APŽVALGINIS STRAIPSNIS

Transcranial magnetic stimulation in clinical practice

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Key words: *cortico-spinal conduction, motor evoked potentials, electrophysiology, neurophysiology, cortical silent period.*

Summary. *Transcranial magnetic stimulation allows a non-invasive and painless stimulation of the human brain and cranial nerves. The method is in use since 1985. Transcranial magnetic stimulation can use single stimuli, pairs of stimuli separated by different intervals (to the same or to several brain areas), or trains of repetitive stimuli at various frequencies. Single stimuli give rise to motor evoked potentials that have clinical use and serve diagnostic and prognostic purposes. Repetitive transcranial magnetic stimulation can modify excitability of cerebral cortex. Repetitive transcranial magnetic stimulation has opened a new field of investigation of the neural circuitry, and is developing into a therapeutic tool.*

This general review considers basic principles of transcranial magnetic stimulation, discusses methodological aspects and techniques, and analyses their utility in clinical practice.

Introduction

In the early 1980s, P. A. Merton and H. B. Morton showed that high voltage electrical stimulation over the scalp was able to activate the motor cortex in man, evoking twitch-like movements in the corresponding muscles (1). This technique was used to investigate the central motor pathways in normal subjects and in patients with various neurological disorders. However, transcranial electrical stimulation was uncomfortable and even painful for patients. Therefore, this technique was not ideal for routine clinical practice.

In 1985, A. T. Barker and colleagues introduced the painless technique of transcranial magnetic stimulation (TMS), which led to a new era of research in motor control and cortical function (2). Since that time, interest in TMS has steadily increased.

This article considers concepts of TMS, reviews different techniques, including the new field of repetitive transcranial stimulation (rTMS), and analyzes their present and potential use in clinical practice.

Motor effects of brain stimulation

When the human brain is stimulated transcranially, a complex sequence of events ensues with excitatory and inhibitory effects. These effects depend on the stimulus intensity and on the excitability of the cortex and spinal cord. Investigation of inhibitory and excitatory neuronal circuits within the motor cortex is made available.

The technical principle of TMS is to pass a brief surge of current through a coil, which induces a rapidly changing magnetic field. This magnetic field passes into the surrounding medium, where it again induces an electrical field. Applied over the human scalp it excites cortical neurons (Fig. 1).

Using a circular coil with the coil current flowing clockwise when viewed from above, the left hemisphere will be excited preferentially. Turning the coil over so that current now flows anticlockwise, the right hemisphere will be excited preferentially. With a figure-eight coil, the central linear segment should be over the motor area. For small hand muscles the optimal orientation of this coil has been determined as about 45° to the parasagittal plane with coil current flowing postero-anteriorly (3, 4). Whereas, peripheral nerve stimuli, when maximal, excite all motor axons and evoke compound muscle action potentials (CMAPs) with latencies and sizes that do not vary if stimulation is repeated, transcranial stimuli evoke multiple descending volleys in corticospinal neurons. The initial volley – the direct (D) wave – is thought to arise from excitation of the pyramidal cell. This D-wave is follo-

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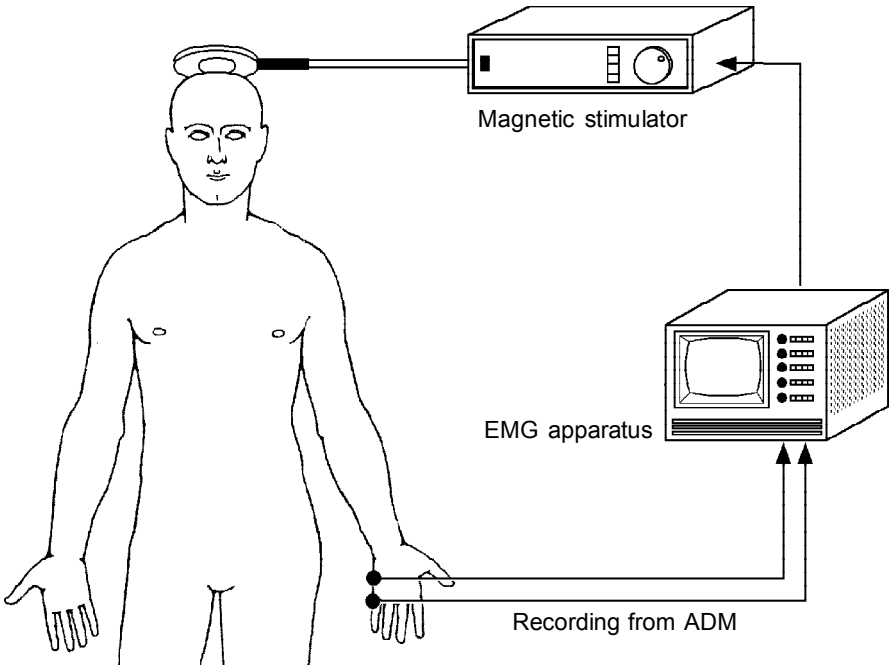

