain pulse during the ramping off of a peripheral pain stimulus potentially provides a quantitative measure of the spinal modulation of pain as described by the gate control theory of pain. However, further studies would be required to confirm the causal mechanisms that generate the observed pulse of pain.

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