Effects of Intermittent Theta Burst Stimulation on Cerebral Blood Flow and Cerebral Vasomotor Reactivity

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Objectives—To determine whether intermittent theta burst stimulation influences cerebral hemodynamics, we investigated changes induced by intermittent theta burst stimulation on the middle cerebral artery cerebral blood flow velocity and vasomotor reactivity to carbon dioxide (CO₂) in healthy participants. The middle cerebral artery flow velocity and vasomotor reactivity were monitored by continuous transcranial Doppler sonography. Changes in cortical excitability were tested by transcranial magnetic stimulation.

Methods—In 11 healthy participants, before and immediately after delivering intermittent theta burst stimulation, we tested cortical excitability measured by the resting motor threshold and motor evoked potential amplitude over the stimulated hemisphere and vasomotor reactivity to CO₂ bilaterally. The blood flow velocity was monitored in both middle cerebral arteries throughout the experimental session. In a separate session, we tested the effects of sham stimulation under the same experimental conditions.

Results—Whereas the resting motor threshold remained unchanged before and after stimulation, motor evoked potential amplitudes increased significantly (P = .04). During and after stimulation, middle cerebral artery blood flow velocities also remained bilaterally unchanged, whereas vasomotor reactivity to CO₂ increased bilaterally (P = .04). The sham stimulation left all variables unchanged.

Conclusions—The expected intermittent theta burst stimulation–induced changes in cortical excitability were not accompanied by changes in cerebral blood flow velocities; however, the bilateral increased vasomotor reactivity suggests that intermittent theta burst stimulation influences the cerebral microcirculation, possibly involving subcortical structures. These findings provide useful information on hemodynamic phenomena accompanying intermittent theta burst stimulation, which should be considered in research aimed at developing this noninvasive, low-intensity stimulation technique for safe therapeutic applications.

Key Words—blood flow velocity; intermittent theta burst stimulation; transcranial magnetic stimulation; vasomotor reactivity

Transcranial magnetic stimulation is a neurophysiologic technique for noninvasive stimulation of the human brain.1 It can be applied in a single-pulse or repetitive modality; low-frequency repetitive transcranial magnetic stimulation (<1 Hz) is thought to have inhibitory effects, whereas high-frequency stimulation (>1 Hz) is known to have facilitatory effects.2 Theta burst stimulation is a type of repetitive transcranial magnetic stimulation. It is applied at a low intensity (80% of the active motor threshold) and high frequency (triplets of stimuli at 50 Hz). Theta burst stimulation–induced changes in cortical excitability depend on the stim-
stimulation in healthy individuals and patients with stroke, old primary motor cortex repetitive transcranial magnetic stimulation during high-frequency (17-Hz) suprathreshold and contralateral middle cerebral artery flow velocity.\textsuperscript{14}

For example, repetitive transcranial magnetic stimulation applied to the primary motor cortex increased the ipsilateral metabolism and hemodynamics in healthy individuals.\textsuperscript{15}

Because theta burst stimulation induces longer-lasting (up to 20 minutes) and more stable effects on cortical excitability than regular repetitive transcranial magnetic stimulation, the technique holds promise as a potential therapeutic tool. Ample evidence describes theta burst stimulation–induced changes in cortical excitability in several pathologic conditions ranging from psychiatric disorders such as depression and obsessive-compulsive disorder\textsuperscript{5–7} to neurologic diseases such as spasticity,\textsuperscript{8} blepharospasm,\textsuperscript{9} and neuropathic pain.\textsuperscript{10} Theta burst stimulation has also been used for therapeutic purposes in patients with chronic and acute stroke.\textsuperscript{11–13}

Numerous studies have described repetitive transcranial magnetic stimulation–induced changes in the cerebral metabolism and hemodynamics in healthy individuals. For example, repetitive transcranial magnetic stimulation applied to the primary motor cortex increased the ipsilateral and contralateral middle cerebral artery flow velocity.\textsuperscript{14}