Fig. 1. **Scheme of a transcranial magnetic stimulation set-up**
A magnetic stimulator, with use of a coil (circular) placed over the motor cortex, is triggered by an EMG apparatus which also serves to record a motor evoked potential from a muscle (here from abductor digiti minimi – ADM).

wed by a number of indirect (I) waves at 1.5 to 2 ms intervals. I-waves possibly stem from transsynaptic excitation of corticospinal cells by different sets of intracortical neurons (5). Motor evoked potentials (MEPs) vary in latency and size from one stimulus to another. If the target muscle voluntarily contracts, "facilitation" ensues. The MEP of the contracting muscle has a shorter latency and larger amplitude. Thus, with an appropriate position of the stimulating coil, facilitation causes a focal response to the magnetic stimulation that is rather diffuse over the scalp (6, 7).

Assessment of cortico-spinal tract conduction

Central motor conduction time

The conduction time from motor cortex to spinal cord alpha-motoneurons is referred as the central motor conduction time (CMCT). It consists in the difference between conduction time from cortex to muscle and peripheral motor conduction time. Calculation of the peripheral motor conduction time can use the F-wave latency (8), electrical (9) or magnetic (10) stimulation of the spinal nerve roots. It is recommended to measure the CMCT while the target muscle contracts at 5% to 20% of its maximum strength (11), because the MEP size saturates for stronger contractions (12). Facilitation is better during phasic contraction than during a steady isometric contraction. The CMCT to the active muscle is shorter by 2–3 ms than to the resting muscle (12). The CMCT is also affected by the position of the stimulating coil. The shortest CMCT is being obtained when the coil is placed at the optimal position for eliciting MEP in the target muscle. Finally, the CMCT also depends on the direction of TMS induced current in the motor cortex.

Normative CMCT data in adults are available for many muscles of the upper and lower limb, and for cranial muscles (13). The main reasons for pathological CMCT lengthening are demyelination of the corticospinal fibers and degenerative or ischemic changes. CMCT measurements are of interest in central demyelinating disorders (*e.g.* multiple sclerosis), cerebral ischemic stroke, myelopathies and neurodegenerative diseases affecting the corticospinal tract (14–17). In these disorders, CMCT may be useful in disclosing changes before clinical manifestation occurs.

Motor evoked potentials size

When TMS is applied to the motor cortex at appropriate stimulation intensity, MEPs can be recorded from muscles of the contralateral extremity (11, 18). If the peripheral nervous system is normal, normal amplitude of the MEP reflects the integrity of the corticospinal tract and also normal excitability of motor cortex and alpha-motoneurons. Patients with dysfunction of any of the above may have MEPs of reduced

size. A difficulty to estimate an abnormal reduction of the size stems from the marked variability of the size of MEPs observed in healthy people. This variability, due to dispersion of the alpha-motoneuron response to the descending volley in the corticospinal tract, leads to a broad range of normal values. This problem has been solved by the "triple stimulation technique" (see "Non-standard methods" below) (7).

Assessment of motor cortex excitability

Motor threshold

Motor threshold may be defined as the lowest intensity required to elicit MEPs of more than 50 μV amplitude in at least 50% of successive trials in resting or activated target muscles (19). Measurement of the threshold is used as a marker of cortico-spinal excitability. A high motor threshold may indicate significant damage of the corticospinal tract after cerebral stroke or spinal cord lesion (14, 20, 21). The inability to elicit MEP in an acute stroke patient predicts a poor functional outcome (22). A low motor threshold suggests increased corticospinal tract excitability; it has been observed in different disorders such as in idiopathic generalized epilepsy, obsessive-compulsive disorder and in early amyotrophic lateral sclerosis (ALS) (23). Patients with ALS show lower motor threshold and increased excitability of hand motor area at an early stage of their disease where hand muscle function remains normal. When the disease progresses and lower motor neuron (or mixed upper and lower) signs appear in the hand muscles, the motor threshold rises (24). Motor threshold is of limited use as a single study in a patient due to large variability between subjects, but longitudinal measurements are feasible.

Cortical silent period

When a subject is requested to maintain a muscle contraction, TMS causes a suppression of the electromyographic activity after the MEP. This period of electromyographic "silence" has been termed the *silent period* (SP) (Fig. 2). It may have an interest in the study of epilepsy, cerebral stroke, movement disorders, ALS, migraine and tetanus (25–28).

The SP observed in ipsilateral muscles can be used to measure transcallosal conduction (see "Non-standard methods" below) (25, 26).

