Perception, 2008, volume 37, pages 367 – 374

doi:10.1068/p5872

Transcranial direct current stimulation and visual perception

Andrea Antal, Walter Paulus

Department of Clinical Neurophysiology, Georg-August University of Göttingen, Robert Koch Strasse 40, D 37075 Göttingen, Germany; e-mail: Aantal@gwdg.de

Presented at Interdisciplinary Conference on Pre-Emptive Perception, Hanse Institute for Advanced Studies, Delmenhorst, Germany, 15 – 18 October 2005

Abstract. Membrane potentials and spike sequences represent the basic modes of cerebral information processing. Both can be externally modulated in humans by quite specific techniques: transcranial direct current stimulation (tDCS) and repetitive transcranial magnetic stimulation (rTMS). These methods induce reversible circumscribed cortical excitability changes, either excitatory or inhibitory, outlasting stimulation in time. Experimental pharmacological interventions may selectively enhance the duration of the aftereffects. Whereas rTMS induces externally triggered changes in the neuronal spiking pattern and interrupts or excites neuronal firing in a spatially and temporally restricted fashion, tDCS modulates the spontaneous firing rates of neurons by changing resting-membrane potential. The easiest and most common way of evaluating the cortical excitability changes is by applying TMS to the motor cortex, since it allows reproducible quantification through the motor-evoked potential. Threshold determinations at the visual cortex or psychophysical methods usually require repeated and longer measurements and thus more time for each data set. Here, results derived from the use of tDCS in visual perception, including contrast as well as motion detection and visuo – motor coordination and learning, are summarised. It is demonstrated that visual functions can be transiently altered by tDCS, as has been shown for the motor cortex previously. Up- and down-regulation of different cortical areas by tDCS is likely to open a new branch in the field of visual psychophysics.

1 Introduction

Modulation of cortical excitability and activity is a central mechanism of neuroplasticity (Bennett 2000). Long-term potentiation (LTP) and long-term depression (LTD) have been shown to be involved in learning processes in animal and human studies. As revealed by early animal studies, stimulation with weak electrical direct currents (DCs) can induce stimulation-polarity-dependent prolonged diminutions or elevations of cortical excitability, most probably elicited by hyperpolarisation or depolarisation of resting-membrane potentials. Most of these studies were performed in the 50s and 60s of the last century (Bindman et al 1964; Creutzfeldt et al 1962; Gartside 1968; Purpura and McMurtry 1964; Terzuolo and Bullock 1956). In the cat, anodal stimulation depolarised the soma of pyramidal tract cells, whilst cathodal stimulation hyperpolarised them (Creutzfeldt et al 1962; Purpura and McMurtry 1964). The induced potential shifts were not totally homogeneous, but depended on stimulation strength, cortical layer, and spatial orientation of the neurons. With sufficient duration of stimulation, the effect of stimulation can outlast the duration of the stimulation by several hours (Bindman et al 1964).

Early human studies were largely confined to the treatment of psychiatric diseases, mainly depression and mania. It was found that weak DC stimulation, with the anode placed supraorbitally, diminished depressive symptomatology (Constain et al 1964), while cathodal stimulation reduced manic symptoms (Carney 1969). In healthy subjects, anodal stimulation resulted in increased activity and elevated mood, while cathodal stimulation was followed by quietness and apathy (Lippold and Redfearn 1964). However, these effects could not be replicated by all follow-up studies, possibly because of different patient subgroups or other technical differences. Concerning healthy subjects, several electrophysiological and psychophysical studies were performed in the 80s.

In the visual cortex it was shown that anodal stimulation could modulate perception, the amplitude of the visual evoked potentials, and slow cortical activity during and after the end of DC stimulation (Korsakov and Matveeva 1982). However, despite these interesting combined electrophysiological and psychophysiological effects, the knowledge about the basic physiological properties of DC stimulation in humans remained incomplete.

