Transcranial magnetic stimulation over the cerebellum and eye movements: state of the art

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Abstract

Transcranial magnetic stimulation (TMS) transientsly induces an electrical field in the tissues beneath the area of application, thereby perturbing local cortical activity if applied over the scalp. It can therefore be used to modulate cerebellar function in healthy humans. Even though the role of the cerebellum in eye movement control and adaptation is well known, few experiments have used eye movements to evaluate the effect of TMS over the cerebellum. Single-pulse TMS over the posterior vermis resulted in impaired accuracy of reflexive saccades, acceleration of smooth pursuit, and coordination of saccades and head movements. TMS over the cerebellar hemisphere decreased pursuit gain. Repetitive TMS (rTMS) over the posterior vermis impaired saccade adaptation in a double-step paradigm. Comparing the effects of TMS on different behavioural paradigms could be useful to test cerebellar control of reflexive and voluntary eye movements, and as a probe of cerebellar plasticity. rTMS appears to be especially interesting since its effects outlast the stimulation period and its behavioural consequences can therefore be measured without interfering with the execution of eye movements or with the experimental procedures.

KEY WORDS: cerebellum, eye movements, transcranial magnetic stimulation

Introduction

Transcranial magnetic stimulation (TMS) over the cerebral and cerebellar cortex in healthy humans can affect processes described in animal studies, and also in IMRI and lesion studies in humans. Single or paired pulse TMS can be used to disrupt activity in any cortical area, making it possible to explore its functional relevance in a given behaviour. Repetitive TMS (rTMS) has long-lasting conditioning effects (1) that give rise to functional changes in interconnected neural circuits. Therefore, it is a technique suitable for investigating plasticity within a distributed functional network, and thus for studying, in humans, the mechanisms of cortical functional reorganisation that have been described, in animal data, both at the level of the synapse and at cellular level. Furthermore, rTMS offers new possibilities for studying and treating dynamic aspects of the pathophysiology of some neurological and psychiatric disorders (2-8). The control of gaze depends on a number of areas in the cerebral cortex, from which parallel projections descend, via the basal ganglia and superior colliculus, to the brainstem and cerebellar circuits that produce the premotor commands [see (9) as a textbook reference]. Saccades are rapid eye movements that enable the eyes to move the fovea to an object of interest. Reflexive saccades are directed towards an abruptly appearing target (Fig. 1, over), while voluntary saccades are directed towards a predictable target, a memorised target position, or executed as part of a complex and purposeful movement. Smooth pursuit eye movements allow continuous, clear vision of objects moving in the visual environment, allowing the image of the object of interest to be kept on the fovea. Smooth pursuit is triggered by optical flow on the retina; the properties of the moving stimulus are coded in the visual cortex and then sent to the superior colliculus and pontine nuclei via the frontal and parietal eye fields and extrastriate areas (Fig. 2, over). Since different cortical and subcortical areas are involved to different degrees in the control of reflexive saccades, volition saccades, and smooth pursuit (Table I, over), in order to understand the function of a specific region it is essential to investigate it using different types of eye movements. Only a few studies have used experimental TMS approaches in conjunction with eye movement recording to investigate cerebellar functions (10-14). Some of these studies provided interesting findings on the pattern of physiological cerebellar ocular motor control in healthy subjects (see Table II, over, for a synopsis). This review presents their main findings, underlining in particular which eye movement paradigms (reflexive saccades, voluntary saccades, smooth pursuit) and parameters (accuracy, velocity, latency) were modulated by single-pulse TMS or rTMS over the cerebellum.
Cerebellar contribution to eye movements

Within the cerebellum two main regions (the vestibulocerebellum and the ocular motor vermis) influence eye movements. The vestibulocerebellum (flocculus, paraflocculus, nodulus and uvula) is interconnected with the vestibular pathway and involved in the control of smooth pursuit and eye-head tracking, and in the control, suppression and plasticity of the vestibulo-ocular reflex (15). Due to its ventral location, the vestibulocerebellum is not suitable for TMS stimulation.

