Motor cortex fatigue in sports measured by transcranial magnetic double stimulation

FRITHJOF TERGAU, ROLF GEESE, AXEL BAUER, SUSANNE BAUR, WALTER PAULUS, and CARL DETLEV REIMERS

Department of Clinical Neurophysiology, University of Göttingen, GERMANY; Institute of Sports Sciences, University of Göttingen, GERMANY; Institute of Sports Medicine, University of Ulm, GERMANY

ABSTRACT

TERGAU, F., R. GEESE, A. BAUER, S. BAUR, W. PAULUS, and C. D. REIMERS. Transcranial magnetic double stimulation in the investigation of motor cortex fatigue in sports. *Med. Sci. Sports Exerc.,* Vol. 32, No. 11, pp. 1942–1948, 2000. **Purpose:** Besides peripheral mechanisms, central fatigue is an important factor limiting the performance of exhausting exercise in sport. The mechanisms responsible are still in discussion. Using noninvasive transcranial magnetic stimulation (TMS) in a double-pulse technique, we sought to assess fatigue of the motor cortex after exhaustive anaerobic strain. **Methods:** 23 male subjects (22–52 yr) taking part in the study were requested to accomplish as many pull-ups as possible until exhaustion. The amount of physical lifting work was recorded. Before and immediately after the task, intracortical inhibition (ICI) and facilitation (ICF) were measured by a conditioned-test double-pulse TMS method for the right brachioradialis (BR) and abductor pollicis brevis muscle (APB). **Results:** After exercise, ICF was significantly reduced in the BR but not in the APB. ICI was not altered. Changes tended to normalize within 8 min after the task. The amount of lifting work accomplished showed significant correlation to the values of ICF reduction ($r = 0.73$). Moreover, the baseline values of ICF before exercise were also significantly correlated to the lifting work ($r = 0.63$). **Conclusions:** Because double-pulse TMS gives access to the motor cortex independently of spinal or peripheral mechanisms, reduced ICF reflects decreased excitability of interneuronal circuits within the motor cortex. We suggest that ICF measures motor cortex fatigue after exhausting strain specifically for the muscles performing the task. γ-Aminobutyric acid (GABA)-ergic neurotransmission is possibly involved in the mechanisms mediating central fatigue. Double-pulse TMS may be a useful tool in the control of training in sports as well as in the detection of pathological central fatigue in overreaching and in the prevention of overtraining. **Key Words:** CENTRAL FATIGUE, EXHAUSTIVE STRAIN, PULL-UPS, INTRACORTICAL FACILITATION, GABA, HUMAN, OVERTRAINING

Muscle fatigue is defined as a loss of muscle strength during sustained dynamic or isometric contraction or the need of enhanced voluntary effort to retain constant power (2,23). It is one of the main limitations in any kind of endurance sport and can be subdivided into central and peripheral fatigue. Although peripheral muscle fatigue has been widely studied (for reviews, see, e.g., 1,14,26,28), only little is known about the mechanisms underlying central fatigue.

As a noninvasive technique, transcranial magnetic stimulation (TMS) is a convenient method for investigating the excitability of the motor pathway involved in the performance of physical exercise. In several studies, augmented amplitudes of motor-evoked potentials (MEP) were found during long-lasting exercise (21,22,24,30), and it was concluded that under these conditions neuronal excitability was increased. On the other hand, decrease of MEPs after exhaustive exercise (3,4,6,16,20,22,27,33) was seen as fatigue of the central nervous system.

By using pairs of subthreshold and suprathreshold stimuli, a new TMS technique has become a valuable tool in the investigation of the excitability of the motor system and of intracortical inhibitory and facilitatory circuits that control the motor cortex (17,36). The present study was performed to provide further and more specific evidence in support of the hypothesis that the motor cortex is less excitable after exhaustive physical strain.

METHODS

Subjects. Twenty-three male volunteers, aged 19–53 yr (mean age 28.7 ± 7.7 yr) participated in the study. Sixteen subjects performed sports regularly, nine of them mainly in disciplines requiring strength endurance of arm muscles (rowing, canoeing, gymnastics). The study was approved by the ethics committee of the University of Göttingen. All subjects gave their written informed consent.