The recent technique of transcranial magnetic stimulation (TMS) makes it possible to quantify the effects of tDCS on the excitability of the human cortex. TMS is ideally suited for the measurement of the effects of tDCS over the motor cortex, since it elicits motor-evoked potentials (MEPs). In the last few years it has been shown that transcranially applied DC can modulate excitability and activity of the motor cortex in healthy subjects, both during and after stimulation in a polarity-dependent way, noninvasively and painlessly (Nitsche and Paulus 2000, 2001; Nitsche et al 2003b). Anodal stimulation increased the amplitude of MEPs while cathodal stimulation decreased them provided the stimulation duration and intensity were sufficient. The relevant stimulation parameters encompass not only the polarity but also the combination of current strength, size of the stimulated area, and duration of the stimulation (Agnew and McCreery 1987), and are considered to be safe (for reviews see Iyer et al 2005 and Nitsche et al 2003a; Poreisz et al 2007). In most studies on humans tDCS is delivered by a battery-driven constant-current stimulator through a pair of conductive rubber electrodes in a $5\ \text{cm} \times 7\ \text{cm}$ isotonic-saline-solution-soaked synthetic sponge placed on the scalp. The current is applied for 3 – 20 min with an intensity of 1 to 2 mA.

2 Applying transcranial direct current stimulation (tDCS) in visual studies

As regards visual modality, the perceptual effects of tDCS were found to be in accordance with its physiological effects and mirrored those produced in the motor cortex: tDCS modulated the amplitude of visual evoked potentials (VEPs) (Antal et al 2004a), evoked by black-and-white sinusoidally modulated gratings in the on – off mode, modified the perception of phosphenes (Antal et al 2003a, 2003b), affected contrast sensitivity (Antal et al 2001) as well as motion detection thresholds (Antal et al 2004c), and reduced the duration of the motion aftereffect (Antal et al 2004d). It was also found that tDCS can modulate visuo – motor performance, and its effect depends on the type of the task and the time of the stimulation related to the phase of the task (learning versus overlearned). Table 1 summarises the stimulation parameters of all the tDCS studies related to the visual cortex.

(1) Cathodal tDCS over the primary visual cortex decreased, whilst anodal tDCS increased, the amplitude of the N70 component of the VEP which is a representative potential with respect to visual-cortex excitability. Significant effects were only observed when low-contrast visual stimuli were applied. High-contrast stimuli may activate the respective visual cortical areas maximally; therefore subthreshold excitability modulation induced by tDCS may produce fewer smaller changes in the VEP in this case. Both anodal and cathodal stimulations were effective, but their effect on the duration of the aftereffect was different: cathodal stimulation was more effective than anodal stimulation. The data are in agreement with other results of using tDCS in animal studies (Bindman et al 1964; Creutzfeldt et al 1962) showing that the effect of cathodal stimulation is stronger than the effect of anodal stimulation when identical stimulation parameters are used. However, as regards the stimulation polarity, an opposite effect was found in a recent study: anodal stimulation resulted in reduced P100 amplitude while cathodal stimulation increased it (Accornero et al 2007). These apparently discrepant results are probably due to different VEP modalities used: sinusoidal pattern onset in the first study and checkerboard pattern reversal stimulation in the second one. Furthermore, the position of the reference electrode (Cz vs neck) could have a strong influence on the tDCS effect.

Table 1. An overview of stimulation parameters and functional effects of tDCS in previous human and animal visual studies (modified after Nitsche et al 2003). Animal studies are greyed out.

