Pain, motion sickness and migraine: effect on symptoms and scalp blood flow

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This thesis is presented for the degree of

DOCTOR OF PHILOSOPHY

Faculty of Health Sciences, Psychology, Murdoch University

Western Australia

2009
DECLARATION

I declare that this thesis is my own account of my research and contains as its main content work which has not previously been submitted for a degree at any tertiary education institution

_____________________________________
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ABSTRACT

Migraine, a neurovascular disorder, is associated with disturbances in brain stem activity during attacks. Interictal persistence of these disturbances might increase vulnerability to recurrent attacks of migraine. To explore this possibility, effects of motion sickness and pain on migrainous symptoms and extracranial vascular responses were investigated in 27 migraine sufferers in the headache-free interval, and 23 healthy age/sex matched controls.

Symptoms of migraine and motion sickness are remarkably similar. As both maladies involve reflexes that relay in the brain stem, they most probably share the same neural circuitry. Furthermore, migraineurs are usually susceptible to motion sickness and, conversely, motion sickness-prone individuals commonly experience migraine. Participants in the present study were exposed to optokinetic stimulation (OKS), a well-established way of inducing symptoms of motion sickness in susceptible individuals.

Sensitivity to painful stimulation of the head and hand was also explored. Head pain is a hallmark of a migraine attack and cutaneous allodynia has been observed elsewhere in the body during attacks. The trigeminal nerve is associated with head pain in migraine, and trigeminal activity evokes reflexes that relay in the brain stem. To stimulate the trigeminal nerve, ice was applied to the temple. To stimulate nociceptors elsewhere in the body the participant immersed their fingers and palm in ice-water.

Procedures used in this study were physically stressful and probably psychologically stressful. The impact of stress in relation to the development of
symptomatic and vascular responses, particularly anticipatory stress-responses, was explored.

This research involved one central experiment that consisted of six experimental conditions. On separate occasions participants were exposed to optokinetic stimulation and painful stimulation of the head or limb, individually and in combination.

In migraine sufferers, symptomatic responses were enhanced during all procedures involving OKS and during temple pain after OKS, in the presence of residual motion sickness. During trigeminal stimulation independent of OKS, headache initially developed followed by nausea as the procedure progressed. In contrast, symptoms barely developed in controls during any of the six procedures except for slight dizziness, self-motion and visual-illusion during conditions involving OKS, and slight nausea when the temple was painfully stimulated during OKS and during OKS alone. Trigeminal stimulation during OKS intensified nausea and headache in migraine sufferers compared to during OKS alone or limb pain during OKS. However, the remaining symptomatic ratings were not affected by temple pain during OKS, suggesting a specific association between nausea and head pain. It may be that these cardinal symptoms compound one another during a migraine attack. Enhanced symptomatic responses in migraine sufferers during the headache interval may indicate activation of hypersensitive neural pathways that mediate symptoms of motion sickness or migraine. Migraineurs found procedures generally more unpleasant, and ice-induced pain ratings more intense and unpleasant, than controls, which may further indicate hyperexcitable nociception in this group, or a difference in their criterion of discomfort.
Vascular responses, particularly during OKS alone, and during painful stimulation independent of OKS, were greater in migraine sufferers than in controls. The added stress of painful stimulation during OKS appeared to boost facial blood flow in controls to approach levels obtained in migraine sufferers. Enhanced vasodilatation was observed in migraineurs prior to painful stimulation, presumably due to anticipatory anxiety.

For both groups ipsilateral vascular responses were greater than contralateral responses when the hand was painfully stimulated. During limb pain before OKS asymmetry was minimal in migraine sufferers but more apparent in controls. An enhanced stress response in migraineurs may have drawn ipsilateral and contralateral responses closer together.