The blood flow velocity also increased when repetitive stimulation was applied at different facilitatory frequencies over the visual cortex and the posterior cerebral artery was monitored.\textsuperscript{15} Studies applying low-frequency repetitive stimulation reported different results: inhibitory repetitive stimulation (0.9 Hz) applied over the right dorsolateral prefrontal cortex led to a decrease in the ipsilateral middle cerebral artery flow velocity followed by a contralateral increase, possibly owing to a compensatory mechanism.\textsuperscript{16}

Similarly, a positron emission tomographic study showed that changes in regional cerebral blood flow induced by repetitive stimulation differed according to the stimulation frequency.\textsuperscript{17} Collectively, these findings suggest that repetitive transcranial magnetic stimulation may cause changes in the blood flow velocity, and the direction these changes take depends on the frequency of stimulation.

Two studies have investigated how repetitive transcranial magnetic stimulation influences vasomotor reactivity, the hemodynamic variable reflecting microcirculatory responses to a vasodilatory stimulus such as carbon dioxide (CO\textsubscript{2}). In a study investigating changes in mean blood flow and vasomotor reactivity by transcranial Doppler sonography during high-frequency (17-Hz) suprathreshold primary motor cortex repetitive transcranial magnetic stimulation in healthy individuals and patients with stroke, the middle cerebral artery vasomotor reactivity decreased after real but not sham stimulation; the vasomotor reactivity decrease was bilateral, suggesting that repetitive stimulation induced a widespread effect on the brain circulation, unrelated to the feeding territory of the artery supplying the stimulated area or to the stimulated hemisphere.\textsuperscript{18}

A similar study concluded that low-frequency (1-Hz) repetitive stimulation led to a significant increase in vasomotor reactivity in healthy individuals presumably because high- and low-frequency stimulation have opposite effects on cortical excitability.\textsuperscript{19}

To our knowledge, no studies have investigated the effects of repetitive transcranial magnetic stimulation delivered as theta burst stimulation on cerebrovascular hemodynamics during simultaneous transcranial Doppler monitoring. Transcranial Doppler sonography is a simple tool for evaluating blood flow velocities and vasomotor reactivity as the vasodilatation/vasoconstriction microcirculatory response to apnea-hypercapnia and hyperventilation-hypocapnia.\textsuperscript{20} Having reliable information on theta burst stimulation and cerebrovascular hemodynamics is an essential prerequisite for assessing whether theta burst stimulation can be safely applied for therapeutic purposes, especially in patients with stroke, who are known to have impaired cerebral vasoregulation.\textsuperscript{21}

In this study, we investigated variations in cerebral hemodynamics and cortical excitability induced by intermittent theta burst stimulation. In a group of healthy participants, as variables to assess cerebral hemodynamics, we tested the cerebral blood flow velocity and vasomotor reactivity to CO\textsubscript{2}, under continuous bilateral middle cerebral artery transcranial Doppler monitoring. As transcranial magnetic stimulation variables to assess cortical excitability, we measured the resting motor threshold and motor evoked potential amplitudes before and after intermittent theta burst stimulation.

Materials and Methods

Participants
We tested 11 healthy participants (3 women and 8 men; mean age ± SD, 31 ± 8.5 years). All participants gave their written informed consent to the experiment, and the local Ethical Committee approved the procedures.

Transcranial Magnetic Stimulation

Stimulation Technique
Transcranial magnetic stimulation and intermittent theta burst stimulation were delivered through a high-frequency magnetic stimulator (Magstim Rapid; Magstim Company,
old intensity, with an interstimulus interval of 10 seconds.

Intermittent theta burst stimulation consisted of 20 trains of stimuli with an intertrain pause of 8 seconds; each train consisted of 10 bursts repeated at 5 Hz (200 milliseconds between each burst); each burst consisted of 3 pulses at 50 Hz (20 milliseconds between each pulse). Overall, 600 pulses were delivered in 190 seconds; the stimulus intensity was set at 80% of the active motor threshold.3

Before and after intermittent theta burst stimulation, we measured the resting motor threshold and motor evoked potential amplitude by single-pulse transcranial magnetic stimulation; to avoid the effects of single pulses on local blood flow, only 3 single magnetic stimulation pulses were delivered over the primary motor cortex of the stimulated hemisphere at 120% of the resting motor threshold, considered the lowest intensity able to elicit 5 motor evoked potentials with an amplitude of at least 200 μV in 10 consecutive trials.