Intracortical inhibition and intracortical facilitation

Inhibitory and facilitatory interactions that appear to take place within the cortex can be studied by combining a subthreshold conditioning stimulus with a suprathreshold test stimulus at different short interstimulus intervals through the same TMS coil (29).

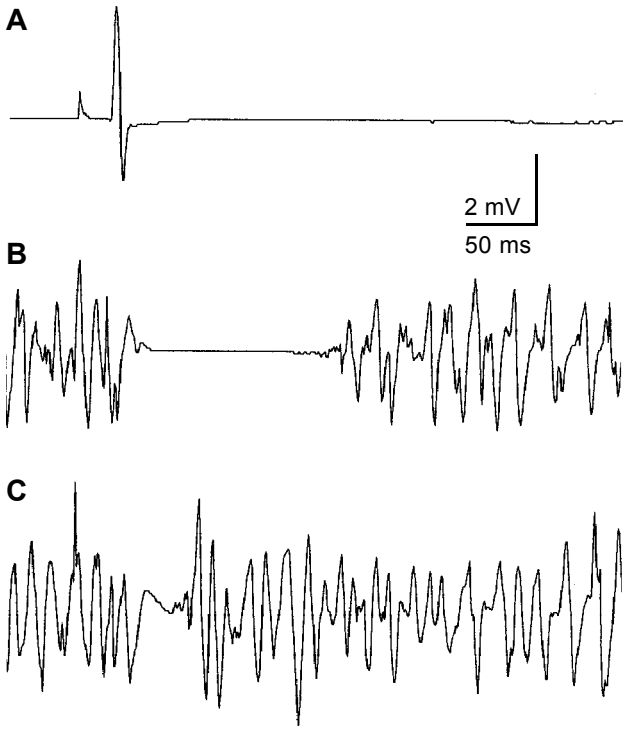

Fig. 2. **Cortical silent period**

A – motor evoked potential at rest; **B** – a magnetic stimulus performed over the contralateral motor cortex stops the ongoing EMG voluntary activity (from abductor digiti minimi), giving rise to a "cortical silent period"; **C** – a magnetic stimulus performed over the ipsilateral motor cortex stops shortly the EMG voluntary activity, giving rise (via transcallosal pathways) to an "ipsilateral cortical silent period".

This paired-pulse technique requires a special set-up, because a standard magnetic stimulator cannot discharge more than once every 2 to 3 seconds. Intracortical inhibition (ICI) is observed for interstimulus intervals between 1 to 5 ms (29, 30), intracortical facilitation (ICF) for intervals between 7 to 20 ms (31, 32) (Fig. 3). ICI and ICF are controlled through the GABA-a and N-methyl-D-aspartate (NMDA) receptors. GABA-a agonist (benzodiazepine) and NMDA antagonist (memantine) increase ICI and decrease ICF (33). Furthermore, several neuromodulating drugs with effects on the systems of dopamine, norepinephrine, serotonin and acetylcholine affect ICI and ICF (31).

Paired-pulse techniques have not entered clinical routine yet. Potential applications of these techniques are broad. Several studies have been conducted in epilepsy, cerebral stroke, movement disorders, ALS, migraine (27, 32, 33). Most of these disorders show a decrease in ICI and/or an increase in ICF. Therefore, although sensitive for the detection of abnormalities of motor cortex excitability, ICI and ICF changes are

