A Neurophysiological Approach to Functional Neurology
“Giornate Pavesi di Neurofisiologia Clinica”

Pavia, 8 November 2002

ABSTRACTS

Edited by
A. Moglia, G. Nappi, G. Sandrini
DIRECT DEMONSTRATION OF CENTRAL FATIGUE IN HUMANS: A TRANSCRANIAL MAGNETIC STIMULATION STUDY

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We recorded descending corticospinal activity evoked by transcranial magnetic stimulation of the motor cortex directly from the high cervical epidural space of three conscious patients who had electrodes inserted for control of intractable lower back pain. A 9 cm diameter figure-of-eight stimulating coil was held so as to induce electric current in a latero-medial direction across the motor cortex. This orientation stimulates corticospinal axons directly in the subcortical white matter (D waves) and also activates the same neurones trans-synaptically via cortico-cortical connections in the grey matter (I waves).

We compared the corticospinal volleys and EMG responses in the first dorsal interosseous (FDI) muscle evoked by transcranial stimulation before and after a strong two-minute voluntary pinch contraction of the index finger and thumb. Contraction had a significant effect on the amplitude of all the descending volleys and on the EMG responses that they evoked in the muscle. The effect on I waves lasted longer than that on D waves or EMG responses; D waves and EMG responses were suppressed at 1 and 2 minutes after the contraction, while I waves were suppressed at 1, 2 and 3 minutes after the contraction. All parameters returned to baseline values after 10 minutes. The effect was substantial: in the first minute after contraction, the total amplitude of descending waves decreased by 50% in these three subjects. The consequence of these changes can be observed in the EMG responses we recorded in the FDI muscle. These were reduced to about 40% of their pre-contraction size 1 minute after the end of contraction.

Acknowledgments: This work was supported by the Italian Health Ministry (Programma di ricerca finalizzata 2001 - Mecanismo centrali e periferici della fatica muscolare).

MEG/EEG–EMG COHERENCE IN DIFFERENT ISOMETRIC CONTRACTION STATES AND FOLLOWING FATIGUE

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Corticomuscular coherence between cerebral cortical rhythms and surface electromyography (EMG) has been observed since the 90s both through electrocorticogram and electroencephalographic (EEG) / magnetoencephalographic (MEG) recordings. Corticomuscular coherence is a tool for the identification and characterization of cortical districts controlling a particular body district, and it is based on frequency selection of the spectral components present in the signals describing cephalic and muscular activities. The coherence of the two signals for each frequency component is quantified by a value between 0 and 1 (= maximal coherence).

Significant levels of coherence have been described below 40 Hz. A clear frequency specificity has been observed, with significant coherence found at around 40 Hz for strong (Piper rhythm) and at around 20 Hz for weak (beta band) isometric contractions. In particular, while coherence at around 40 Hz continues to be significant during phase contraction, in this condition it loses significance in the beta range.

No significant contraction level-/coherence frequency-dependent difference in cortical region identification has been detected.

Our unit has decided to focus on interhemispheric relationships of coherence, and their modifications due to muscular fatigue. Moreover, in relation to fatigue the relationship between sensory and motor areas for hand control will be evaluated in the two hemispheres.

With this aim, MEG and EEG recordings of sensorimotor contralateral cerebral activity and muscular activity from right and left extensor carpi radialis (ECR) will be studied. Subjects will be required to contract separately their left and right ECR, at 30% of maximal voluntary contraction force, for periods lasting around 20 sec. The evaluation will be repeated following maximal contraction maintained as long as possible. Coherence, as well as activated cerebral source and sensory evoked response morphology will be analyzed, to characterize both motor and sensory cortical representations.

MULTIPARAMETRIC ANALYSIS OF MUSCULAR, CARDIOPULMONARY AND METABOLIC FUNCTIONS DURING EXERCISE IN SUBJECTS WITH MYOPATHIES

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Sports physicians investigate fatigue through the study of metabolic parameters that indicate the onset of “an acute limitation of performance that implies an increased perception of the effort to sustain a certain workload, as well as the inability to sustain intensity, length and eventually imposed pace”. These parameters can be investigated directly (maximal oxygen consumption, blood lactate, etc.), or be derived (i.e., from heart rate or ventilation). This kind of analysis also lends itself well to the study of neurological disease, and of myopathies in particular. Examining cases of mitochondrial myopathy, we showed an important reduction of working capacity and peak oxygen consumption, with a premature increase of blood lactate (that in more severe cases was already high in the resting condition).
during maximal cycloergometer stress test, in respect to normal values.