Transcranial Doppler Sonography

Transcranial Doppler data were acquired with a Multidop DWL X-Digital apparatus (software release 2.6; DWL, Sipplingen, Germany) equipped with 2 2-MHz probes and a probe holder (Marc 600; Spencer Technologies, Seattle, WA) fixed on the temporal bone windows. As previously described,24 the middle cerebral arteries were insonated bilaterally, continuously and simultaneously, at a depth of 54 mm. To monitor flow changes throughout the experimental session, mean middle cerebral artery flow velocities (centimeters per second) were visualized on the “trend” window. A time-averaged mean middle cerebral artery flow velocity was evaluated 2 minutes before, during, and 2 minutes after transcranial magnetic stimulation. Other variables monitored throughout the study were inspired and end-tidal CO₂, measured directly on the transcranial Doppler machine through a breathing mask connected to the device.

Middle cerebral artery vasomotor reactivity to CO₂ was evaluated twice, immediately before and after repetitive transcranial magnetic stimulation. After 5-minute baseline monitoring of middle cerebral artery blood flow velocities, responses to hypercapnia and hypocapnia were simultaneously assessed for both sides, as described elsewhere.25,26 Hypercapnia was induced with an anesthetic mask, fitted tightly over the mouth and nose, providing a 5% CO₂ mixture with a flow rate of 8 L/min for 2 minutes, long enough to reach the maximum increase in flow velocity and steady state. Subsequently, for the hypocapnic condition, after baseline velocities returned, the participant was asked to hyperventilate for 3 minutes to reach a maximal decrease in Pco₂ (end-tidal CO₂ <20%). Total vasomotor reactivity was calculated as the sum of mean percent blood flow velocity changes for hypercapnia and hypocapnia on each side, according to the formula (mean CO₂ flow velocity – mean hyperventilation flow velocity/mean basal flow velocity) × 100.

Arterial blood pressure was monitored manually with a cuff placed on the right arm. Measurements were repeated under baseline conditions, at the maximum increase in flow velocity during hypercapnia, and at the maximum decrease in
flow velocity during hypocapnia. All transcranial Doppler data were stored digitally for offline analysis.

**Statistical Analysis**

Unless otherwise stated, all values are mean ± SD. A paired Student t test was used to compare the resting motor threshold and motor evoked potential amplitude before and immediately after intermittent theta burst stimulation. A 1-way analysis of variance (ANOVA) was used to compare mean blood flow velocities before, during, and after stimulation. A paired Student t test was used to compare mean changes in vasomotor reactivity before and after stimulation. \( P \leq .05 \) was considered statistically significant.

**Results**

None of the participants reported adverse effects or had discomfort during the experimental sessions related to either intermittent theta burst stimulation, transcranial Doppler monitoring, or CO\(_2\) inhalation. Intermittent theta burst stimulation caused no significant change in resting motor threshold values (before stimulation, 58.3% ± 6.8% of maximum stimulator output; after stimulation, 58.4% ± 7.6%). The motor evoked potential amplitude was significantly increased by intermittent theta burst stimulation (2.1 ± 1.3 mV before stimulation; 2.6 ± 1.5 mV after stimulation; \( P = .04 \); Table 1). Sham stimulation caused no significant change in the resting motor threshold (before stimulation, 56.6% ± 2.8%; after stimulation, 55.6% ± 0.5%) and motor evoked potential amplitude (before stimulation, 1.4 ± 1.8 mV; after stimulation, 1.5 ± 1.9 mV; Table 2).