The Purkinje cells (PCs) of the ocular motor vermis (OMV: posterior vermis, lobule VIc and VII) and the caudal fastigial nucleus (FN) calibrate saccade amplitude, and play a critical role in saccade adaptation (16). A saccade is characterised by a burst discharge (pulse) of the active motor neuron that moves the eye rapidly against elastic and viscous forces; this is followed by a tonic level of activity (step), aimed at maintaining the eyeball in the new position. The pulse is generated by the burst neurons located in the pons and midbrain. The saccade will be slow if there is impairment of the pulse amplitude, while it will be inaccurate if there is impairment of the pulse duration (resulting in hypo- or hypermetric saccades). Adaptive mechanisms can detect erroneous performance and recalibrate the motor response to a given visual stimulus in order to eliminate the dysmetria. For instance, in the presence of ocular motor palsy saccadic pulse amplitude can be readjusted in order to improve the performance of the paretic eye. A saccade occurs so quickly that there is not enough time for any visual information to be used as a feedback to modify it before it has finished. If the eye movement is not accurate at

![Diagram of saccadic eye movements](image1)

**Figure 1** - Neural structures involved in saccadic eye movements.

Saccades are initiated by activity in neurons of the frontal and parietal eye fields of the cerebral cortex. The signals generated then follow two pathways, one projecting to the nucleus reticularis tegmenti pontis (NRTP) and the other to the superior colliculus (SC). The NRTP sends projections to the Purkinje cells of the ocular motor vermis which, in turn, send GABAergic inhibitory projections to the caudal fastigial nucleus (cFN). Two pathways leaving the cFN influence saccade execution and adaptation: a short pathway [1] projects directly to the brainstem saccade pulse generator that generates the premotor saccade commands and provides the cerebellum with the efference copy of its premotor command; a long pathway [2] ascends via the thalamus to various cortical eye fields, which may then influence the SC and the pulse generator.

![Diagram of smooth pursuit eye movements](image2)

**Figure 2** - Neural structures involved in smooth pursuit eye movements.

Optic flow in the periphery of the visual fields is coded in the striate cortex and extrastriate areas. The signals generated are sent to the cerebellum via the nucleus of the optic tract (NOT), the inferior olive (IO) and the dorso-lateral pontine nuclei. The cerebellar areas involved in smooth pursuit control include the ocular motor vermis and fastigial nucleus, the flocculus and paraflocculus (projecting to the medial vestibular nucleus), and the hemispheres.

### Table 1 - Neural pathways involved in control of different types of eye movements

<table>
<thead>
<tr>
<th>Behavioural types of eye movements</th>
<th>VC</th>
<th>ESC</th>
<th>PPC</th>
<th>PEF</th>
<th>SEF</th>
<th>PFC</th>
<th>FEF</th>
<th>DLPN</th>
<th>SC-NRTP</th>
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Abbreviations: VC=visual cortex; ESC=extrastriate cortex; PPC=posterior parietal cortex; PEF=parietal eye fields; SEF=supplementary eye fields; FEF=frontal eye fields; DLPN=dorso-lateral pontine nuclei; SC=superior colliculus; NRTP=nucleus reticularis tegmenti pontis. Visually guided saccades=reflexive saccades externally triggered by a visual stimulus; Voluntary saccades=internally triggered saccades towards a target not yet or no longer visible; Smooth pursuit=tracking movement executed to maintain the fovea on a moving object.

Lesions of the ocular motor vermis and fastigial nucleus impair accuracy and adaptation of reflexive saccades (while voluntary saccade accuracy may be spared) and affect velocity and adaptation of smooth pursuit.
the end of its course, a corrective saccade will be generated, and corrective movements can occur even when the target is extinguished before the initial saccade is completed. The occurrence of corrective saccades depends on the availability of visual feedback, and of a non-visual signal linked to monitoring of the efferent ocular motor commands or efference copy, that can provide information about how accurate the previous saccade was. The cerebellum receives the efference copy signal for forward control of saccades (17) from the superior colliculus and is responsible for saccade adaptation (18). Thus, like diseases affecting the cerebellum (19), TMS over the cerebellum could disrupt not only the control of eye movements, but also the individual’s ability to correct them, through modulation of cerebellar plasticity.

Moreover, the OMV and FN encode gaze velocity during smooth pursuit and combined eye-head tracking (20,21), and play a critical role both in the immediate online and in the short-term adaptive control of pursuit (22). Unilateral lesion of the OMV in humans impairs ipsilateral smooth pursuit (23). The caudal FN neurons help to accelerate the eyes at the onset of contralateral pursuit. Unilateral inactivation in monkeys decreases the acceleration of contralateral and increases the acceleration of ipsilateral pursuit onset (24).

The anatomically equivalent area of the OMV is located under the inion, and it can be stimulated using a TMS device.

Some fMRI studies showed that the cerebellar hemispheres, too, could contribute to the control of eye movements, in particular memory-guided saccades and coordinated eye-hand movements (25,26). During voluntary saccades made between either two visual targets, or remembered target locations in darkness, fMRI demonstrates increased activation in the hemispheres as well as in the vermis and fastigial nuclei (25,27).