Exercise. On a horizontal bar, subjects had to perform as many pull-ups as possible until exhaustion. During the procedure the feet did not touch the ground, and between the pull-ups the arms had to be stretched completely. Pull-ups

0195-9131/00/3211-1942/0
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Submitted for publication June 1999.
Accepted for publication December 1999.

were counted only when the chin was lifted above the bar. After breaks of 1 min, the procedure had to be repeated until a single pull-up could not be performed any more or exercise was stopped after the seventh set.

Assessment of lifting work. For further analysis, the physical work accomplished was estimated. The extent of lifting was recorded for each pull-up by lengthening of a cord, which was hooked into the belt. After each pull-up the cord was automatically drawn back by a spring. A computer program, Myosoft 1.5, Noraxon registered the time course of the lengthening and shortening of the cord indicating the subject's position. The physical work (W) and the individual work normalized to the subject's body mass (W_m) were calculated using the following algorithms:

$$W[J] = body\ mass[kg] \times 9.81\ [m/s^2] \times \sum l\ [m]$$

$$(l = lift\ at\ each\ pull\text{-}up)$$

and

$$W_m\ [J/kg] = W\ [J]/body\ mass[kg]$$

Transcranial magnetic stimulation. Before and after the completed exercise, investigation of intracortical excitability was performed. Postexercise measurement of intracortical excitability started some 1–2 min after the final pull-up. Subjects were seated in a comfortable reclining chair. The right brachioradialis muscle (BR) was investigated as one of the muscles mainly involved in pull-ups. Simultaneously, the right abductor pollicis brevis muscle (APB), which was of minor importance for the task, was studied as a control. Surface electromyography (EMG) was recorded using 9-mm Ag-AgCl electrodes in a belly-to-tendon montage. The raw EMG signal was amplified, filtered with a time constant of 10 ms and a low-pass filter of 2.5 kHz, and digitized (5-kHz sampling rate) and recorded on an IBM-PC/486-AT-compatible laboratory computer using the NeuroSCAN 3.0 data collection and analyzing software. Focal transcranial magnetic stimulation was delivered by two MAGSTIM 200 stimulators (max. 2.2 T, each) connected by a BiStim module (The Magstim Company, Whitland, Dyfed, U.K.) and was applied through a focal figure-of-eight magnetic coil (70-mm diameter of each wing). The optimal position of the magnetic coil for eliciting an MEP in the target muscles was determined by moving the coil in small steps over the forearm area of the left primary motor cortex. The center of the coil was held tangentially to the scalp with the handle of the coil pointing backward and 45° laterally from the interhemispheric line. This orientation was described to be most effective to activate the corticospinal system transsynaptically for eliciting MEPs in the particular target muscles (5,32). The optimal position was defined as the stimulation site that yielded the largest-possible MEP in both the resting BR and APB at slightly suprathreshold stimulus intensities. The optimal position was marked with a pen to assure constant placement of the coil throughout the sessions.

Before the baseline measurement, resting (RMT) and active motor thresholds (AMT) in the BR were obtained, and stimulus intensities for double-pulse TMS were determined. Threshold values and stimulus intensities were expressed as percentage of the maximal stimulator output. Thresholds were approached from a slightly suprathreshold intensity by reducing the stimulus intensity in 1% steps and were defined as the highest stimulus intensity that did not produce MEPs of more than 50 μV in at least 5 of 10 trials in the resting target muscle (for the RMT) and in the averaged EMG of eight trials in the slightly tonically activated target muscle (for the AMT), respectively. RMTs after exercise were briefly retested (see below).

Intracortical excitability. The intracortical (or cortico-cortical) excitability was studied in the resting target muscles using the conditioning-test double-pulse paradigm (17,37). Both conditioning and test stimulus were delivered through the same coil. Intensity of the test stimulus was selected to produce a control MEP of about 0.75 mV peak-to-peak amplitude in the BR and was kept constant throughout the procedures by adjusting the intensity of the test pulse if necessary. The preceding conditioning stimulus was fixed at 90% of AMT. It has been described that such a conditioning stimulus has no effect on spinal excitability and its effect on the control MEP presumably occurs at supraspinal, most likely cortical level (10,17,37) (for detail, see Discussion). Because retesting of thresholds showed no significant changes after exercise, the intensity of the conditioning pulse was kept constant after exercise. The measurement was performed in one session consisting of 15 consecutive sets of seven randomly intermixed conditions: six different double-pulse conditions with interstimulus intervals (ISI) of 2, 3, 4, 8, 10, and 15 ms and the test stimulus alone. Time between two consecutive trials was 5 s; thus, the procedures took 8:25 min each. To assure complete relaxation of the target muscles, auditory and visual feedback was given throughout the procedure. Trials in which background EMG activity could be detected were discarded from the analysis. Single trial peak-to-peak MEP amplitudes were measured off-line and averaged for each condition separately. Size of the conditioned MEP was expressed as a percentage of the test MEP. The short ISIs (2, 3, 4 ms) result in a diminution (so-called intracortical inhibition, ICI), whereas the long ISI (8, 10, 15 ms) lead to an increment of the conditioned MEP (so-called intracortical facilitation, ICF) (36).