Continuous transcranial Doppler monitoring showed only a transient intermittent theta burst stimulation artifact (during both real and sham stimulation) on the Doppler spectrum and did not interfere with its interpretation (Figure 1). During stimulation, the middle cerebral artery flow velocity remained unchanged in both the stimulated (ANOVA, \( F = 0.018; P > .05 \)) and contralateral (ANOVA, \( F = 0.009; P > .05 \)) hemispheres (Table 1 and Figure 2). After stimulation, vasomotor reactivity increased bilaterally (t test, \( df = 10 \); stimulated hemisphere, \( P = .0417 \); nonstimulated hemisphere, \( P = .0464 \); Table 1 and Figure 3). During sham stimulation, the middle cerebral artery flow velocity remained unchanged bilaterally (ANOVA, stimulated hemisphere, \( F = 0.021; P > .05 \); nonstimulated hemisphere, \( F = 0.012; P > .05 \)); no significant changes were observed in vasomotor reactivity measured before and after sham stimulation (Table 2). The end-tidal CO\(_2\) values after hyperventilation were 19% ± 3% for the stimulation group and 19% ± 2% for the sham group. Arterial blood pressures (systolic and diastolic) did not show significant changes between basal, hypercapnic, and hypocapnic conditions induced for vasomotor reactivity calculations in both the stimulation and sham groups (Tables 1 and 2).

**Discussion**

In this study, designed to investigate changes in cerebral hemodynamics and cortical excitability induced by intermittent theta burst stimulation compared to sham stimulation, we measured the flow velocity and vasomotor reactivity throughout continuous middle cerebral artery

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**Table 1. Transcranial Doppler and Magnetic Stimulation Measurements: Intermittent Theta Burst Stimulation**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before Stimulation</th>
<th>During Stimulation</th>
<th>After Stimulation</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middle cerebral artery mean flow velocity, cm/s</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulated hemisphere</td>
<td>58.4 ± 12.7</td>
<td>58.1 ± 12.8</td>
<td>57.1 ± 13.4</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Nonstimulated hemisphere</td>
<td>57.8 ± 13.6</td>
<td>58.9 ± 13.1</td>
<td>57.7 ± 12.6</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Vasomotor reactivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulated hemisphere</td>
<td>571 ± 7.3</td>
<td>ND</td>
<td>69.4 ± 8.4</td>
<td>.0417</td>
</tr>
<tr>
<td>Nonstimulated hemisphere</td>
<td>577 ± 8.3</td>
<td>ND</td>
<td>70.2 ± 8.6</td>
<td>.0464</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal condition</td>
<td>115 ± 10</td>
<td>ND</td>
<td>118 ± 9</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Carbon dioxide inhalation</td>
<td>113 ± 12</td>
<td>ND</td>
<td>115 ± 6</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>114 ± 9</td>
<td>ND</td>
<td>112 ± 9</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
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<td></td>
</tr>
<tr>
<td>Basal condition</td>
<td>68 ± 6</td>
<td>ND</td>
<td>66 ± 5</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Carbon dioxide inhalation</td>
<td>65 ± 5</td>
<td>ND</td>
<td>65 ± 3</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>67 ± 5</td>
<td>ND</td>
<td>67 ± 4</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Resting motor threshold, %</td>
<td>58.3 ± 6.8</td>
<td>ND</td>
<td>58.4 ± 7.6</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Motor evoked potential amplitude, mV</td>
<td>2.1 ± 1.3</td>
<td>ND</td>
<td>2.6 ± 1.5</td>
<td>.04</td>
</tr>
</tbody>
</table>

Values are mean ± SD. \( P \) values were determined by a 1-way analysis of variance for mean flow velocity and by a paired Student t test for vasomotor reactivity, resting motor threshold, and motor evoked potential amplitude. ND indicates not determined.
Transcranial Doppler monitoring; although the hemodynamic variable middle cerebral artery flow velocity measured in the stimulated and contralateral hemispheres remained unchanged before, during, and after intermittent theta burst stimulation, the middle cerebral artery vaso-motor reactivity, the hemodynamic variable reflecting microcirculatory responses to a vasodilatory stimulus such as CO₂, increased significantly from baseline after stimulation. As previously reported by others, intermittent theta burst stimulation induced changes in cortical excitability, leaving the resting motor threshold unchanged but increasing motor evoked potential amplitudes. As previously reported, intermittent theta burst stimulation induced changes in cortical excitability, leaving the resting motor threshold unchanged but increasing motor evoked potential amplitudes. 3, 4, 27