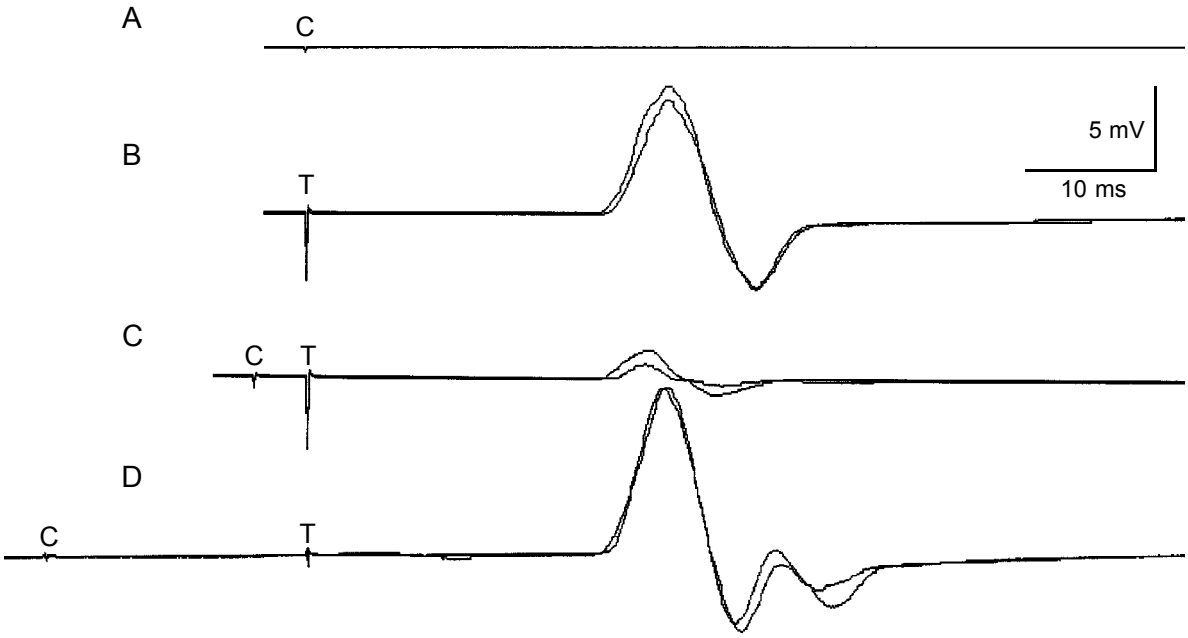

Fig. 3. **Intracortical inhibition and facilitation with paired-pulse technique**
A – conditioning stimulus (C) alone; **B** – test stimulus (T) alone gives rise to a motor evoked potential (MEP); **C** – C+T with 3 ms interval gives rise to a MEP of smaller size than in B due to "intracortical inhibition"; **D** – C+T with 20 ms interval gives rise to a MEP of larger amplitude due to a "intracortical facilitation".

not specific. Furthermore, disorders without clear motor cortex pathology, such as schizophrenia or depression, have been found to be associated with changes in TMS paired-pulse curves, hence raising further questions about the specificity of the findings (34–36).

Investigation of interhemispheric interaction

Paired-pulse stimulation technique can also refer to the application of single stimuli to two different brain regions. A first conditioning stimulus is given to a motor cortex area and after a short interval a second, test stimulus, is applied to another motor cortex area in order to examine interregional or interhemispheric interactions and transcallosal conduction times (37). They are influenced by the intensity of the conditioning TMS, with stronger conditioning TMS pulse inducing greater and longer interhemispheric inhibition.

The interhemispheric influence of the left dominant hemisphere is more pronounced in right-handed people (38). This technique allows the investigation of interhemispheric interactions in motor control and movement disorders (39, 40). Further studies may establish this paired-pulse method as a diagnostic tool to elucidate mechanisms of pathological interhemispheric and intracortical interactions in neurological and psychiatric diseases. This should expand our understanding of disconnection syndromes, in cognition, and in diseases.

TMS methods in clinical practice

Both standard and non-standard methods are used in the investigation of patients presenting with neurological disorders (Table).

Standard methods

The size of MEPs is measured on neurographic recordings. The amplitude of the negative phase (in mV) may be expressed as a percentage of the amplitude of the maximum M-wave recorded from the same muscle following supramaximal electrical stimulation of the corresponding peripheral nerve. A reduced size ratio is suggestive of either a reduced excitability of the cortico-spinal motoneurons, or a conduction block on the cortico-spinal tract, or a loss of cortical motoneurons or axons.

The MEP latency is measured in milliseconds from the stimulus artifact to the motor response onset. To assess conduction along the corticospinal tract the CMCT is determined by subtracting the peripheral conduction time. Increased CMCT indicates slowing of conduction of descending impulses, or loss of fast conducting axons.

Non-standard methods

The triple stimulation technique (TST) provides a quantitative electrophysiological measurement of central motor conduction failures (7). This technique involves three stimuli (transcranial, distal and proximal on the peripheral nerve) timed to produce two

Table. **Variables of transcranial magnetic stimulation in neurological disorders**

Neurological disorder	MEP amplitude	CMCT	MTh	SP
Multiple sclerosis	Reduced	Increased	Increased	Prolonged
Stroke	Reduced	Increased	Increased or reduced	Shortened
Cervical myelopathy	Reduced	Increased	Increased	Shortened
Amyotropic lateral sclerosis	Reduced	Increased	Reduced (early) increased (late)	Normal or shortened
Parkinson's disease	Facilitated at rest	Normal	Normal	Shortened
Dystonia	Normal	Normal	Normal	Shortened
Cerebellar ataxias	Normal or reduced	Increased	Increased	Prolonged
Epilepsies	Normal or reduced	Normal	Normal, reduced or increased	Prolonged

MEP – motor evoked potential, CMCT – central motor conduction time, MTh – motor threshold, SP – silent period.

collisions. The TMS descending impulses collide with the antidromic impulses from the distal stimulus. The third stimulus, proximal on the nerve, evokes orthodromic impulses, which cancel any uncollided impulses from the distal stimulus. The response from the third stimulus therefore reflects the number of peripheral neurons activated from TMS. By suppressing the phase cancellation due to the dispersion of the MEP, the TST is markedly more sensitive than conventional MEPs in detecting corticospinal conduction failures and it provides a precise assessment of corticospinal tract conduction (41) (Fig. 4).