Statistical procedures. Motor thresholds, stimulus intensity as well as MEP control sizes of pre- and post-exercise measurements were compared using the two-tailed paired Student's *t*-test. Also, values of MEP peak-to-peak amplitudes after double-pulse TMS of pre- and post-exercise results were compared using the two-tailed paired Student's *t*-test for each interstimulus interval separately. ICF of preexercise and postexercise measurement and also the changes in ICF were analyzed for linear Pearson-Bravais' correlation to the normalized lifting work W_m. In all tests, statistical significance was assumed with $P < 0.05$.

RESULTS

Pull-ups. In all subjects, the number of pull-ups per set declined rapidly (cf. Fig. 1A). Eighteen of the subjects

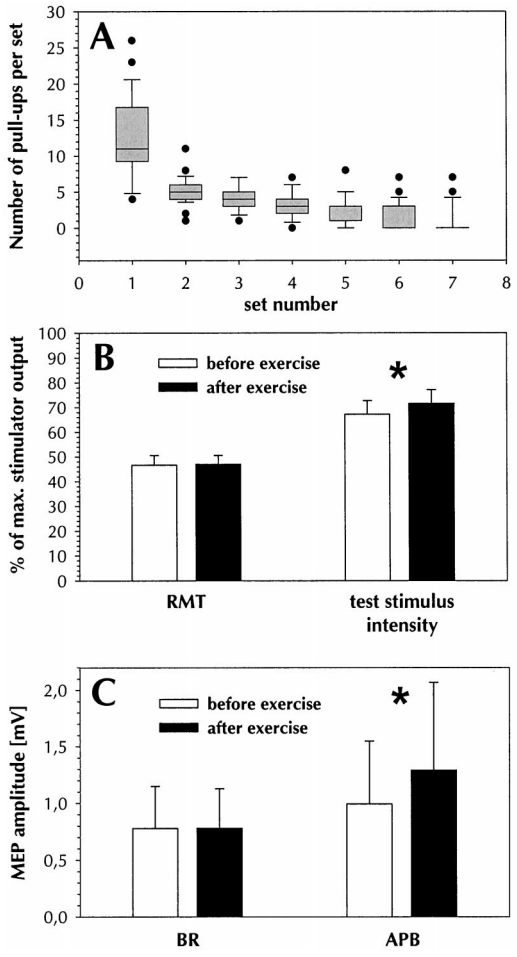

Figure 1—A, The number of pull-ups for each of the seven sets are displayed. Box plots represent all subjects by the median (*horizontal line in the box*), 25th and 75th percentile (*box*), 10th and 90th percentile (*error bars*), and values below 10th or above 90th percentile (*circles*). Ordinate indicates the number of pull-ups per set. Note the marked decrease of pull-ups after the first set indicating beginning fatigue. B, Resting motor threshold (RMT) and test stimulus intensity are displayed for the pre- and the post-exercise measurement. Values are expressed on the ordinate [as percentage of the maximum stimulator output]. C, The peak-to-peak amplitudes of the test MEP of both BR and APB muscle are given for the pre- and post-exercise measurement. Values are expressed on the ordinate [in mV]. Note that test MEP for the BR were constant although stimulus intensity was increased (cf. Fig. 1B), whereas they were enhanced in the APB.

reached complete exhaustion within less than 7 sets of pull-ups. Five subjects performed 7 sets; three of them reached a steady state of number of pull-ups without further decline within the last three sets. Over all sets, subjects performed on average 30.4 ± 16.3 (range 5–66) pull-ups in 5.2 ± 1.1 (range 3–7) sets. Mean physical lifting work (W) was 13.6 ± 7.8 kJ (range 2.3–31.8 kJ). Normalized work per body mass (W_m) was on average 173.7 ± 95.2 $J \cdot kg^{-1}$ (range 28.3–372.1 $J \cdot kg^{-1}$).

