The use of a Brain Computer Interface (BCI) in Gait Rehabilitation of Stroke Patients

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Introduction

Stroke has an incidence of 150/100,000 inhabitants per year, 30-40% of which will present invalidating sequelae [1]. It may affect a wide variety of body functions, including walking capacity. Improvement of function will be seen mainly in the first 3 months [1], and it may extend further up to 6 months. Short-term improvement is due to the reperfusion of the ischemic penumbra while long-term improvement (usually scarce) will be due to neuroplasticity.

Even though little improvement in strength is usually seen, motor control training and adaptations can improve function and participation, exceeding what would be expected if only strength was taken into account. Rehabilitation will enable the achievement of these goals. However, current neurorehabilitation treatment is time-consuming and results are suboptimal if a complete recovery is aimed. In these rehabilitation programs, patients perform passive and active movements as well as proprioceptive exercises hoping that these will restore normal motor patterns.

A current and novel approach is the ability to potentiate neuroplasticity through correct timing of interventions. This is achieved through the combination of the top-down and bottom-up approaches. It has been demonstrated that, if both motor intention and sensory biofeedback are precisely timed, neuroplasticity may be enhanced beyond what current rehabilitation programs are able to achieve and this may translate into better walking performance [2, 3].

During gait, lower limb muscles can be classified into two main groups depending on their overall function: 1. restoration of spring torque (regarding lower limb elasticity), and 2. restoration of energy lost in each stride. The first group comprises proximal lower limb muscles (quadriceps and ischiotibial muscles) and are in charge of modulating walking cadence and hence speed, while the second group includes distal lower limb muscles [4].

Aim

1. Confirm in a larger population of chronic stroke patients the increase in corticospinal excitability after a training session using a BCI.
2. Quantify the clinical implications of such treatment, taking into account the results obtained in the feasibility study [5].

Material and Methods

Patient characteristics
- Chronic lacunar stroke
- Some degree of hemiparesis
- Able to walk

Clinical Assessment

We will use both objective biomechanical measures as well as clinical evaluation scales, both for balance and gait performance. We will evaluate overall gait performance and fall risk with IBV FallSkip. Independence will be determined by means of the Barthel Index.

TMS Assessment

Corticospinal excitability will be used to measure synaptic enhancement and therefore the potential to induce neuroplasticity. It will be determined by generating and quantifying the motor evoked potentials (MEP) through the use of single-pulse transcranial magnetic stimulation (TMS) and surface electromyography (sEMG) on rectus femoris, biceps femoris, tibialis anterior and soleus. This procedure is performed before, immediately after and 30 minutes after the training session with the BCI.

Brain-computer Interface

The BCI will detect movement-related cortical potentials (MRCPs) by means of EEG when the patient is asked to initiate a volitional lower limb cycling movement. Functional electrical stimulation (FES) over rectus femoris, which is one of the main muscles involved in walking speed, will be timely activated in order to assist the cycling movement.

Discussion

Current neurorehabilitation treatments lack integration of both the bottom-up and top-down approaches. By means of a BCI we will be able to couple motor intention (MRC) with afferent sensory feedback (caused by FES) and hopefully enhance neuroplasticity beyond what is currently being achieved. However, it is not clear whether this neuroplasticity will suffice to increase walking speed and/or improve overall function and participation.

References