Authors	Electrode position	Electrode size/cm^2	Stimulation duration/min	Current strength/A	Effects
Accornero et al 2007	Oz vs posterior neck-base	40	3–10	0.001	anodal stimulation reduced P100, cathodal stimulation increased
Antal et al 2001	Oz vs Cz	35	7	0.001	elevated visual perception threshold by cathodal tDCS
Antal et al 2003b	Oz vs Cz	35	10	0.001	phosphene threshold reduced by anodal and increased by cathodal tDCS
Antal et al 2003a	Oz vs Cz	35	10	0.001	moving phosphene threshold reduced by anodal and increased by cathodal tDCS
Antal et al 2004c	left V5 vs Cz	35	7	0.001	modified motion perception threshold by anodal and cathodal tDCS
Antal et al 2004a	Oz vs Cz	35	5–15	0.001	elevated N70 amplitude by anodal and reduced N70 amplitude by cathodal tDCS
Antal et al 2004b	left V5 vs Cz	35	10	0.001	improved visuo–motor learning by anodal tDCS
Antal et al 2004e	Oz vs Cz	35	10	0.001	elevated gamma and beta oscillatory activities by anodal and reduced by cathodal tDCS
Creutzfeldt et al 1962	intracortical, visual, and motor cortex	0.001	seconds	up to 0.001	most neurons activated by anodal and deactivated by cathodal stimulation; reversed effect in deep layers and in sulci
Korsakov and Matveeva 1982	occipital vs mastoid	0.79	130–200	0.0002	VEP-modulations by anodal stimulation; slow cortical activity changes; decreased perception sensitivity
Kupfermann 1965	visual cortex, implanted epidural electrodes	400 (proposed)	0.2 s	0.0002	cathodal stimulation impairs learning, anodal stimulation not effective
Landau et al 1964	motor, visual, somatosensory cortex surface	10–30 (proposed)	0.25 s	0.0001 –0.0025	anodal stimulation increases amplitude of negative and decreases that of positive EP-waves, cathodal effect opposite; in surface and deep layers opposite effect; dependent on neuronal orientation
Morrell and Naitoh 1962	epidural electrodes, visual cortex	600 and more	0.05 s	0.00005	cathodal stimulation impairs performance, anodal improves it the next day
Szeligo 1976	epidural electrodes, visual cortex	45			increased negative VEP-waves by anodal stimulation; more effective with repetitive stimulation, less time needed to learn a visual avoidance task
Varga et al 2007	P6–P8 vs CZ	35	10	0.001	cathodal stimulation reduced the duration of gender-specific aftereffect
Ward and Weiskrantz 1969	epidural electrodes, visual cortex	1800			visual discrimination decreased by anodal stimulation; effects depends on stimulation duration and intensity

(2) Similarly to the effect of tDCS on VEP, cathodal tDCS resulted in decreased static and dynamic contrast sensitivities of healthy human subjects after stimulation, probably by decreasing cortical excitability (Antal et al 2001). However, the excitability enhancement effect of anodal stimulation was not apparent, probably owing to a ceiling effect: the stimulus used in this study had 'optimal' spatial frequency and its perception could not be improved further by anodal stimulation.
(3) Magnetic stimulation over V1 results in stationary phosphenes. For quantification of tDCS-induced excitability changes, phosphene thresholds (PTs), that is the lowest intensity at which the phosphenes are detectable, were measured with short trains of TMS pulses in healthy subjects before and after the end of anodal or cathodal stimulation. Reduced PT was detected immediately and 10 min after the end of anodal stimulation while cathodal stimulation resulted in an opposite effect (Antal et al 2003b). However, we have to consider that phosphene perception is very subjective and phosphenes are relatively variable in position and form. Furthermore, the PTs are highly dependent on the technical implementation of the experiment, for example the shape and size of the stimulating coil and the induced current directions (Kammer et al 2001; Meyer et al 1991).
(4) Our recent results provide evidence that external modulation of neural excitability in human MT+/V5 affects the strength of perceived MAE and support the involvement of MT+/V5 in motion adaptation processes (Antal et al 2004d). Both cathodal and anodal stimulation over MT+/V5 resulted in a significant reduction of the perceived MAE duration, but had no effect on performance in a luminance-change-detection task used to determine attentional load during adaptation. Similarly, a recent study demonstrated that cathodal stimulation of the right temporo-parietal cortex reduces the magnitude of facial adaptation, while stimulation over the V1 produces no significant effects (Varga et al 2007). These data imply that mainly lateral temporo-parietal cortical areas play role in facial adaptation and in facial gender discrimination, supporting the idea that the observed aftereffects are the result of high-level, configural adaptation mechanisms. In agreement with previous studies, we found that the inhibitory effect of cathodal tDCS on adaptation is possibly related to the focal diminution of cortical excitability due to membrane hyperpolarisation.
(5) With respect to the functional effects of tDCS, it was surprisingly found that the percentage of correct tracking movements increased significantly during and immediately after cathodal tDCS of V5, whilst anodal stimulation had no effect when an already learned manual visuo–motor tracking task was applied (Antal et al 2004c). The highly specific effect of reducing excitability in V5 but enhancing performance of this visually guided tracking task is most likely explained by the complexity of perceptual information processing needed for the task. The complexity of the task probably produces a kind of noisy activation state of the encoding neuronal pattern in response to different movement directions. Apart from this activation, not only the correct, but also some incorrect, directions are activated simultaneously to a certain degree. In this 'noisy' activation state cathodal stimulation may focus correct perception of these parameters by decreasing the global excitation level and thus diminishing the amount of activation of concurrent patterns below threshold. Therefore it improves the signal-to-noise ratio and improves performance. This hypothesis was supported by other studies with random-dot kinetograms (Antal et al 2004c). However, when tDCS was applied *during the learning phase* of the same visuo–motor coordination task in a different subject group, the performance increased significantly during a 2–5 min time interval after the beginning of *anodal* stimulation of V5 or M1, whereas cathodal stimulation had no significant effect (Antal et al 2004b). The positive effect of anodal tDCS was indeed restricted to the learning phase, thus suggesting a highly specific effect of the stimulation.