Motor threshold, test stimulus intensity, and MEP sizes. Mean resting and active motor threshold at baseline were on average 46.7 ± 3.9 (range 40–53) and 33.9 ± 4.2 (range 27–43) % of maximum stimulator output, respectively. Retesting of RMT after exercise yielded on average 47.0 ± 3.6%, which was not significantly different from baseline ($P = 0.31$). Intensity for the test stimulus as to produce a constant average MEP in the BR was 67.2 ± 5.5% for the baseline measurement and was significantly increased to 71.5 ± 5.6% ($P < 0.001$) after exercise (cf. Fig. 1B). During the baseline measurement of intracortical excitability the mean control MEPs were 0.78 ± 0.37 mV in the BR and 0.99 ± 0.55 mV in the APB. Mean MEPs after exercise were kept constant at 0.78 ± 0.35 mV in the BR, whereas they were markedly increased to 1.29 ± 0.78 mV in the APB ($P = 0.02$; cf. Fig. 1C).

Intracortical excitability. As expected, for both muscles, baseline measurements showed small MEPs after double-pulse stimulation for inhibitory interstimulus intervals (ISI) of 2–4 ms and large MEPs for facilitatory ISIs of 8, 10, and 15 ms, respectively. After exercise, MEPs after facilitatory ISIs were significantly decreased in the BR ($P = 0.033$, $P = 0.009$, and $P = 0.012$ for ISI 8, 10, and 15, respectively; cf. Figs. 2 and 3). ICF in the BR as average over ISI 8, 10, and 15 decreased from 127.9 ± 30.7% to 117.4 ± 24.9% ($P = 0.0009$). ICF in the APB remained unchanged. For inhibitory ISIs MEPs were nearly unchanged in either muscle (cf. Figs. 2 and 3).

Separate analysis of the first (8 values for each condition) and the second half (7 values of each condition) of the postexercise measurement showed that values in facilitatory MEPs of the BR were markedly decreased during the first half (116.4 ± 23.1) but nearly normalized for the second half (123.7 ± 33.7) when compared with baseline (127.9 ± 30.7; cf. Fig. 3C). Neither inhibitory MEPs of the BR nor MEPs in the APB were altered in this analysis (data not shown). Thus, only the results of the first part of the postexercise measurement were taken for further analysis.

Intracortical facilitation (ICF) in the BR was calculated for each subject individually by averaging the values of ISI 8, 10, and 15. Subjects were ranked according to their lifting work W_m. Subjects with high W_m started from high baseline ICF values, whereas subjects with low W_m had low ICF baseline values (cf. Fig. 4A). Overall linear correlation showed statistical significance ($r = 0.63$; $P = 0.0014$). After exercise, correlation between W_m and ICF remained significant ($r = 0.44$; $P = 0.036$), but the correlation line flattened (cf. Fig. 4B). The change in ICF (ΔICF) due to exercise was calculated as ICF after minus ICF before exercise. Subjects with higher W_m showed markedly decreased ΔICF whereas subjects with low W_m showed only a slight decrease or even a slight increase in ΔICF (cf. Fig. 4C). Linear correlation between ΔICF and W_m was statistically significant ($r = 0.73$, $P < 0.0001$).

DISCUSSION

The principal findings of our study are presented in Figure 3 showing that muscle fatigue after exhausting physical strain—in this study a maximum number of pull-ups—is accompanied by reduced ICF. We will argue that reduced ICF by double-pulse TMS reflects central fatigue specifically for the part of the motor cortex involved in the task. To the best of our knowledge, it is shown for the first time that the baseline ICF values and