The cortical SP consists in an inhibition of voluntary activity in a target muscle contralateral to the stimulated hemisphere. It is defined as the time interval from the end of the MEP to the return of voluntary electromyographic activity (25, 26). The silent period associates an inhibition of the spinal motoneuron (early part), and of the cortical motoneuron (late part) (25).

When TMS is applied to the motor cortex ipsilateral to the target muscle, an "ipsilateral silent" period can be recorded (42). This silent period is mediated mainly via transcallosal pathways. In the patients with lesions in the corpus callosum, this inhibition is delayed or absent (42, 43). This transcallosal technique adds functional information to the anatomical information provided from MRI studies in patients with multiple sclerosis (44). In multiple sclerosis, the involvement of the corpus callosum can be associated with a poor prognosis regarding cognitive functions (45). This TMS method can be associated with the paired-pulse TMS technique to investigate further interhemispheric interactions.

Intracortical excitatory or inhibitory mechanisms can be analyzed by using paired-pulse techniques. TMS methods testing input-output curves, mapping of cortical muscle representation, interhemispheric inhibition and central fatigue are not commonly applied in clinical practice.

TMS in clinical neurology

Multiple sclerosis

In multiple sclerosis (MS), the central white matter lesions disseminated in time and space, frequently affect both cortico-nuclear and cortico-spinal conduction. Cortico-motoneuronal function can be assessed by studying MEPs to cranial and peripheral muscles. Various abnormalities can be observed in MS that relate to demyelination and to axonal loss (46–48). Demyelination of central motor pathways induces slowed conduction or conduction block. The latency of MEPs can be prolonged and the response may be dispersed, of smaller size, or absent. A reduced MEP size may indicate a central conduction deficit, but this relation is obscured by the desynchronization of the descending action potentials in response to TMS. The TST