What distinguishes this study from others is that simultaneous transcranial Doppler monitoring allowed us to measure the middle cerebral artery flow velocity and vaso-motor reactivity under tightly controlled experimen-

**Table 2. Transcranial Doppler and Magnetic Stimulation Measurements: Sham Stimulation**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before Stimulation</th>
<th>During Stimulation</th>
<th>After Stimulation</th>
<th>P</th>
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<tr>
<td>Middle cerebral artery mean flow velocity, cm/s</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulated hemisphere</td>
<td>54.0 ± 14.7</td>
<td>55.0 ± 16.0</td>
<td>53.6 ± 15.0</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Nonstimulated hemisphere</td>
<td>54.6 ± 15.0</td>
<td>54.0 ± 16.0</td>
<td>55.0 ± 14.0</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Vasomotor reactivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulated hemisphere</td>
<td>36.2 ± 8.0</td>
<td>ND</td>
<td>35.6 ± 9.0</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Nonstimulated hemisphere</td>
<td>36.2 ± 8.4</td>
<td>ND</td>
<td>35.5 ± 8.9</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal condition</td>
<td>113 ± 9</td>
<td>ND</td>
<td>117 ± 6</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Carbon dioxide inhalation</td>
<td>112 ± 11</td>
<td>ND</td>
<td>116 ± 7</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>114 ± 8</td>
<td>ND</td>
<td>114 ± 11</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal condition</td>
<td>67 ± 6</td>
<td>ND</td>
<td>65 ± 4</td>
<td>&gt;.05</td>
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<tr>
<td>Carbon dioxide inhalation</td>
<td>66 ± 3</td>
<td>ND</td>
<td>65 ± 4</td>
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<td>67 ± 5</td>
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<td>Resting motor threshold, %</td>
<td>56.6 ± 2.8</td>
<td>ND</td>
<td>55.6 ± 0.5</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>Motor evoked potential amplitude, mV</td>
<td>1.4 ± 1.8</td>
<td>ND</td>
<td>1.5 ± 1.9</td>
<td>&gt;.05</td>
</tr>
</tbody>
</table>

Values are mean ± SD. P values were determined by a 1-way analysis of variance for mean flow velocity and by a paired Student t test for vasomotor reactivity, resting motor threshold, and motor evoked potential amplitude. ND indicates not determined.

**Figure 1.** Left middle cerebral artery Doppler spectrum before, during, and after a 2-second train of transcranial magnetic stimulation burst stimuli in a representative healthy participant. Top line, left to right, Machine settings: depth of insonation, gain, sample volume, power, and filters. Second line, left to right, Maximum, mean, and diastolic blood flow velocities. Pulsatility and resistive indices are above and below the 0 line, respectively.
tal conditions, thereby providing previously unavailable information on intermittent theta burst stimulation–induced variations in cerebral hemodynamics.

Why intermittent theta burst stimulation did not affect the cerebral hemodynamic variable flow velocity in the stimulated and contralateral hemispheres remains unclear. A plausible explanation is that changes in the cerebral blood flow velocity depend closely on the transcranial magnetic stimulation intensity. In fact, the stimulation intensity in theta burst stimulation paradigms is 80% of the active motor threshold, which is far below the threshold level for activation of the corticospinal tract at rest and roughly lower than the stimulus intensity in regular repetitive transcranial magnetic stimulation. A repetitive transcranial magnetic stimulation study comparing the effects of different stimulus frequencies and intensities on the middle cerebral artery flow velocity monitored by transcranial Doppler sonography showed that flow velocity variations depended strictly on the stimulation intensity, with a larger increase at a suprathreshold than a subthreshold intensity.28 Others reported similar intensity-dependent changes in a study testing the effects of repetitive transcranial magnetic stimulation on the cerebral metabolism measured by H215O positron emission tomography.29,30

