

Arthurs, O., Donovan, Tim ORCID: https://orcid.org/0000-0003-4112-861X, Carpenter, T. Adrian, Pickard, John D. and Boniface, S. (2001) Neurovascular relationships in human sensorimotor cortex can be modulated by movement. NeuroImage, 13 (6). S968-S968.

Downloaded from: http://insight.cumbria.ac.uk/id/eprint/1050/

Usage of any items from the University of Cumbria's institutional repository 'Insight' must conform to the following fair usage guidelines.

Any item and its associated metadata held in the University of Cumbria's institutional repository Insight (unless stated otherwise on the metadata record) may be copied, displayed or performed, and stored in line with the JISC fair dealing guidelines (available here) for educational and not-for-profit activities

provided that

- the authors, title and full bibliographic details of the item are cited clearly when any part of the work is referred to verbally or in the written form
 - a hyperlink/URL to the original Insight record of that item is included in any citations of the work
- the content is not changed in any way
- all files required for usage of the item are kept together with the main item file.

You may not

- sell any part of an item
- refer to any part of an item without citation
- amend any item or contextualise it in a way that will impugn the creator's reputation
- remove or alter the copyright statement on an item.

The full policy can be found here.

Alternatively contact the University of Cumbria Repository Editor by emailing insight@cumbria.ac.uk.

PHYSIOLOGY

Neurovascular relationships in human sensorimotor cortex can be modulated by movement

O J Arthurs, T Donovan, T A Carpenter, J D Pickard, S J Boniface

Wolfson Brain Imaging Centre, Box 65, Addenbrooke's Hospital, Hills Road, Cambridge, CB2 2QQ. UK.

The interpretation of fMRI is critically dependent on understanding the relationship between observed blood flow responses and the underlying neuronal changes. We have established a linear neurovascular coupling relationship between fMRI BOLD and somatosensory evoked potential (SEP) amplitude in human sensorimotor cortex using changes in stimulus intensity ¹. Finger movement during a stimulus is known to attenuate electrical SEP responses in somatosensory cortex (movement gating) ^{2,3}, however it is also a common method of inducing fMRI BOLD activity in sensorimotor cortex, suggesting that electrical and haemodynamic cortical responses diverge. Here we examined the effects of finger movement during sensory stimulation on fMRI BOLD and SEP amplitude in sensorimotor cortex, in order to establish whether movement can modulate this relationship.

Methods

Normal volunteers were subject to 0.2 millisecond square-wave electrical pulses delivered to the median nerve at the wrist, at 50 – 175 % of predetermined motor threshold. SEPs were recorded from contra-lateral parietal cortex, Cp3/4, referenced to Fz, over 450 averages. Gradient-echo EPI BOLD imaging was performed on a 3.0 Tesla Bruker Medical S300 using a blocked design with TR 4 sec. Imaging was acquired for all intensities, which were pseudo-randomised, then the experiment was repeated with subjects performing a self-paced thumb twitch during the stimulus. Images were analyzed using SPM99.

Results

Without movement, fMRI BOLD responses paralleled changes in SEP N20-P27 amplitude (p<0.05) as both increased with increasing intensity (p<0.05; p<0.05 respectively). However, thumb movement increased fMRI BOLD signal intensity change (p<0.05) but caused a significant decrease in SEP N20-P27 amplitude across all intensities in all subjects (p<0.05).

Conclusion

SEP amplitudes increased with increasing stimulus intensity, as did fMRI BOLD signal intensity changes, consistent with a close neurovascular coupling relationship. However the effect of simultaneous movement is to reset this relationship by increasing the cerebral blood flow response and suppressing the electrical response. The apparent coupling relationship can therefore be modulated by movement.

References

- 1. Arthurs et al., 2000. Neuroscience 101; 4; 803-806.
- 2. Jones SJ et al., 1981. Electroenceph. clin. Neurophysiol 52; 517-530
- 3. Cheron G & Borenstein S, 1987. Electroenceph. clin. Neurophysiol 67; 537-548.

Acknowledgements

Bruker Medizintechnik, Ettlingen, Germany Merck Sharpe and Dohme, Harlow, UK Oxford Instruments, Surrey, UK.