The development of symptoms during the procedures of this study provides an insight into how symptoms might develop sequentially in a migraine attack. Once the headache is in motion, nausea and headache may mutually exacerbate one another. In turn, trigemino-vascular responses and stress appear to be associated with the migraine crisis. Given the interactive nature of symptomatic, vascular, and stress responses, it may be more effective to target multiple, rather than individual, symptoms, in prophylactic or acute chemical and psychological interventions.
Publications related to this thesis


Part of this thesis was platform presented at an international conference in London, United Kingdom. Refer to publications related to this thesis, Granston and Drummond (2002), Appendix 14, page 443-444. Slides illustrating the content of this PowerPoint presentation are presented in Appendix 14, pages 459-464. *
Other publications in response to this thesis


See Appendix 14, pages 418-464, for copies of publications *

* Copies not available in the online digital version of this thesis
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Acknowledgements

At the commencement of my candidature, I met with my supervisor, Professor Peter Drummond to discuss aspects of this forthcoming research. The pathophysiology of migraine, which in many ways despite the groundbreaking and promising research to date, still remains an enigma. The opportunity to contribute practically toward the knowledge-base in understanding this disease was an exciting challenge, which I eagerly looked forward to starting. I was also daunted at the mere thought of the research journey that lay ahead of me.

However, thanks to Professor Drummond’s unfailing and expert guidance, and calming influence during times of pressure, my enthusiasm and excitement never waned. Learning to become a researcher has been the pinnacle of my career to date, and in many ways, now that my thesis has been submitted; I suspect that I will miss the whole researcher-experience terribly.

Professor Drummond is highly regarded internationally across disciplines and is a prolific contributor to the research literature. Consequently I feel privileged to have been under his tutorage, as my association with him has no doubt had a positive impact on my development as a researcher. Apart from being a prominent neuroscientist he is also an adept clinical psychologist and academic. His talents were formally recognized by Murdoch University when he was recently awarded the ‘Vice-Chancellor’s Excellence in Supervision Award’.
Professor Drummond was always accessible throughout my candidature regardless of how pressured was his timetable. He was consistently focused on the issues at hand, an impeccable critic, communicated his ideas clearly and encouraged lateral thinking. Professor Drummond encouraged me to present findings of this study to international and local audiences. He also encouraged me to contribute sections of my thesis findings for publication in various prestigious journals, which helped me to become increasingly familiar with my results, their interpretation, and how to communicate them orally and to a written publishable standard. From a personal viewpoint, my association with Professor Drummond these past years has instilled in me a desire for the pursuit of excellence, and taught me that a daunting task is only as big as the next manageable step ahead of you.

Financial assistance by way of a scholarship from Murdoch University Research and Development Board is appreciatively acknowledged. Other financial assistance included imbursement to participants for their assistance, and also the Research Student Conference Travel Award, towards expenses to attend an international conference in Britain, where I presented sections of this thesis. Special thanks to Karen Olkowski (Department Manager) and Emma Thorp - your professionalism and people skills made a big difference.

I gratefully acknowledge Murdoch University, Faculty of Health Sciences, Psychology technicians (David Nicholson, Man Trac, Francis Lee) for their expert technical assistance when needed.
Early in my research, during the testing of participants I shared our laboratory with a number of colleagues – all busy collecting data for their own Ph.D.’s. I thank each of them for their patience, consideration, mutual support and assistance as we worked around one another to complete our individual data collections. I particularly enjoyed the many enjoyable chats and coffees with Shiree Treleavan-Hassard (colleague and friend), when we weren’t busy testing, about the neurology of migraine and psychophysiological recording!

A heartfelt thank you goes to my family - my parents, and Sandra, Michael, and Marisa for their encouragement, enthusiasm, and faith in me. I also sincerely thank Ilma, an especially dear friend, for her encouragement and love. Also, cheers to my very good friends Leonie and Kevin, and Betty and Harry, your enjoyable and positive company always kept me in good spirits. Further appreciation goes to Paul, my husband, for his confidence in me and for tolerating my perpetual ‘organized-mess’ of journal papers, files, and textbooks, which completely filled every available breathing space in my office.

My sincere gratitude extends to those migraine sufferers and controls who altruistically volunteered to participate in procedures for this study. Impressively, despite considerably uncomfortable and unpleasant procedures, participants returned to the laboratory (often reluctantly) to endure 3 separate testing sessions all in all. Indeed, without their dedicated help this research would not have been possible.
Finally I acknowledge the pleasing and therapeutic presence of our pets: Willy (a delightfully affectionate chicken) and Minx and Tim (two playful, sometimes naughty, sun conures – that had total disregard to my allocated ‘thesis-time’). Much gratitude to Maria Gardiner of the Staff Development and Training Unit, Flinders University South Australia for her helpful hints on the management of these pesky but adorable Ph.D. - sabotaging parrots! Her Clinical Psychology skills apparently extend to remedying behavioural problems in those with feathers.

No help from these two (Minx and Tim)