These studies imply that the effect of tDCS depends not only on the electrode position and the polarity of stimulation but also the type of the task and the *duration* of the stimulation with respect to the phase of the task. Previous studies on the motor cortex indicate that the direction of rTMS-induced plasticity critically depends on the pre-existing level of excitability (Lang et al 2004), suggesting the existence of a homeostatic mechanism in the human cortex that stabilises cortical excitability within a physiologically useful range. A similar mechanism in the visual cortex probably exists, and therefore the application of the tDCS before and during the task may result in a different functional state.

2.1 *Molecular mechanism of tDCS*

Whereas the effects during stimulation are most probably due to the DC-induced shifts of resting-membrane potential, this mechanism does not explain the induction of long-lasting aftereffects. As shown by previous studies (Gartside 1968; Islam et al 1997), the excitability-enhancing effects of anodal stimulation depend on protein synthesis. Additionally, they seem to involve cAMP modulations, changes in intracellular calcium level (Hattori et al 1990; Islam et al 1995a), and early gene expression (Islam et al 1995b). Additionally, it was also suggested that the aftereffects of cathodal tDCS include non-synaptic mechanisms based on changes in neuronal membrane function (Ardolino et al 2005).

Many recent pharmacological studies proved that the aftereffects are NMDA-receptor dependent (Liebetanz et al 2002; Nitsche et al 2004). It is known that long-lasting NMDA-receptor dependent cortical excitability and activity shifts are involved in neuroplastic modification. NMDA-receptor and intracellular sigma 1 receptor blocker dextromethorphan (DMO) intake prevented both anodal and cathodal tDCS-induced aftereffects, demonstrating that DMO critically interferes with the functionality of tDCS, irrespective of the polarity of DC stimulation (Liebetanz et al 2002). It was also demonstrated that D-cycloserine, a partial NMDA-agonist, selectively potentiated the duration of motor cortical excitability enhancements induced by anodal tDCS (Nitsche et al 2004). Dopaminergic mechanisms can stabilise these processes. In a recent study, the dopaminergic influence on NMDA receptor-dependent neuroplasticity was investigated using tDCS. The enhancement of D2 and—to a lesser degree—of D1 receptors by pergolide consolidated tDCS-generated excitability diminution up until the morning after stimulation (Nitsche et al 2006).

2.2 *Duration of the aftereffects of tDCS*

Previous studies on the motor cortex determined that by varying the duration of tDCS one could change the duration of the aftereffects (Nitsche and Paulus 2000, 2001). Motor cortical tDCS lasting 2 – 5 min was already effective in eliciting aftereffects, however, in a VEP study (Antal et al 2004a) this was the case only for cathodal stimulation. Stimulation lasting 10 min resulted in a 10 min aftereffect; 15 min of stimulation produced longer (20 min) aftereffects from cathodal stimulation. However, this duration is relatively short compared to the 30 – 60 min aftereffects elicited by motor cortical tDCS produced by the same stimulation durations. A possible explanation for this difference is provided by anatomical and physiological characteristics of the motor and visual cortices: there are partly different types of neurons and neurotransmitters/neuromodulators in the two cortices, and the motor cortex is more an indirect 'output' area while the visual cortex is mainly responsible for filtering and transfering the information towards other cortical areas; therefore it is more like an 'input' area. However, it could also be argued that the primary visual cortex is less 'plastic' compared to the primary motor cortex under tDCS.