Moreover, lesions restricted to one cerebellar hemisphere impair ipsilateral smooth pursuit (28).

**Single-pulse TMS over the cerebellum and eye movements**

Single-pulse TMS offers advantages in terms of temporal resolution. Allowing interference with brain functioning with a precision in the order of milliseconds, it can be used to investigate the time course of the planning and execution of eye movements. For instance, neural processing in the cerebral cortex, superior colliculus and cerebellum presumably accounts for the 200 msec saccade latency from stimulus appearance. 30° saccades last about 100 msec; saccades show a consistent relationship between their amplitude and peak velocity [main sequence (29)], with asymptotic peak velocity values of about 500°/sec for larger saccades. TMS pulses over the frontal, dorsolateral-prefrontal, and posterior-parietal cortex can modulate saccadic eye movements, and have been used to study the cerebro-cortical control of reflexive and volitional saccades [see (30) for a review].

Amassian et al. (31) found that focal stimulation over the human cerebellum with a figure-of-eight magnetic coil results in an evoked potential recorded from bipolar scalp electrodes reflecting either disfacilitation through TMS activation of PCs, or feed-forward facilitation through transsynaptic or antidromic activation of dentate neurons. When applied over the lateral cerebellum, single-pulse TMS diminishes the excitability of the contralateral motor cortex most likely through transsynaptic activation of the PCs, whose enhanced discharge should, in turn, inhibit the tonic activity of deep cerebellar nuclei neurons and therefore of the entire cerebello-thalamo-cortical pathway (31,32).

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**Table II - Synopsis of studies using transcranial magnetic stimulation over the cerebellum and eye movement recording**

<table>
<thead>
<tr>
<th>Authors</th>
<th>Site</th>
<th>Parameters</th>
<th>Recording device</th>
<th>Paradigm</th>
<th>Parameters</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hashimoto and Ohtsuka, 1995</td>
<td>ocular motor</td>
<td>single pulse</td>
<td>infrared oculography</td>
<td>reflexive saccades</td>
<td>amplitude, latency and peak velocity</td>
<td>hypometric contralateral saccades, hypermetric ipsilateral saccades</td>
</tr>
<tr>
<td>Nagel and Zangemeister, 2003</td>
<td>ocular motor</td>
<td>single pulse</td>
<td>infrared oculography</td>
<td>reflexive saccades</td>
<td>latency and peak velocity</td>
<td>reduced or reversed delay between eye and head movements</td>
</tr>
<tr>
<td>Ohtsuka and Enoki, 1998</td>
<td>ocular motor</td>
<td>single pulse</td>
<td>infrared oculography</td>
<td>smooth pursuit</td>
<td>velocity and acceleration</td>
<td>acceleration of ipsiversive pursuit and deceleration of contraversive pursuit</td>
</tr>
<tr>
<td>Haarmeier and Kammer, 2010</td>
<td>right hemisphere</td>
<td>3 pulses at 10 Hz</td>
<td>video oculography</td>
<td>smooth pursuit</td>
<td>peak velocity</td>
<td>decrease of pursuit gain without modification of perceptual stability</td>
</tr>
<tr>
<td>Jenkinson and Miall, 2010</td>
<td>ocular motor</td>
<td>1 Hz repetitive</td>
<td>infrared oculography</td>
<td>double step saccades</td>
<td>amplitude and peak velocity</td>
<td>impaired saccade adaptation</td>
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**Functional Neurology 2010; 25(3): 165-171**
Reflexive saccades (13) and smooth pursuit (10) were recorded to evaluate the effect of single-pulse TMS over the posterior vermis, aimed at simulating a unilateral lesion of the FN in humans. In both studies, TMS was delivered by a figure-of-eight coil positioned at a right angle to the sagittal plane ~7 mm lateral and caudal to the inion. After the end of the experiments, the stimulation site was identified by MRI on each subject. When triggered after the onset of horizontal visually guided saccades (13), TMS produced hypometric contralateral saccades followed by corrective saccades, and hypermetric ipsilateral saccades followed by post-saccadic drift and significantly greater peak velocity (Fig. 3). These results have been attributed to transient inhibition of the FN, and are similar both to the effects of microstimulation of the vermal cortex at the onset of saccades in monkeys (33-35), and to the pattern of saccadic dysmetria shown in patients with Wallenberg’s lateral medullary syndrome (19,36,37). In the second experiment (10), single-pulse TMS was delivered during the initial acceleration phase and during the steady-state tracking phase of smooth pursuit. TMS caused abrupt acceleration of ipsiversive pursuit and abrupt deceleration of contraversive pursuit in both initial acceleration and steady-state tracking phases. These results suggested that the OMV controls smooth pursuit velocity in a direction-sensitive manner in both initial acceleration and steady-state tracking phases. In these experiments, direct inhibition of the FN could be ruled out, given that the coil used for delivering TMS is believed to be able to activate neurons lying at depths of only 1.5 to 2.0 cm below the surface of the scalp (38,39), corresponding to the cortical layer of the cerebellum. Therefore, the observed effects may have been due to an increase in the inhibitory output of the PCs to the FN, showing a specific effect of the stimulus on the OMV.

