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Coupling between human brain activity and body movements: Insights from non-invasive electromagnetic recordings

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ABSTRACT

Electroencephalographic and magnetoencephalographic data have characterized two types of brain–body interactions observed during various types of motor actions, "*corticokinematic*" and "*corticomuscular*" coupling. Here, we review the literature on these interactions in healthy individuals, discuss several open debates, and outline current limitations and directions for future research.

Corticokinematic coupling (commonly referred to as corticokinematic coherence) probes the relationship between activity of sensorimotor network nodes and various movement-related signals (*e.g.*, speed, velocity, acceleration). It is mainly driven by movement rhythmicity during active, passive, and observed dynamic motor actions. It typically predominates at the primary sensorimotor cortex contralateral to the moving limb, occurs at movement frequency and its harmonics, and predominantly reflects the cortical processing of proprioceptive feedback driven by movement rhythmicity in a broad range of dynamic motor actions.

Corticomuscular coupling (commonly referred to as corticomuscular coherence) probes the interaction between sensorimotor cortical rhythms and electromyographic (EMG) activity that mainly occurs during steady isometric muscle contraction. We will here focus on the ~20-Hz coupling that is observed during weak isometric contraction and is linked to the modulation of the descending motor command by the ~20-Hz sensorimotor rhythm.

This review argues that *corticokinematic* and *corticomuscular* couplings have different neural bases. *Corticokinematic* coupling is mainly driven by afferent signals, while *corticomuscular* coupling is mainly (but not solely) driven by efferent signals. This distinction should be considered when investigating interactions between brain and body movements.

1. Introduction

The central nervous system interacts with body parts through peripheral and autonomic nervous systems. Efferent neural pathways originating from motor, premotor, as well as somatosensory neocortical areas convey the motor command through the spinal cord and efferent peripheral nerves to control voluntary limb and body movements. By contrast, afferent spino-cortical and spino-cerebellar neural pathways contribute to somatosensory perception and sensorimotor feedback for motor control. How the human brain (i) generates voluntary efferent motor actions (static or dynamic), (ii) processes afferent somatosensory information, and (iii) integrates both efferent and afferent information to achieve efficient sensorimotor control are major questions that have been the topic of extensive research in animals and humans for several centuries. The advent of human functional neuroimaging has paved the way for the non-invasive investigation of human brain activity to address

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these fundamental questions. Additionally, electrophysiological techniques, such as electroencephalography (EEG) and magnetoencephalography (MEG), that have an exquisite millisecond temporal resolution, have brought critical knowledge about the temporal and spectral dynamics of sensorimotor neural processes (Hari and Puce, 2017). In particular, these electrophysiological techniques have allowed scientists to closely investigate these processes during brain–body *interactions* ("interaction" is here used to refer to *synchronization*, i.e., a term referring to the adjustment of two ongoing oscillations). This research line has highlighted two main types of brain–body interactions during various types of motor actions: "*cortex–kinematic*" interactions that we shall refer to as *corticokinematic coupling* (CKC; often called corticokinematic coherence), and "*cortex–muscle*" interactions that we shall refer to as *corticomuscular coupling* (CMC; classically called corticomuscular coherence, cortex–muscle coherence or cerebro-muscular coherence depending on the authors). Coupling is here used to refer to statistical dependencies between two signals. Coherence has been the most commonly used coupling measure to study these brain-body interactions. Coherence analysis is a direct generalization of the correlation analysis to the frequency domain (see Fig. 1). It quantifies (from 0, no association; to 1, perfect association) the degree of linear dependence (i.e., coupling) between two signals (here, between brain and peripheral signals) as a function of frequency (Halliday et al., 1995).

This paper will review MEG and EEG studies that have contributed to the characterization of CKC and CMC in healthy subjects during various types of motor actions such as isometric contractions or active, passive, and observed dynamic movements. In particular, for both types of coupling, we will first describe the coupling frequency and associated neural generators, discuss their neurophysiological basis (including the efferent vs. afferent contributions), and lastly develop some perspectives for their use in human neuroscience. We will finally highlight that these couplings actually index two different brain–body interactions that may co-occur during certain types of motor actions.

Of note, we will not review here the extensive literature on "*muscle–muscle*" interactions that is thought to build on different mechanisms than CMC (Boonstra, 2013; Boonstra et al., 2009). Nor will we cover the alterations of CKC and CMC described in various disorders of the nervous system (see, *e.g.*, Sridharan et al., 2019). Finally, we will not cover the use of transcranial magnetic stimulation to probe cortico-spinal interactions (for a review on the topic, see, *e.g.*, Valero-Cabré et al., 2017).

2. Corticokinematic coupling

2.1. Coupling frequency and neural generators

Human scalp EEG and MEG recordings have demonstrated a robust relationship between time-varying brain activity and movement velocity (O'Suilleabhain et al., 1999). Of note, "movements" here refer to dynamic motor actions characterised by noticeable change in muscle length and joint angle. Using advanced source reconstruction methods and complex visuomotor adaptation tasks, MEG studies identified significant coupling between slow (2–5 Hz) neural activity at the primary motor (M1) cortex contralateral to hand movements and time-varying hand movement velocity (Bradberry et al., 2009; Jerbi et al., 2007).