Another possible reason why the middle cerebral artery flow velocity remained unchanged is that transcranial Doppler monitoring achieves lower sensitivity than other techniques in disclosing changes in the cerebral metabolism. Near-infrared spectroscopy is a noninvasive technique for evaluating changes in hemoglobin concentration reflecting cerebral activation or deactivation patterns; a previous study investigating changes in cerebral metabolism during and after theta burst stimulation as measured with near-infrared spectroscopy showed a deactivation pattern in the hemisphere contralateral to stimulation.31

The most interesting hemodynamic finding in this theta burst stimulation study in healthy participants was the bilateral increase in vasomotor reactivity, an index of cerebral vasodilatory capacity that reflects microcirculation reactivity to vasoactive stimuli. The widespread effect of intermittent theta burst stimulation on the cerebral microcirculation is in line with current evidence on changes in middle cerebral artery vasomotor reactivity after repetitive transcranial magnetic stimulation over the motor cortex. Whereas some reported that after facilitatory repetitive transcranial magnetic stimulation (17 Hz), vasomotor reactivity decreased bilaterally,18 others showed that after inhibitory repetitive stimulation (1 Hz), it increased.19 Collectively, these bilateral microcirculatory changes support the hypothesis suggested by Vernieri et al18 that an autonomic response induced by repetitive transcranial magnetic stimulation alters the vascular sympathetic tone and therefore induces diffuse changes in vasomotor reactivity, regardless of the side stimulated.

Although intermittent theta burst stimulation has known facilitatory effects on neural structures in the motor cortex,3 the stimulation-induced vasomotor reactivity changes we observed went in the same direction as those reported after an inhibitory repetitive transcranial magnetic stimulation protocol.19 Hence, we hypothesize that repet-

![Figure 2](image-url)
itive transcranial magnetic stimulation might influence the autonomic nervous system’s response independently from its effects on cortical excitability. What direction the magnetic stimulation–induced vasomotor reactivity changes take might depend on other stimulus variables, such as intensity and frequency (which may differ even among protocols having similar, e.g., facilitatory, effects on cortical excitability), but also on the coil position and orientation. Precisely which mechanism is responsible for the bilateral changes in vasomotor reactivity remains unclear.

Going one step further than the explanation proposed by Vernieri et al., we hypothesize the involvement of subcortical vasoactive pathways. These pathways are among the best-studied neural pathways regulating cortical cerebral blood flow through neurons originating in the basal ganglia and projecting to cortical microvessels. Therefore, repetitive transcranial magnetic stimulation might act on cortical perivascular nerves in the stimulated hemisphere, triggering a generalized (i.e., bilateral) autonomic response conveyed by the subcortical vasoactive pathways.

Another hypothesis to consider is that the hemodynamic changes (increased vasomotor reactivity) observed in the hemisphere contralateral to stimulation depend on interhemispheric connections directly activated by intermittent theta burst stimulation. Evidence shows that intermittent theta burst stimulation not only facilitates the motor evoked potential on the stimulated hemisphere but also suppresses the potential on the contralateral one; the mechanisms underlying this contralateral inhibition are still unclear, yet it is conceivable that this inhibitory activity...
(which implies metabolic activity just as the facilitation occurs on the stimulated hemisphere) might be responsible for the observed bilateral increase in vasomotor reactivity.

In conclusion, despite the relatively small study sample, we were able to disclose previously unavailable information on the cerebral hemodynamic changes accompanying intermittent theta burst stimulation, which will help in developing theta burst stimulation as a safe therapeutic tool. The unchanged cerebral blood flow velocities during intermittent theta burst stimulation are reassuring for applications in patients with stroke, especially in the acute phase when massive effects on cerebral hemodynamics might raise considerable safety issues. The intermittent theta burst stimulation—induced increase in vasomotor reactivity requires further investigation in studies including participants most likely to have microcirculatory malfunctioning, such as elderly persons and patients with stroke.

References