Nagel and Zangemeister (11) studied the effect of TMS over the posterior vermis on coordinated eye-head movements. During natural activities gaze shifts larger than 15° are accomplished by eye-head movements, and in the case of unexpected target presentation, the saccade usually starts 200 msec after the appearance of the target and precedes the head movement by about 20-25 msec. For predictable gaze shifts, the head begins to move several hundred milliseconds before the eyes. Since the FN projects to motor neurons in the cervical spinal cord that control voluntary head movements (40), it could have a role in coordinating eye-head movements. TMS was delivered by a circular coil with the centre placed over the inion, with an estimated field...
power of 0.1 V/m at a depth of 3.5 cm. The subjects were asked to perform horizontal reflexive saccades towards visual targets displaced, in a predictable or unpredictable temporal sequence, at an angle of 60°, which should elicit synergistic eye and head movements. The same tasks were repeated while delivering single-pulse TMS over the cerebellum before the start of the eye movements, and at 5, 50 and 100 msec following the displacement of the visual target. TMS caused a reduction of the delay, or a reversal of the timing between the start of the eye movement and the head movement, a decrease of saccadic latency, particularly in the predictable task, and an increase of saccadic peak velocity. The authors concluded that TMS was able to give rise to a transient deficit in the OMV and could thus interfere with the synkinesis of eye and head movements.

Haarmeier and Kammer (14) used single TMS pulses over the cerebellar hemisphere to investigate interactions between smooth pursuit and optokinetic stimuli. Subjects were asked to track a target moving to the right at a constant velocity. When the eyes, while tracking the target, reached the straight-ahead position, a background stimulus was briefly presented in order to induce the erroneous perception of background motion. Three pulses of TMS were applied at 10 Hz during the background presentation. At the end of each trial subjects were required to indicate whether they had perceived the background motion, to the right or to the left. The authors found a decrease of pursuit gain during cerebellar TMS and no modification of motion perception, therefore they hypothesised that perceptual stability is accomplished by subtracting the efference copy of the eye movement motor command from the retinal signal, and concluded that the lateral cerebellum contributes to pursuit, while motion perception is based on a more widely distributed network featuring parallel processing.

Repetitive TMS over the cerebellum and eye movements

rTMS exerts a long-lasting effect on brain function by inducing plastic changes in neuronal excitability possibly mimicking the effect of lesions or pharmacological inactivation. Low-frequency (1 Hz) rTMS over the motor cortex reduces neural excitability, whereas >5 Hz rTMS increases neural excitability [see (41) for a review]. Long-term potentiation (LTP) or long-term depression (LTD)-like mechanisms have been proposed to explain these effects (42). Jenkinson et al. (12) delivered rTMS over the posterior vermis following the completion of a pre-adaptation task (100 reflexive visually guided saccades), and throughout the execution of an adaptive task (100 double-step saccades). The double step is an adaptive paradigm in which saccadic pulse dysmetria is simulated by changing the position of the target before the eye reaches it, thereby inducing the execution of a corrective movement. After about 100 trials the pulse amplitude adapts, allowing the target to be reached with the first saccade (43). The amount of adaptation taking place at the end of the adaptive task was calculated as the difference between the mean amplitude of the final 10 saccades in the pre-adaptation task, and the mean amplitude of the final 10 saccades in the adaptive task. rTMS was delivered by a double-cone coil placed over the inion and directed at the posterior cerebellum. The authors tested three different conditions: no TMS, 120 pulses of 1 Hz rTMS at 45%, and 120 pulses of 1 Hz rTMS at 55% of the maximal intensity. Saccade adaptation was significantly impaired only after the 55% intensity rTMS. No significant effect was seen on the mean peak velocity or on mean amplitude and peak velocity of the reflexive saccades.

