Why is the neural control of lengthening contractions considered so unique?

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A large number of studies from the last few decades have established that, during lengthening contractions, the control strategy employed by the nervous system differs from those used during isometric or shortening ones (for a review see [1]). Most of the studies first concluded that the muscle is not fully activated during a lengthening maximal voluntary contraction (MVC), as supported by a decrease in electromyographic activity (EMG) that may occur [2], and/or by a voluntary activation level often not maximal [3], when compared to isometric and shortening MVCs. More recently, the interaction between cortical and spinal neural mechanisms involved in the modulation of the neural drive during lengthening contractions has been investigated by different research groups by comparing the effect of the muscle contraction type on: i) the motor-evoked potential elicited by transcranial magnetic stimulation [4], ii) ~~on~~ the cervicomedullary motor-evoked potential [5], and iii) ~~on~~ the Hoffman-reflex obtained by electrical stimulation [6]. In most cases, a specific depression of the corticospinal excitability was found during lengthening contractions, which was mainly attributed to pre and post synaptic inhibitory mechanisms acting at the spinal level, although their relative contribution may be modulated by the descending neural drive from supra-spinal centres [7].

This interaction between cortical and spinal neural mechanisms could be investigated by computing corticomuscular coherence (CMC), i.e., the frequency coupling between electroencephalography and EMG oscillatory activities. While CMC has mostly been investigated during isometric contractions (for a review see [8]), we recently were the first to quantify CMC during both isokinetic lengthening and shortening contractions compared to isometric contractions [9]. We thus highlighted a specific decrease in CMC during lengthening contractions compared to isometric ones, with a concurrent decrease in spinal excitability. This specific behaviour indicates a decrease of the mutual interactions between cortical and muscle oscillatory activities, associated with a higher spinal inhibition, during lengthening contractions.

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