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CLINICAL REVIEW

Distinctive patterns of cortical excitability to transcranial magnetic stimulation in obstructive sleep apnea syndrome, restless legs syndrome, insomnia, and sleep deprivation

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SUMMARY

Altered responses to transcranial magnetic stimulation (TMS) in obstructive sleep apnea syndrome (OSAS), restless legs syndrome (RLS), insomnia, and sleep-deprived healthy subjects have been reported. We have reviewed the relevant literature in order to identify eventual distinctive electrocortical profiles based on single and paired-pulse TMS, sensorimotor modulation, plasticity-related and repetitive TMS measures. Although obtained from heterogeneous studies, the detected changes might be the result of the different pathophysiological substrates underlying OSAS, RLS, insomnia and sleep deprivation rather than reflect the general effect of non-specific sleep loss and instability. OSAS tends to exhibit an increased motor cortex inhibition, which is reduced in RLS; intracortical excitability seems to be in favor of an "activating" profile in chronic insomnia and in sleep-deprived healthy individuals. Abnormal plasticity-related TMS phenomena have been demonstrated in OSAS and RLS. This review provides a perspective of TMS techniques by further understanding the role of neurotransmission pathways and plastic remodeling of neuronal networks involved in common sleep disorders. TMS might be considered a valuable tool in the assessment of sleep disorders, the evaluation of the effect of therapy and the design of non-pharmacological approaches.

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Introduction

Transcranial magnetic stimulation (TMS), first introduced by Barker et al. in 1985 [1], is a painless and non-invasive neurophysiological technique specifically capable of assessing the primary motor cortex (M1) and the cortical–spinal tract excitability *in vivo*. In the last years, several TMS studies have been carried out to evaluate the neurophysiological pattern of cortical excitability in different sleep disorders, including obstructive sleep apnea syndrome (OSAS), restless legs syndrome (RLS), insomnia as well as experimentally sleep-deprived healthy subjects.

However, although the findings from these reports seem to reveal substantial changes of cortical excitability compared to healthy good sleepers, the complexity and heterogeneity of sleep disorders, the relatively low number of investigations and the heterogeneity in the methods employed preclude a comprehensive understanding. Studies assessing whether these changes might be distinctively related to the underlying pathophysiologic mechanisms of the different sleep disorders or they merely reflect the effect of disturbed nocturnal sleep are missing. It is well known, indeed, that sleep loss and instability are common features of OSAS [2,3], RLS [4,5] and insomnia [6,7].

When considering the wide spectrum of sleep loss disorders, it is useful to clarify the definition of different categories of sleep loss, such as total or partial sleep deprivation (SD), sleep fragmentation (SF) and insomnia. SD involves getting less than a sufficient amount of sleep and can include an acute total sleep-restricted state and a chronic partial sleep restriction state, as the complete absence of sleep over long periods is impossible for healthy humans. Total SD

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Abbreviations

CMCT	central motor conduction time
CSP	cortical silent period
EEG	electroencephalography
EMG	electromyography
GABA	gamma-aminobutyric acid
ICF	intracortical facilitation
ISI	interstimulus interval
LAI	long-latency afferent inhibition
LTD	long-term depression
LTP	long-term potentiation
M1	primary motor cortex
MEP	motor evoked potential
MRI	magnetic resonance imaging
MT	motor threshold
NMDA	N-methyl-D-aspartate
OSAS	obstructive sleep apnea syndrome
PAS	paired associative stimulation
REM	rapid eye movement
RLS	restless legs syndrome
rMT	resting motor threshold
rTMS	repetitive transcranial magnetic stimulation
SAI	short-latency afferent inhibition
SD	sleep deprivation
SF	sleep fragmentation
SICI	short-latency intracortical inhibition
TBS	theta burst stimulation
TMS	transcranial magnetic stimulation

Glossary of terms

Transcranial magnetic stimulation (TMS)
: non-invasive and painless neurophysiological technique specifically able to evaluate the excitability of motor cortical area and the cortical–spinal pathways conductivity through the administration of magnetic stimuli on the scalp.

Motor evoked potential (MEP)
: muscular response obtained after a single TMS pulse applied over the contralateral primary motor cortex at appropriate stimulation intensity.

MEP latency
: time interval between the administration of the TMS pulse on the motor cortex and the onset of the MEP from the contralateral target muscle; it reflects the conductivity of both the central and peripheral nervous systems, as well as neuromuscular junctions and muscles.

MEP amplitude
: it mainly reflects the excitation state of output cells in the motor cortex, nerve roots and the conduction along the peripheral motor pathway to the muscles.

Central motor conduction time (CMCT)
: latency difference between the MEPs induced by stimulation of the motor cortex and those evoked by spinal (motor root) stimulation; it reflects the integrity of the cortical–spinal tract, from the upper to the lower motor neurons.

Motor threshold (MT)
: lowest TMS intensity necessary to evoke MEPs in the target muscle when single-pulse stimuli are applied to the motor cortex, at rest (resting MT) or during contraction (active MT); it is a global measure of cortical excitability reflecting the excitability of cortical–spinal neurons and interneurons projecting onto these neurons in the motor cortex as well as of spinal motor neurons, neuromuscular junctions and muscle.

Cortical silent period (CSP)
: inhibitory MEP recorded during a sustained voluntary contraction of the target muscle followed by a suppression of the electromyographic activity evoked by a suprathreshold TMS stimulus applied to the contralateral motor cortex.

Paired-pulse transcranial magnetic stimulation
: TMS paradigm allowing to study intracortical inhibitory and excitatory phenomena by means of a conditioning subthreshold stimulus preceding a suprathreshold test stimulus by a programmable interstimulus interval.

Short-latency intracortical inhibition
: paired-pulse TMS measure obtained at short interstimulus interval in which the conditioning stimulus determines an inhibition with respect to the test stimulus; it is attributed to an activation of inhibitory neuronal system transmission.

Intracortical facilitation (ICF)
: paired-pulse TMS measure obtained at long interstimulus interval in which the conditioning stimulus determines an enhanced response with respect to the test stimulus; it is modulated by multiple neurotransmission pathways.

Short-latency afferent inhibition (SAI)
: inhibitory MEP resulting from an electric conditioning pulse applied on a peripheral nerve that precedes cortical TMS by a short interstimulus interval; it is considered as a putative marker of central cholinergic activity and allows to investigate the sensory-motor interaction within the cerebral cortex.

Long-latency afferent inhibition (LAI)
: as for SAI, but at longer interstimulus interval; in addition to the direct effects on MEP amplitude it also interacts with other cortical inhibitory circuits.

Paired associative stimulation (PAS)
: protocol consisting of slow-rate repetitive low-frequency nerve stimulation combined with TMS over the contralateral motor cortex; it has been shown to induce plastic changes of excitability in the human motor cortex.

Repetitive transcranial magnetic stimulation
: train of TMS pulses of the same intensity applied to a single brain area at a given frequency, that can transiently influence the function of stimulated and connected brain areas, mainly depending on the frequency of stimulation.

Theta burst stimulation (TBS)
: a form of high frequency repetitive TMS that, when applied to motor cortex, leads to after-effects on cortical–spinal and cortical–cortical excitability and that may reflect synaptic plasticity effects.

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