Continuous theta burst stimulation (cTBS), i.e. three pulses of 50 Hz stimulation delivered every 200 msec, is a short (about 45 sec) repetitive TMS paradigm that gives rise to long-lasting effects. 1 Hz rTMS and cTBS over the frontal eye fields have inhibitory effects on saccade triggering, resulting in increased saccadic latency that lasts about 8 minutes following 1 Hz rTMS, and up to 30 minutes after cTBS, the decay of the effect being faster after 1 Hz stimulation than after theta burst stimulation (44). When applied over the cerebellar cortex, cTBS reduced excitability of the contralateral motor cortex (45), possibly by interfering with the pre- and postsynaptic connections of the PCs, resulting in inhibition of the deep nuclei and disruption of their excitatory connections to the motor cortex through the ventral thalamus. rTMS delivered over the lateral cerebellum induces long-lasting changes in cortical excitability, resulting in suppression of the cerebello-cortical inhibition lasting up to 30 minutes after 900 pulses of 1 Hz rTMS or 600 stimuli of cTBS (46). Similarly, cTBS over the posterior vermis can exert an inhibitory effect on the FN, resulting in modifications of the reflexive saccades that resemble those described in Wallenberg's syndrome (19), and in unilateral inactivation of the caudal FN in monkeys (34). Furthermore, the cTBS after-effect in the cerebral cortex has been attributed to LTP or LTD phenomena, which are likely to impact directly on the mechanisms of long-term synaptic plasticity (42).

Concluding remarks

The studies here described showed that TMS over the cerebellar hemispheres and over the posterior vermis produces changes in the motor behaviour of the subject which are measurable by means of eye movement recording. Eye movements are easier to interpret than movements of the axial or limb musculature. Moreover, when used to evaluate TMS effect over the cerebellum, eye movements will not be affected by the stimulation of afferent fibres in the back of the neck, which could bias effects achieved by means of TMS and observed on the spinal muscles (47). Two main experimental approaches have been used: the cerebellum was stimulated by single-pulse TMS triggered during the execution of eye movements, in order to interfere with the movement control within a determined period of time (in the order of milliseconds), or by rTMS, in order to study the effects of transient inactivation (in the order of several minutes) of a node of the network subserving a given function. Analysis of reflexive, visually guided saccades showed that the effects of single-pulse TMS over the posterior vermis are similar to those obtained by unilateral inactivation of the caudal FN (34). Given the specificity of these findings, reflexive saccades proved to be suitable
for studying the effect of TMS on cerebellar control of eye movements. Voluntary eye movement paradigms could be used to better define cerebellar control of reflexive and voluntary eye movements. A previous study has shown that saccadic dysmetria resulting from cerebellar lesions may spare volitional saccades in a scanning paradigm (48), yet data regarding the effect of cerebellar dysfunction on the accuracy of memory guided saccades (49-51) are contradictory. An interesting approach could be that of studying cognitive functions and plasticity at cerebellar level in subjects involved in volitional paradigms that involve procedural memory, working memory, and learning.

All the studies described in this review were conducted in healthy subjects; the application of TMS in patients promises to be a good tool not only to investigate cerebellar physiology, but also to modulate cortical excitability disorders underlying specific deficits. With regard to TMS parameters and techniques, single-pulse TMS allows definition of the temporal sequence in which different central structures are involved in the control of eye movements, while rTMS could be used to affect cerebellar plasticity. Since the rTMS effect outlasts the stimulation period, its consequences could be measured without interfering with either the eye movement execution or the experimental procedures. Several forms of long-term synaptic plasticity that are known to exist in the cerebellum (52,53) and subtend eye movement adaptation (16,54,55) could be influenced by cTBS without exerting a direct effect of the stimulus on the movement execution and recording procedures. For this purpose, further studies could aim at defining the time frame of the effect of rTMS over the cerebellum on eye movements. Transcranial direct current stimulation (tDCS) has been tested as a method for modulating cerebellar excitability in humans (56). Cathodal tDCS applied over the cerebellar hemisphere with a stimulation intensity of 2mA can increase the inhibitory tone exerted by the cerebellum over the primary motor cortex and this effect lasts up to 30 minutes after the cessation of stimulation. Thus, tDCS could also be used in conjunction with eye movement recording.

Finally, the cellular mechanism by which TMS and rTMS influence cerebellar excitability is not yet understood. Additional research addressing the mechanisms of the induced changes and the synaptic mechanisms involved in these circuits is therefore required.

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