3 Conclusions and perspectives

Weak DC stimulation can induce acute as well as prolonged cortical excitability and activity changes over the motor and visual areas. It influences the activity of the brain electrically and changes organised cortical activity transiently and reversibly in a non-invasive, painless way, like repetitive TMS (rTMS). With respect to the mode of action, however, tDCS and rTMS are somewhat different. Whereas rTMS induces externally triggered changes in the neuronal spiking pattern and interrupts or excites cortical activity in a spatially and temporally restricted fashion, tDCS most likely modulates the spontaneous firing rates of neurons by changing resting-membrane potential and thus modifies ongoing neuronal activity and related functional performance. rTMS is loud and often induces muscle contraction during the stimulation. Furthermore, when stimulus duration is longer than 2 min, a coil-holder is needed. tDCS induces only light itching of the skin under the electrode at the beginning of the stimulation. Therefore it is more suited for studies where sham stimulation is also necessary.

Both methods are safe and their applications over the motor and visual areas are well-documented. However, compared to TMS, tDCS is less focal. Spatially, relatively large electrode size (35 cm^2) is used and, temporally, resolution of the stimulation is not so optimal. Nevertheless, in the case of both methods, we also have to consider that modulation of the excitability of a given brain area is unlikely to affect neuronal function only in that targeted brain region. When activity of a given brain area is modified, the behavioural impact is the consequence of how the rest of the brain copes with the modulation of the activity. Our studies show that excitability modulation in a neuronal network of V1, M1, and V5 leads to changes in brain activity which can influence behaviour in different ways.

The early effects of DC stimulation in humans are attributed to changes in membrane polarisation by analogy with the animal studies. However, the current densities applied in the human studies are much lower than those in animal studies (see table 1). Probably the position of the electrodes is more critical in the case of human studies. Future studies will, no doubt, be devoted to the calculation of the effective current, depending on the electrode positions and distances, required to change a perceptual function. The other critical point related to membrane polarisation is the difference in timing between the animal and human studies. However, technically it is not yet possible to determine the lag at the beginning of the DC effects in humans.

To make tDCS relevant not only for basic research purposes, but also for clinical applications, additional studies are necessary, especially studies on extending the duration of the effects and accompanying safety studies. In principle, the use of tDCS could be beneficial in conditions and diseases accompanied by pathological changes of the cortical excitability of the visual areas, eg in amblyopia, migraine, photosensitive epilepsy, and neglect. Our results also raise the possibility of using tDCS in the rehabilitation from brain injuries in which visuo – motor coordination is impaired because of deficient visual processing.

Acknowledgment. Parts of these studies were funded by the Bernstein Center for Computation Neuroscience (01GQ0432).

References

Accornero N, Li Voti P, La Riccia M, Gregori B, 2007 "Visual evoked potentials modulation during direct current cortical polarization" *Experimental Brain Research* **178** 261 – 266

Agnew W F, McCreery D B, 1987 "Considerations for safety in the use of extracranial stimulation for motor evoked potentials" *Neurosurgery* **20** 143 – 147

Antal A, Kincses T Z, Nitsche M A, Bartfai O, Paulus W, 2004a "Excitability changes induced in the human primary visual cortex by transcranial direct current stimulation: direct electrophysiological evidence" *Investigative Ophthalmology and Visual Science* **45** 702 – 707

Antal A, Kincses T Z, Nitsche M A, Paulus W, 2003a "Modulation of moving phosphene thresholds by transcranial direct current stimulation of V1 in humans" *Neuropsychologia* **41** 1802 – 1807

Antal A, Kincses Z T, Nitsche M A, Paulus W, 2003b "Manipulation of phosphene thresholds by transcranial direct current stimulation in man" *Experimental Brain Research* **150** 375 – 378