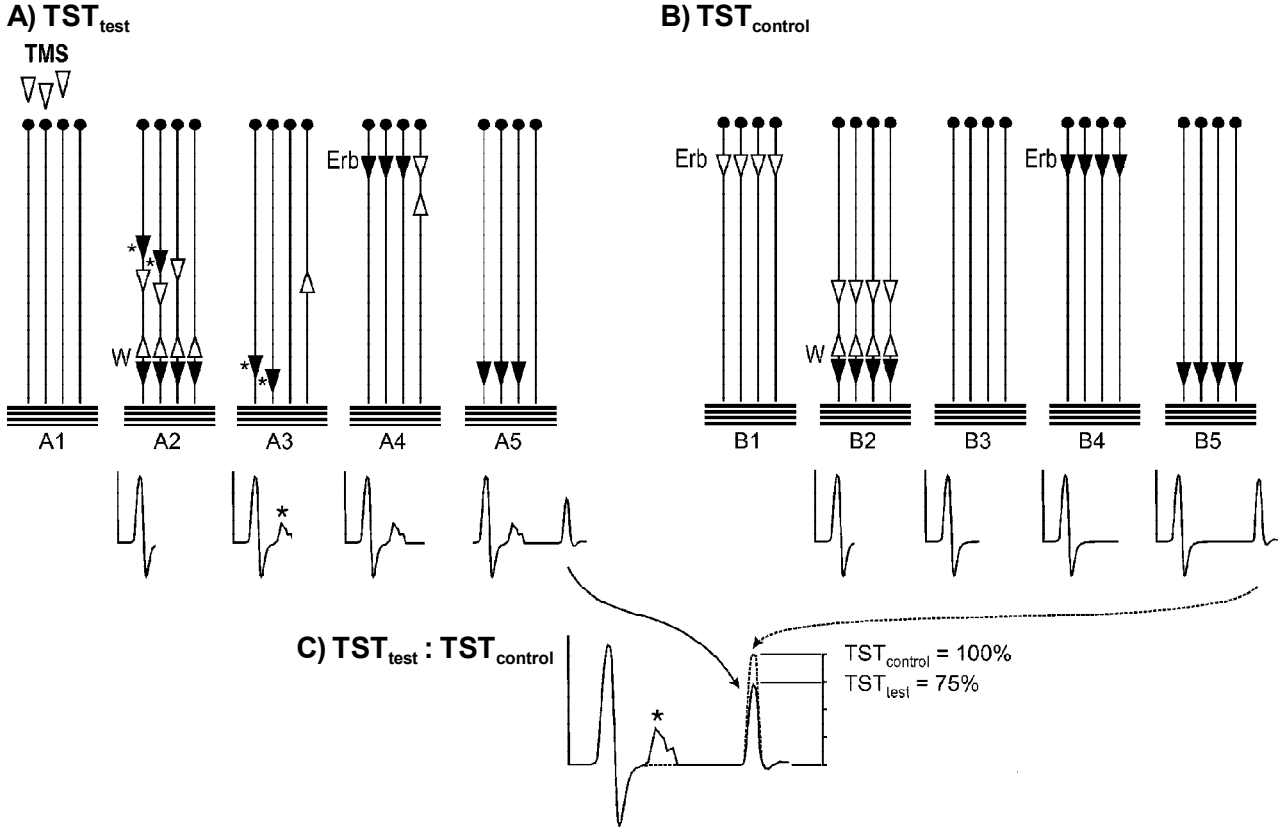

Fig. 4. **Scheme of the triple stimulation technique (TST)**

The motor tract is simplified to four spinal motor neurons with their axons. Horizontal lines represent the muscle fibers of the four motor units. Solid arrows depict action potentials giving rise to a trace deflection, open arrows depict action potentials that are not recorded. **A1** – in the example, only three of four motor neurons are brought to discharge by the brain stimulus due to upper motor neuron lesion; **A2** – following the brain stimulus, action potentials descend in axons 1–3. Desynchronization of the three action potentials has occurred. Motor neurons 1 and 2 discharge twice so that a second action potential descends (*). After a delay, a maximal second stimulus is given at the wrist (W), leading to descending (orthodromic) action potentials causing a first negative deflection of TST_{test} curve, and to ascending (antidromic) action potentials in all axons. Three of the ascending action potentials collide and cancel with the action potentials descending in axons 1–3. The sites of collision are different due to the desynchronization of the descending action potentials; **A3** – the multiple discharges (*) on motor neurons 1 and 2 are not cancelled and continue to descend. They give rise to a small deflection in the trace (*). The action potential on axon 4 continues to ascend, since no collision occurred; **A4** – after a delay, a maximal third stimulus is given at Erb's point, evoking action potentials, which descend on axons 1–3, while a collision occurs in axon 4; **A5** – finally, a synchronized response from the three axons (1–3), which were initially excited by the transcranial stimulus, is recorded as a second main deflection of the TST_{test} curve; **B1-B5** – the $TST_{control}$ curve is recorded by replacing the first stimulus at the cortex by a supramaximal stimulus at Erb's point (succession of stimuli: Erb-wrist-Erb) with appropriate adjustments of the delays; **C** – superimposition of TST_{test} and $TST_{control}$ curves. The TST amplitude ratio is 75%, indicating that three of four neurons were excited by the transcranial stimulus (from Rösler and Magistris, Handbook of Clinical Neurophysiology, Eisen Ed., 2004).

that eliminates these effects allows quantification of conducting central motor neurons. Thereby, it increases the sensitivity to detect a central motor conduction deficit (41). The motor threshold can be moderately increased in MS. The silent period is usually prolonged (49). Data that concern cortical excitability changes seem of little clinical value. Abnormalities of interhemispheric inhibition may be observed, that reflect demyelination or axonal lesions of corpus callosum fibers (50). The combination of CMCT and transcallosal inhibition data may be useful to estimate the disease progression and prognosis (51).

Stroke

In stroke patients with hemiplegia, MEPs after cor-

tical stimulation of the damaged hemisphere are often absent. Low amplitude MEPs with increased motor threshold and prolonged CMCT can be observed in patients with paresis (52). TMS is a good predictor of stroke outcome. During the early stage, obtainable MEPs correlate with a favorable outcome, whereas absent responses predict a poor recovery (53, 54).

ICI mechanisms may be modified in stroke patients, for instance: ICI was found to be reduced in the affected hemisphere, a shorter SP duration was reported after lesion of the primary motor cortex, whereas SP duration was prolonged in patients with subcortical or nonprimary motor areas involvements (28).

Amyotrophic lateral sclerosis

In amyotrophic lateral sclerosis (ALS) patients, MEPs are often of reduced size or absent. This relates to the inexcitability or to the lesion of cortical or spinal motoneurons, or both. CMCT can be prolonged in ALS but the degree of prolongation is usually modest (55). The TST is of interest in detecting and quantifying the central conduction deficit while simultaneously yielding information concerning the peripheral motoneuron (41, 56). Information on the responses of single spinal motoneurons to the corticospinal input in ALS reveals evidence of reduced firing frequency in corticospinal fibers with consequent impaired temporal summation of the motoneurons (57).