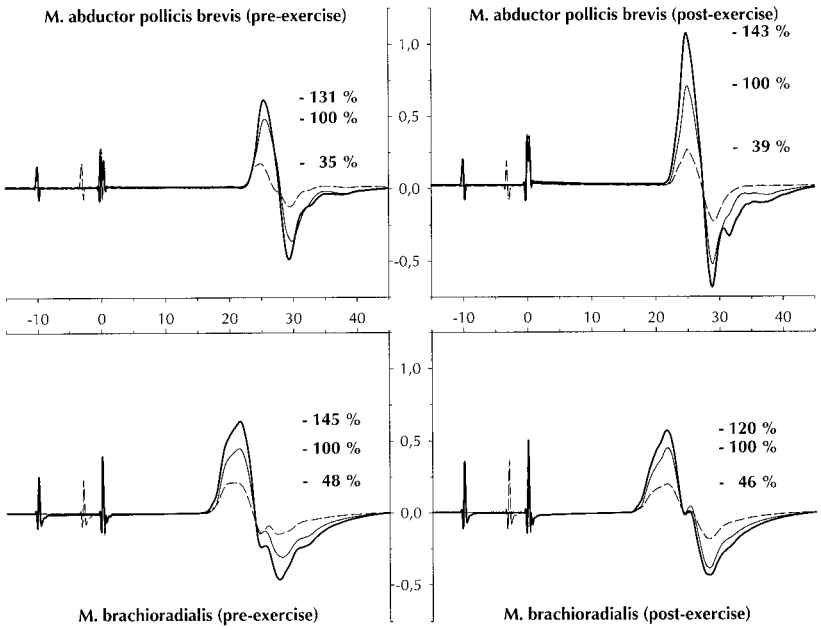

Figure 2—Surface EMG recordings from a representative subject (*open circle* in Fig. 4) are displayed for both muscles before and after exercise. Traces are averages of the 15 trails. The test MEP after the test pulse alone (*thin solid line*) and double-pulse MEPs after inhibitory (3 ms, *dashed line*) and after facilitatory (10 ms, *thick solid line*) interstimulus intervals are displayed. The left part of each trace shows the stimulus artifacts: the test pulse at 0 ms, the conditioning pulse 3 or 10 ms before the test pulse. Percentages indicate the MEP size referring to the test MEP. Reduction of MEP size is only seen for the facilitatory 10 ms double-pulse MEP in the BR (right lower diagram). Due to the increase of test stimulus intensity, MEP sizes in the APB after exercise were enlarged (upper right diagram) but double-pulse MEPs show only small changes relative to the test MEP.

also the changes in ICF correlate with the physical work accomplished. Double-pulse TMS much better than the single-pulse technique may be of interest in the control of training in sports.

Limitations of single-pulse TMS. By stimulating the motor cortex and recording MEPs from a target muscle, TMS gives access to the entire neuronal pathway from the motor cortex to the muscle performing a task. Using the single-pulse technique, numerous studies have demonstrated changes in the central nervous system during and after exhaustive exercise (e.g., 4,6,12,13,16,22,27,30,33). From those studies, it was suggested that the reduction of MEP amplitudes after exhaustive exercise corresponds to muscle fatigue. From unchanged M-waves (6,33), H-reflexes (6), F-waves (33), and MEPs after magnetic stimulation of spinal roots (16), it was discussed that the mechanisms representing fatigue mainly originate upstream from the spinal level. Furthermore, the comparison between TMS and transcranial electrical stimulation (TES) is argued to point to a cortical origin of this phenomenon: TES is assumed to activate corticospinal neurons directly preferentially at the axon hillock. In contrast, TMS is known to elicit corticospinal discharges transsynaptically through intracortical interneurons (9,11). Thus, it was concluded that, under fatigue, depressed MEPs by TMS but not by TES were due to these cortical differences (6,33). In support, another parameter of TMS, the so-called "silent period" was assumed to reflect enhanced inhibition of intracortical neurons when it was found prolonged after fatiguing exercise (22,30). Nevertheless, some observations remain unclear from those studies: In fact, MEPs by TES under fatigue were also markedly reduced by 15–30% (6,33), which was half as much as was seen by TMS. The silent period after electrical brain stem stimulation also showed prolongation half as much as was seen after TMS (30). Investigating a fatiguing task, Mills and Thomson (24) described a significant decrease of M-waves but failed to find central fatigue by TMS. Thus, we have to assume that during muscle fatigue electrophysiological changes occur at several stages of the cortico-muscular pathway (e.g., (29)). Because fatigue is defined by a loss of muscle force, accompanying electrophysiological changes may or may not contribute to fatigue. Moreover, unchanged M-waves, F-waves, and H-reflexes do not exclude at all fatigue downstream from the spinal level.