Antal A, Nitsche M A, Kincses T Z, Kruse W, Hoffmann K P, Paulus W, 2004b "Facilitation of visuo – motor learning by transcranial direct current stimulation of the motor and extrastriate visual areas in humans" *European Journal of Neuroscience* **19** 2888 – 2892

Antal A, Nitsche M A, Kruse W, Kincses T Z, Hoffman K P, Paulus W, 2004c "Direct current stimulation over V5 enhances visuo – motor coordination by improving motion perception in humans" *Journal of Cognitive Neuroscience* **16** 521 – 527

Antal A, Nitsche M A, Paulus W, 2001 "External modulation of visual perception in humans" *NeuroReport* **12** 3553 – 3555

Antal A, Varga E T, Kincses T Z, Nitsche M A, Paulus W, 2004e "Oscillatory brain activity and transcranial direct current stimulation in humans" *NeuroReport* **15** 1307 – 1310

Antal A, Varga E T, Nitsche M A, Chadaide Z, Paulus W, Kovacs G, Vidnyanszky Z, 2004d "Direct current stimulation over MT+/V5 modulates motion aftereffect in humans" *NeuroReport* **15** 2491 – 2494

Ardolino G, Bossi B, Barbieri S, Priori A, 2005 "Non-synaptic mechanisms underlie the after-effects of cathodal transcutaneous direct current stimulation of the human brain" *Journal of Physiology* **568** 653 – 663

Bennett M R, 2000 "The concept of long term potentiation of transmission at synapses" *Progress in Neurobiology* **60** 109 – 137

Bindman L J, Lippold O C, Redfearn J W T, 1964 "The action of brief polarizing currents on the cerebral cortex of the rat (1) during current flow and (2) in the production of long-lasting after-effects" *Journal of Physiology* **172** 369 – 382

Carney M W P, 1969 "Negative polarisation of the brain in the treatment of manic states" *International Journal of Medical Science* **2** 133 – 135

Constain R, Redfearn J W, Lippold O C, 1964 "A controlled trial of the therapeutic effect of polarisation of the brain depressive illness" *British Journal of Psychology* **110** 786 – 799

Creutzfeldt O D, Fromm G H, Kapp H, 1962 "Influence of transcortical de-currents on cortical neuronal activity" *Experimental Neurology* **5** 436 – 452

Gartside I B, 1968 "Mechanisms of sustained increases of firing rate of neurones in the rat cerebral cortex after polarization: Role of protein synthesis" *Nature* **220** 383 – 384

Hattori Y, Moriwaki A, Hori Y, 1990 "Biphasic effects of polarizing current on adenosine-sensitive generation of cyclic AMP in rat cerebral cortex" *Neuroscience Letters* **116** 320 – 324

Islam N, Aftabuddin M, Moriwaki A, Hattori Y, Hori Y, 1995a "Increase in the calcium level following anodal polarization in the rat brain" *Brain Research* **684** 206 – 208

Islam N, Aftabuddin M, Moriwaki A, Hori Y, 1997 "Effects of anodal polarization on protein kinase Cgamma (PKCgamma) in the rat brain" *Indian Journal of Physiology and Pharmacology* **41** 204 – 210

Islam N, Moriwaki A, Hattori Y, Hayashi Y, Lu Y F, Hori Y, 1995b "c-Fos expression mediated by N-methyl-D-aspartate receptors following anodal polarization in the rat brain" *Experimental Neurology* **133** 25 – 31

Iyer M B, Mattu U, Grafman J, Lomarev M, Sato S, Wassermann E M, 2005 "Safety and cognitive effect of frontal DC brain polarization in healthy individuals" *Neurology* **64** 872 – 875

Kammer T, Beck S, Erb M, Grodd W, 2001 "The influence of current direction on phosphene thresholds evoked by transcranial magnetic stimulation" *Clinical Neurophysiology* **112** 2015 – 2021

Korsakov I A, Matveeva L V, 1982 "Psychophysical characteristics of perception and of brain electrical activity during occipital micropolarization" *Human Physiology* **8** 259 – 266

Kupfermann I, 1965 "Effects of cortical polarisation on visual discriminations" *Experimental Neurology* **12** 179 – 189