Cervical spondylotic myelopathy

Cervical spondylotic myelopathy (CSM) is characterized by a marked and early CMCT prolongation. Sometimes clinically, and with routine electroneuromyography (ENMG) examination, distinction between CSM and ALS may be difficult. These disorders, that impair both upper and lower motoneurons, may share similar clinical features, including muscle wasting and fasciculations. TMS enables to distinguish these disorders. CMCT is usually more prolonged in CSM than in ALS, however this may not be discriminative in an individual patient. Studies performed on the muscles spared in CMS but concerned in ALS such as the masseter (58) or the trapezius muscles (59) are helpful for this distinction.

Parkinson's disease

CMCT is normal in Parkinson's disease and other movement disorders. Motor threshold can be reduced, especially in patients with predominant rigidity in whom there is an enhanced facilitatory effect of voluntary contraction. Moreover, the SP has been shown to be shorter in Parkinson's disease patients (60), whereas it is lengthened by L-DOPA therapy, not only in Parkinson's disease patients, but also in healthy subjects (61). One presumes that in Parkinson's disease there is a reduced basal ganglia inhibition of the motor cortex leading to a shorter than normal SP and that this disbalance is corrected by L-DOPA. The tonic effect of thalamic output on motor cortex excitability has been studied in a patient undergoing thalamotomy for hemiparkinsonism. The facilitatory effect of a voluntary contraction was enhanced and the SP lengthened after thalamotomy (62).

Dystonia

In secondary dystonias a prolonged CMCT has been reported (63). Cortical motor threshold and MEP amplitude are normal at rest, but MEP size increases more steeply in patients than in control subjects with increasing levels of muscle contraction or stimulus intensities. Additionally, an abnormal size and location of cortical representation of the dystonic muscles has been consistently reported (reversed by botulinum toxin injections). These findings suggest the occurrence of abnormalities in the excitability or plasticity of motor cortical areas in dystonia. The SP duration is shorter than in normal subjects (64). ICI at short interstimulus intervals of paired-pulse is reduced at rest (65) and normalized after botulinum toxin injection (66). ICI at long interstimulus intervals is increased during contraction. Overall, TMS findings reflect hyperexcitability of motor cortex areas in focal dystonias.

Cerebellar disorders

Longer CMCT and higher than normal motor threshold have been described in various spinocerebellar ataxias and in other cerebellar degenerations. The occurrence of prolonged cortical SP suggests a reduced cortical excitability, possibly related to the enhancement of inhibitory activities (67).

Epilepsy

TMS has been used to study generalized and focal epilepsies. Different results probably relate to the multiform types of epilepsies, the presence of drugs and the different techniques used. The most common abnormality in the motor cortex of patients investigated with paired-pulse TMS, is an increased excitability with a reduction of intracortical inhibitory mechanisms (68). Motor threshold and MEP amplitude are also variable in different forms of epilepsies. TMS proved useful to test the mode of action and the responsivity to antiepileptic drugs (68, 69).

Facial palsies

The clinical and electrophysiological spectrum of facial palsies is broad and differential diagnosis may be difficult. The lesion of the facial nerve frequently lays within the skull, where the nerve is not accessi-

ble to conventional electrical stimulation. TMS changed this situation, because the proximal intracranial part of the facial nerve and the contralateral hemisphere facial associated cortex became accessible to stimulation (70, 71). This gave new insights into the dynamics and pathophysiology of facial palsies (72). In idiopathic facial palsy, an absent response of the facial nerve to TMS may be observed on the clinically affected side, and may follow the palsy long after clinical recovery (73). Such particular patterns of electrophysiological abnormalities are suggestive of the etiology of different facial palsies (72).

Repetitive transcranial magnetic stimulation

The technique of repetitive transcranial magnetic stimulation (rTMS) allows cortical motor areas to be activated with trains of stimuli evoking successive MEPs. Trains of stimuli at various frequencies and intensities induce excitatory and inhibitory effects both during and after the train.

Effects of repetitive brain stimulation

The effects on cortical excitability during the trains of rTMS can be evaluated by measuring the size and threshold of MEPs (74–76) and the duration of the SP (77, 78).

The effects that follow the trains of rTMS can be evaluated by studying intracortical inhibition and facilitation. Trains of rTMS can induce short-term changes in cortical excitability – immediately after the train (75, 79), and long-term changes of cortical excitability. This effect may range from inhibition to facilitation, depending on the stimulation frequency. Lower frequencies of rTMS, in the 1 Hz range, can suppress excitability of motor cortex (80–82), while 20 Hz stimulation trains seem to lead to a temporary increase in cortical excitability (75, 82, 83). While these effects vary between individuals (82, 83), the effect of low frequency rTMS is robust and long lasting (79, 82) and can be applied to the motor cortex and to other cortical regions to study brain-behavior relations.