Keeping this in mind, single-pulse TMS in combination with spinal reflexes, M-waves or TES as described in the above mentioned studies may not be a sufficient tool to assess central fatigue. For quantifying fatigue, TES, M-waves, H reflexes, F-waves, and spinal root stimulation are neither sensitive nor reliable enough and, in addition, these methods are inconvenient.

The new double-pulse TMS technique. The conditioning-test double-pulse paradigm first described by Kujirai et al. (17) is thought to be a suitable technique to investigate at motor cortex level inhibitory and excitatory interneuronal circuits, at least for the following reasons: Subthreshold magnetic conditioning stimuli such as those used here have no effect on the excitability of spinal motoneurons tested by H reflexes (17,37), and the effect of the conditioning stimulus was assumed to take place at a cortical level (10). Furthermore, as mentioned above, TMS, in contrast to TES, is assumed to activate cortico-spinal neurons indirectly via intracortical interneurons (9,11). Subthreshold magnetic conditioning stimuli

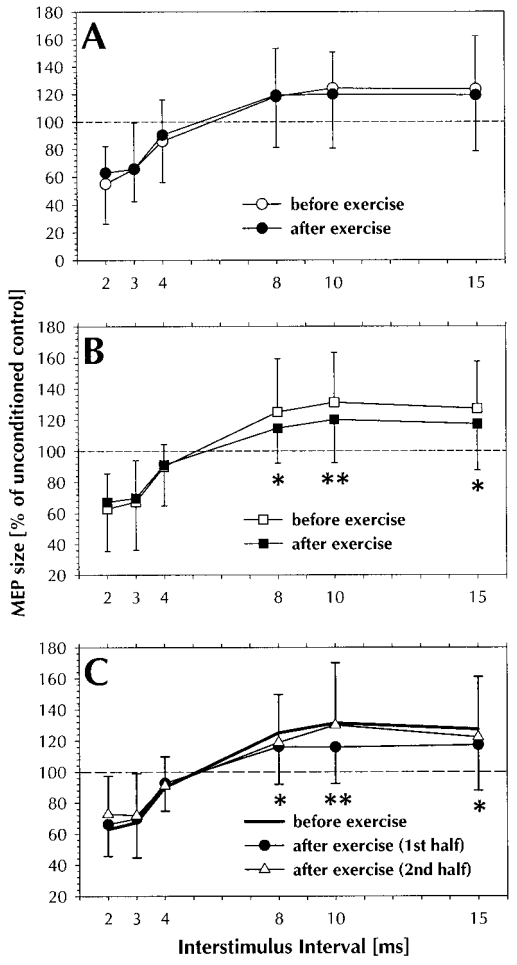

Figure 3—Mean values of intracortical excitability over all subjects as obtained by the paired conditioning-test-stimulus paradigm. Results for both muscles investigated, APB and BR, are displayed plotted in diagram A and B, respectively. The conditioned MEP size after paired TMS is expressed on the ordinate [as percentage of the unconditioned test MEP size]. The abscissa indicates the interval [in ms] between the conditioning and the test stimulus. *Open and filled symbols* represent pre- and post-exercise measurements, respectively. C: Postexercise values of the BR were separated into the first (*filled circles*) and second (*open circles*) half of the measurement in comparison to the values before exercise (*solid line*). The diminution of facilitatory MEPs seems reversible within 8 min after exercise. *Error bars* represent standard deviation. Statistical significance is marked by * ($P < 0.05$) and ** ($P < 0.01$).

have a stronger effect on following test MEPs by TMS than by TES (17,37), which points to an intracortical interaction. Thus, diminution and augmentation of MEPs after double-pulse TMS reflect inhibitory and facilitatory interneuronal circuits. Because double-pulse MEPs are referred to the single-pulse test MEPs (and both MEPs are elicited via the same motor pathway neurons), changes are specific for the motor cortex irrespective of alterations of the motor pathway downstream from the spinal level. Therefore, we can conclude that reduced ICF after exhaustive strain indicated a less excitable motor cortex. Although the term "fatigue" itself is not defined by changes in EMG response, the intracortical changes for some reason may be seen as cortical fatigue. Indeed, we cannot prove directly that the intracortical changes are responsible for the fatigue. However, it seems very likely that a decrease in motor cortex excitability is at least in part involved in the mechanisms leading to a loss of force. Nevertheless, many other central and peripheral mechanisms are surely involved, not only since ICF reduction was reversible within 5–8 min, whereas the force should have taken some more time to recover.