Landau W M, Bishop G H, Clare M H, 1964 "Analysis of the form and distribution of evoked cortical potentials under the influence of polarizing currents" *Journal of Neurophysiology* **27** 788 – 813

Lang N, Siebner H R, Ernst D, Nitsche M A, Paulus W, Lemon R N, Rothwell J C, 2004 "Preconditioning with transcranial DC stimulation sensitizes the motor cortex to rapid-rate TMS and controls the direction of after-effects" *Biological Psychiatry* **56** 634 – 639

Liebetanz D, Nitsche M A, Tergau F, Paulus W, 2002 "Pharmacological approach to the mechanisms of transcranial DC-stimulation-induced after-effects of human motor cortex excitability" *Brain* **125** 1 – 10

Lippold O C, Redfearn J W, 1964 "Mental changes resulting from the passage of small direct currents through the human brain" *British Journal of Psychology* **110** 768 – 772

Meyer B U, Diehl R R, Steinmetz H, Britton T C, Benecke R, 1991 "Magnetic stimuli applied over motor cortex and visual cortex: Influence of coil position and field polarity on motor responses, phosphenes and eye movements" *Electroencephalography and Clinical Neurophysiology* **43** 121 – 134

Morell F, Naitoh P, 1962 "Effect of cortical polarization on a conditioned avoidance response" *Experimental Neurology* **6** 507 – 523

Nitsche M A, Jaussi W, Liebetanz D, Lang N, Tergau F, Paulus W, 2004 "Consolidation of human motor cortical neuroplasticity by D-Cycloserine" *Neuropsychopharmacology* **29** 1573 – 1578

Nitsche M A, Lampe C, Antal A, Liebetanz D, Lang N, Tergau F, Paulus W, 2006 "Dopaminergic modulation of long-lasting direct current-induced cortical excitability changes in the human motor cortex" *European Journal of Neuroscience* **23** 1651 – 1654

Nitsche M A, Liebetanz D, Antal A, Lang N, Tergau F, Paulus W, 2003a "Modulation of cortical excitability by weak direct current stimulation—technical, safety and functional aspects" *Clinical Neurophysiology Supplement* **56** 255 – 276

Nitsche M A, Nitsche M S, Klein C C, Tergau F, Rothwell J C, Paulus W, 2003b "Level of action of cathodal DC polarisation induced inhibition of the human motor cortex" *Clinical Neurophysiology* **114** 600 – 604

Nitsche M A, Paulus W, 2000 "Excitability changes induced in the human motor cortex by weak transcranial direct current stimulation" *Journal of Physiology* **527** 633 – 639

Nitsche M A, Paulus W, 2001 "Sustained excitability elevations induced by transcranial DC motor cortex stimulation in humans" *Neurology* **57** 1899 – 1901

Poreisz C, Boros K, Antal A, Paulus W, 2007 "Safety aspects of transcranial direct current stimulation concerning healthy subjects and patients" *Brain Research Bulletin* **72** 208 – 214

Purpura D P, McMurtry J G, 1964 "Intracellular activities and evoked potential changes during polarization of the motor cortex" *Journal of Neurophysiology* **28** 166 – 185

Szeligo F, 1976 "Electrophysiological and behavioral effects of transcortical polarizing current: comparison with the behaviorally determined characteristics of learning" *Brain Research* **103** 463 – 475

Terzuolo C A, Bullock T H, 1956 "Measurement of imposed voltage gradient adequate to modulate neuronal firing" *Proceedings of the National Academy of Sciences of the USA* **42** 687 – 694

Varga E, Kaya E, Antal A, Zimmer M, Harza I, Paulus W, Kovacs G, 2007 "Cathodal transcranial direct current stimulation over the parietal cortex modifies facial gender adaptation" *Clinical Neuroscience* [Ideggyogyaszati Szemle] **60** 474 – 479

Ward R, Weiskrantz L, 1969 "Impaired discrimination following polarisation of the striate cortex" *Experimental Brain Research* **9** 346 – 356

ISSN 0301-0066 (print) ISSN 1468-4233 (electronic)

PERCEPTION
VOLUME 37 2008

www.perceptionweb.com