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<thead>
<tr>
<th></th>
<th>Sedentary</th>
<th>PEO</th>
<th>MERRF</th>
</tr>
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<tr>
<td>peak V\textsubscript{O}_2</td>
<td>1715</td>
<td>937</td>
<td>716</td>
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<tr>
<td>Max load (Watt)</td>
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<td>40</td>
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<tr>
<td>Basal BL mM</td>
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<td>1.9</td>
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<td>BL at max load (mM)</td>
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<td>4.7</td>
<td>5</td>
</tr>
<tr>
<td>10\textsuperscript{th} Rec BL (mM)</td>
<td>2.2</td>
<td>6.1</td>
<td>9.0</td>
</tr>
</tbody>
</table>

Metabolic parameters during maximal cycloergometer stress test in a sedentary subject, in one subject with progressive external ophthalmoplegia syndrome (PEO), and in one with myoclonus epilepsy with ragged red fibers syndrome (MERRF). Legend: V\textsubscript{O}_2 = oxygen consumption; BL = blood lactate.

CENTRAL AND PERIPHERAL MECHANISMS OF NEUROMUSCULAR FATIGUE


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Fatigue is a common experience in our daily life. It can be defined as “any reduction in the maximal capacity to generate force or power output”. Many mechanisms are responsible for fatigue and they can act at different levels in the central and peripheral nervous systems. Central fatigue is reflected in exercise-induced reduction in maximal voluntary contraction accompanied by a parallel reduction in maximal force (changes in activation of the motoneurons including motivational factors as well as integration of sensory information leading to generation of action potentials in the sarcolemma). Different methods can be used to highlight central factors of fatigue: magnetic stimulation of the motor cortex, twitch interpolation, velocity changes in repetitive voluntary twitches, ratio between mechanogram produced by maximum voluntary contraction force and tetanic mechanogram. Peripheral factors of fatigue can be investigated through low frequency fatigue methods, twitch parameter changes after effort, and tetanic mechanogram changes. Kind of exercise is a very important factor when seeking to identify central or peripheral mechanisms of fatigue.

FATIGUE OF NECK MUSCLES IMPAIRS POSTURAL CONTROL

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Neck problems may cause dizziness or imbalance, sometimes termed “cervical vertigo”. Objective assessment of postural stability has yielded conflicting indications, although subjects barely complain of instability under static conditions. We hypothesised that, since anomalous neck muscle input can produce postural effects, neck muscle fatigue could favour the onset of dizziness through a mechanism connected to abnormal afferent inflow.

Eighteen normal subjects underwent 5-min fatiguing contraction of head extensors. Sway during quiet stance was recorded by a platform both prior to and after fatigue and recovery. Subjects exerted a force 25-35% of maximal voluntary effort against a device exerting a head-flexor torque. EMG activity from dorsal neck muscles was recorded. EMG median frequency progressively decreased and EMG amplitude progressively increased during the fatiguing contraction, providing evidence of the occurrence of muscle fatigue.

Stabilometric recording showed an increase in body sway immediately after fatigue, particularly when the eyes were closed. Prolonged muscle contraction per se was not responsible for the observed effects on sway, since lower-weight loads did not produce either EMG signs of fatigue or increased sway.

We conclude that intra-muscular neural afferents sensitive to metabolites produced by muscle work or “continuous muscle tension” could be responsible for impaired postural control.

MUSCLE SYMPATHETIC NERVE ACTIVITY AND ITS RELATIONSHIP WITH MUSCLE FATIGUE: AN EXPERIMENTAL SETTING

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Striated muscle sympathetic nerve activity (mSNA) is influenced (increased) by muscle contractions. The increase in sympathetic drive occurs via muscle chemoreceptor discharge activated by metabolites produced in muscle when it contracts. This activation of sympathetic drive is a cardiovascular adaptive mechanism, being part of the regulation of blood pressure during physical activity.

Many data are available on how physical exercise modifies, by activating it, vasomotor sympathetic drive. In contrast, few studies have investigated the possible
inverse interaction between sympathetic activation and muscle, i.e., whether the sympathetic nervous system is able to influence striated muscle activity with particular reference to the mechanisms of muscle fatigue. This is particularly important in pathologies such as Parkinson’s disease in which the sympathetic outflow is altered and fatigue is one of the main complaints.

A major problem arises when planning experiments to evaluate whether sympathetic activation is in some way correlated to changes in parameters of localised muscle fatigue.

A suggested experimental setting is the following:

a) Sympathetic activation is induced by a period of post-handgrip ischemia for 3 minutes.

b) Tibialis anterior muscle activity is electrically provoked and assessed by surface electromyography before and after sympathetic activation. Median frequency (MDF) and muscle fibre conduction velocity (CV) should be calculated.

c) Sympathetic activation of striated muscles (mSNA) is followed throughout the experiment by means of multifibre intraneural recordings. A tungsten needle electrode (tip diameter 2-5 micron) is percutaneously inserted into a muscle fascicle of the peroneal nerve of the fibular head of the non-fatiguing leg. mSNA is calculated per time unit (mSNA/min) and per 100 heart beats (mSNA/HB) and normalised to 100.

Acknowledgments: This experimental setting has been used within the framework of the research: Meccanismi centrali e periferici della fatica muscolare, SP 2001 MinSan, UO6.