Some TMS studies with single-pulse technique found MEP facilitation shortly after the exercise followed by the MEP depression (e.g., 27). In our study, we did not find MEP facilitation in the target muscle, which is probably due to the time gap of 1–2 min between the last pull-up and the postexercise measurement.

Mechanisms influencing ICF. What are the mechanisms underlying reversible reduction of ICF? Extensive pharmacological studies (e.g., 36) have shown that intracortical inhibition and facilitation as partially separate mechanisms (37) are mainly controlled by γ-aminobutyric acid (GABA)-ergic mechanisms. The decrease of ICF found here is reminiscent of reversible effects of benzodiazepines (35).

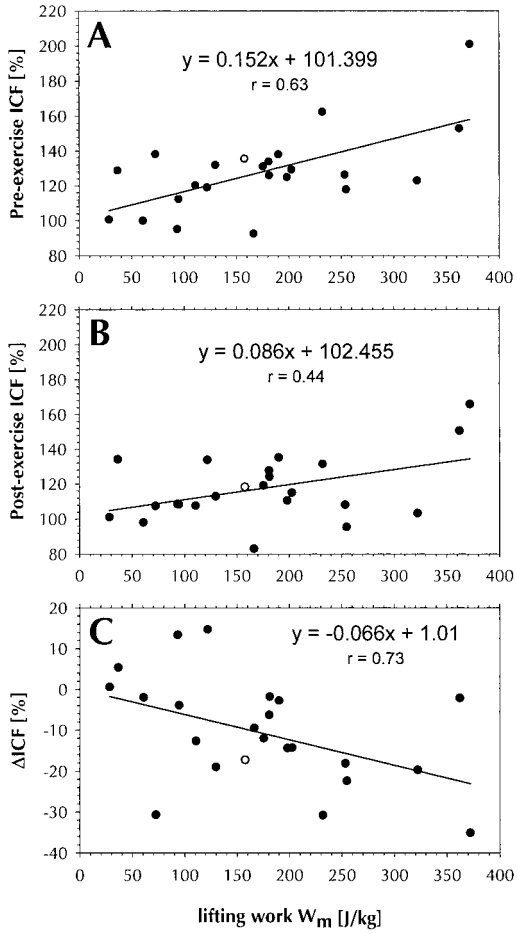

Figure 4—The values of intracortical facilitation (ICF, as average of the relative MEP sizes for interstimulus intervals of 8, 10, and 15 ms) are plotted for every subject separately and the calculated line (including the equation) of linear correlation analysis to the individual lifting work W_m is shown. Values of ICF are displayed on the ordinate [as percentage of the test MEP], lifting work W_m is indicated on the abscissa [in $J \cdot kg^{-1}$]. A, Values of the preexercise measurement. B, Values of the postexercise measurement. C, The diminution in ICF is indicated on the ordinate [as difference in the percentage of test MEP amplitude]. *Open circle*, representative EMG traces of this subject are shown in Figure 2.

But the effects of inhibition and facilitation within the motor cortex are obviously influenced by other brain areas since it has been described that, e.g., motor diseases as well as ischemic brain lesions (15,31) may influence ICI and ICF. Furthermore, glutamatergic agents as well as dopaminergic and antidopaminergic drugs are also believed to affect motor cortex neurons via GABA-ergic mechanisms (34,38). Thus, reduction of ICF indicates decreased motor cortex excitability upstream from the motor cortex output neurons. Further studies are needed to clarify the neurotransmitter systems and the higher brain regions involved in this mechanism.