Several studies in humans that combined rTMS and functional neuroimaging techniques have detected suppressed or increased cerebral blood flow and metabolism in the stimulated area after slow (1 Hz) or rapid (10–20 Hz) rTMS of the motor cortex (84). The combination of TMS and neuroimaging can be most helpful in the investigation of functional connectivity among regions in the human brain (85). Moreover, the combination of rTMS with tracer PET or magnetic resonance spectroscopy may become a novel tool to investigate neurochemical functional anatomy (85, 86).

rTMS in clinical neurology – therapeutic use

The lasting modulation of cortical activity by rTMS is not restricted to motor cortical areas and long-term effects of rTMS can be induced in visual (87), prefrontal (88), parietal cortex (89) and in the cerebellum (90). This finding raises the possibility of therapeutic applications of rTMS in case of pathologically decreased or increased cortical excitability.

rTMS in the treatment of depression

Effect on depression is the most thoroughly studied therapeutical application of rTMS (91, 92). Both high frequency stimulation of the left dorsolateral prefrontal cortex, and low frequency stimulation of the right side can improve depression. T. A. Kimbrell and colleagues (93) suggested that patients with decreased cerebral metabolism might respond better to high frequency, and those with hypermetabolism may respond better to low frequency stimulation. This fits with the frequency-dependent effects of rTMS on the motor cortical excitability.

Parkinson's disease

A. Pascual-Leone and colleagues (94) first reported that in patients with Parkinson's disease subthreshold high frequency rTMS to the motor cortex improved contralateral hand function. There are two applications of this method in Parkinson's disease: increasing cortical excitability to thalamocortical drive, which is believed to be lacking in this disease and modifying catecholamine metabolism subcortically through cortical stimulation (95, 96). Different studies have shown contradictory results for rTMS in patients with Parkinson's disease (97) that draws attention to the difficulty of proving a clinical therapeutic effect and variability of TMS effects across individuals.

Epilepsy and related disorders

Some investigators have attempted to use low frequency rTMS to treat seizure disorders and other manifestations of cortical hyperexcitability, but effects were transient and controversial (98, 99).

Stroke

Attempts have been made to influence favorably outcome after stroke by rTMS suppressing maladaptive cortical plasticity and improving adaptive cortical activity to neurorehabilitation (100). It is premature to propose such trials as realistic therapeutic applications (101, 102). However, rTMS of the region of interest detected in functional images could highlight the property of plastic changes of the cortical circuitry and hint at future novel clinical interventions.

Conclusions

Transcranial magnetic stimulation introduced 20

years ago has developed as an interesting non-invasive tool for neuroscience research. It is an effective diagnostic tool that carries potential therapeutic uses.

The main clinical application of transcranial magnetic stimulation concerns testing of the functional integrity of the corticospinal tract in patients with disorders affecting the central nervous system. Use of standard transcranial magnetic stimulation in these neurological disorders provides several information: detection of subclinical upper motoneuron involvement, at times localization of anatomical site of lesions, longitudinal monitoring of motor abnormalities during course of diseases, and valuable aid to differential diagnosis. The more complex transcranial magnetic stimulation applications provide information on the central mechanisms underlying changes in the corticomotoneuronal excitability in various neurological conditions.

Repetitive stimulation of the brain opens a new field of investigations of cognitive function and mood, and of therapeutic possibilities. There are interesting results in the short-term treatment of refractory depression by daily sessions of repetitive transcranial magnetic stimulation. By changing the frequency of stimulation, it may be possible either to up- or down-modulate cortical excitability for therapeutic benefit.

The ability of transcranial magnetic stimulation to measure and modify cortical activity offers possibilities to apply this methodology to clinical neurology, neurorehabilitation and psychiatry.

Transkranijinė magnetinė stimuliacija klinikinėje praktikoje

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Raktažodžiai: kortikospinalinis laidumas, motoriniai potencialai, elektrofiziologija, neurofiziologija, žievės tylusis periodas.

Santrauka. Transkranijinė magnetinė stimuliacija – tai neinvazinė ir neskausminga galvos smegenų ir galvos nervų stimuliacija, taikoma nuo 1985 metų. Transkranijinės magnetinės stimuliacijos metu galima stimuliuoti pavieniais ar skirtingų intervalų poriniais impulsais (tas pačias ar skirtingas smegenų sritis) bei įvairaus dažnio pasikartojančių ritminių impulsų grupėmis. Pavieniai impulsai sukelia motorinius potencialus, kurie taikomi diagnozuojant ligas bei prognozuojant jų eigą. Pasikartojančių impulsų transkranijinė magnetinė stimuliacija gali keisti galvos smegenų žievės jaudrumą. Taigi atsirado galimybė tyrinėti neuronų ryšius ir diagnozuoti bei gydyti jų sutrikimus.

Šiame straipsnyje apžvelgiami pagrindiniai transkranijinės magnetinės stimuliacijos principai, metodologiniai aspektai, atlikimo būdai, taip pat analizuojamas jų panaudojimas klinikinėje praktikoje.

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