Are the results due to an artifact? To investigate the intracortical changes independently from spinal, α-motoneuronal, or peripheral alterations, we decided to keep the test MEP constant. Although the thresholds for eliciting small MEPs did not change significantly (Fig. 1B), higher test pulse intensities for the postexercise measurement were needed (Fig. 1C) to produce test MEPs of a given amplitude (0.75 mV in the BR). These findings are congruent with those TMS studies showing reduced MEPs after fatiguing exercise (see above). Nevertheless, with this adjustment of the test pulse, we could have induced the changes in ICF artificially. This, however, is unlikely, because only ICF but not ICI in the BR was changed. Even ICI and ICF in the APB remained unchanged, although APB test MEPs were markedly increased by this procedure (Fig. 1C). Why were test pulse MEPs in the BR reduced? This may refer to the mode of neuron activation by TMS: Brasil-Neto et al. (4) postulated a "decreased efficiency in the generation of the descending volleys in the motor cortex" under fatigue. It is known that one single TMS pulse like the test pulse used here elicits multiple descending volleys in one cortico-spinal neuron (so-called I-waves) (9,11). Those I-waves are controlled within the motor cortex and can be suppressed by GABA-ergic mechanisms (39). Thus, reduced MEP recruitment may be due to a GABA-mediated loss of cortico-spinal volleys reflecting fatigue of the motor cortex. Reduced ICF despite of the counterbalanced reduction of test MEP gives evidence that the observed effect was true.

From our data, we also may conclude that cortical fatigue occurred specifically for the muscle involved in the task. ICF was reduced for the BR but not for the APB of the same limb (Figs. 2 and 3). This seems to be in line with the study of Taylor et al. (30) showing silent period prolongation specifically for the muscle involved in an exercise, although it is not clear yet why McKay et al. (22) also found a silent period prolongation in a contralateral control muscle. Further studies have to focus on the distribution of fatigue within the ipsi- and contra-lateral cortex.

ΔICF in the quantification of cortical fatigue. Support of the hypothesis that ICF reflects fatigue (see above) derives from the findings that the reduction of ICF was significantly correlated to the normalized work W_m (Fig. 4C). Subjects who were able to perform a great number of pull-ups, thus resulting in a high value of W_m, showed a marked decrease of ICF. In contrast, subjects who were only able to perform a small number of pull-ups showed only a slight decrease or even an increase of ICF. This might be due to the subject's inability to perform pull-ups mainly because of low muscle capacity rather than fatigue, because a relatively high power is necessary to pull up one's own body mass. The increase in ICF seems to be similar to what other authors described as postexercise facilitation under exercises before fatigue occurred (6,22,27,30). Nearly all subjects reached exhaustion under exercise and in subjects with high W_m the drop in ICF was marked (cf. Fig. 4, B and C). That means subjects with high capacity needed more reduction of power compared with subjects with lower capacity to meet the study's criteria of fatigue. From our data, we conclude that the assessment of ΔICF may be a useful tool not only to demonstrate but to quantify fatigue of the motor cortex after exhaustive physical strain.

The reduction of ICF after exercise investigated here was reversible after approximately 5–8 min. Nevertheless, the course of ICF recovery, which was not investigated in detail here, should be the aim of another study.

Preexercise ICF as a tool in training control? As shown in Figure 4A, the value of ICF before exercise is significantly positively correlated to the normalized work W_m. Subjects with high capacity of work started from a higher level of ICF. Although we cannot exclude that ICF was a constant inherent individual parameter, it seems very likely that increased preexercise ICF was a result of plasticity of the motor cortex due to specific training of those movements. Classen et al. (8) have shown that short-term training of particular, not-fatiguing movements improves cortical excitability of the neurons needed for the task. In our study, ICF seems to reflect long-term changes of the motor cortex. Thus, ICF measures the subject's cortical constitution due to training, and it may be hypothesized that ICF could be used as a parameter of cortical capacity or resistance against cortical fatigue in training control.

Central fatigue, overreaching, and overtraining. Overreaching and overtraining are short- and long-term conditions of reduced physical capacity in endurance athletes (7,19,25). Although the former may be seen as a normal part of athletic training, the latter describes a pathological state of burn-out with persistent performance incompetence and high rates of fatigue. Among a variety of factors—such as cardiopulmonary function, blood-chemical and neuroendocrine parameters as well as mood state—elevated activity of GABA or an endogenous benzodiazepine-like substance are discussed as responsible mechanisms in the early central fatigue in the overtraining syndrome (18). This could be in line with the observations mentioned above that benzodiazepines have the same reducing effect on intracortical excitability as seen under fatigue. It may be assumed that ICF can be used as a parameter helping to detect overreaching and to prevent overtraining in endurance sport. Further studies to prove this hypothesis have already begun.

Address for correspondence: Dr. Frithjof Tergau, Department of Clinical Neurophysiology, University of Göttingen, Robert-Koch-Straβe 40, D-37075 Göttingen, Germany; E-mail: f.tergau@med.uni-goettingen.de.

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