Subsequent studies used a MEG-compatible 3-axis accelerometer to extend these seminal findings to movement acceleration. Indeed, significant coupling between finger movement acceleration and MEG signals was observed at the primary sensorimotor (SM1) cortex contralateral to movements during active (repetitive) non-goal-directed and goal-directed finger movements (Bourguignon et al., 2012, 2011; Marty et al., 2015a). This coupling was then coined CKC, because both velocity and acceleration are kinematic parameters. CKC typically peaks at finger movement frequency (F0, typically <5 Hz for active movements) and its first harmonic (F1), with its main cortical source located at the SM1 hand area contralateral to finger movements (Bourguignon et al., 2012, 2011; Jerbi et al., 2007) (see Fig. 2, ***left*** part). It is characterised by a high coupling level (typically 0.2–0.8 coherence level) and is seen in almost all subjects. CKC was also found within an extended sensorimotor network comprising the contralateral dorsolateral prefrontal cortex and the posterior parietal cortex, as well as the SM1 cortex and the cerebellar lobule VIII ipsilateral to movements (Bourguignon et al., 2012, 2011; Marty et al., 2018). Finally, CKC has been demonstrated during various movement rates (from ~1 Hz to 4 Hz) with no influence of the movement rate on CKC level and main source location (Marty et al., 2015b).

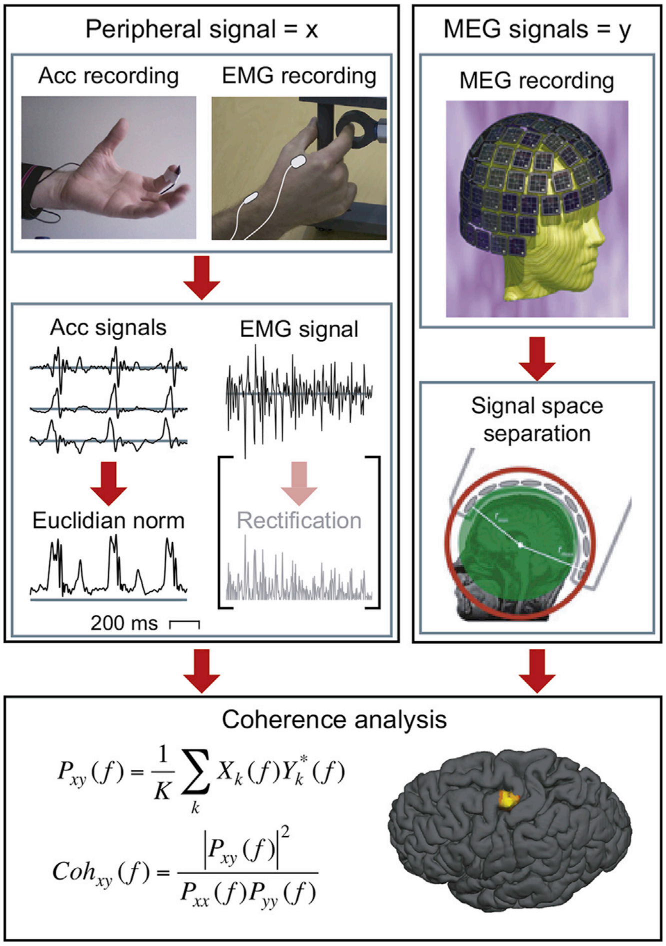

Fig. 1. Summary of the coherence method used to study brain-body interactions during movements. ***Top.*** Kinematic (*e.g.*, here acceleration) or electromyographic (EMG) signals are recorded simultaneously to electromagnetic brain signals (magnetoencephalography (MEG) as here, or electroencephalography) to estimate corticokinematic coupling or corticomuscular coupling (respectively). In the case of corticokinematic coupling here recorded using a three-axis accelerometer (Acc), the three orthogonal Acc signals are band-pass filtered and then combined into a single Acc signal using the Euclidean norm. For corticomuscular coupling, EMG rectification is optional and controversial. MEG signals are filtered using signal space separation (SSS) method to correct for head movements and subtract external interferences. ***Bottom.*** Both signals are epoched to compute coherence, which is a frequency-dependent measure of phase-coupling. Inverse modeling is then used to identify the brain areas of peak coherence level. This figure has been adapted from (Bourguignon et al., 2012).

CKC can also be estimated based on force, pressure, accelerometer and even *rectified* electromyographic (EMG) signals (Piitulainen et al., 2013a). This latter finding demonstrated that CKC is actually largely driven by movement rhythmicity/frequency. CKC can thus be properly estimated based on any type of peripheral signal, including surface EMG (see Fig. 2, ***left*** part), that accurately captures this movement rhythmicity/frequency.

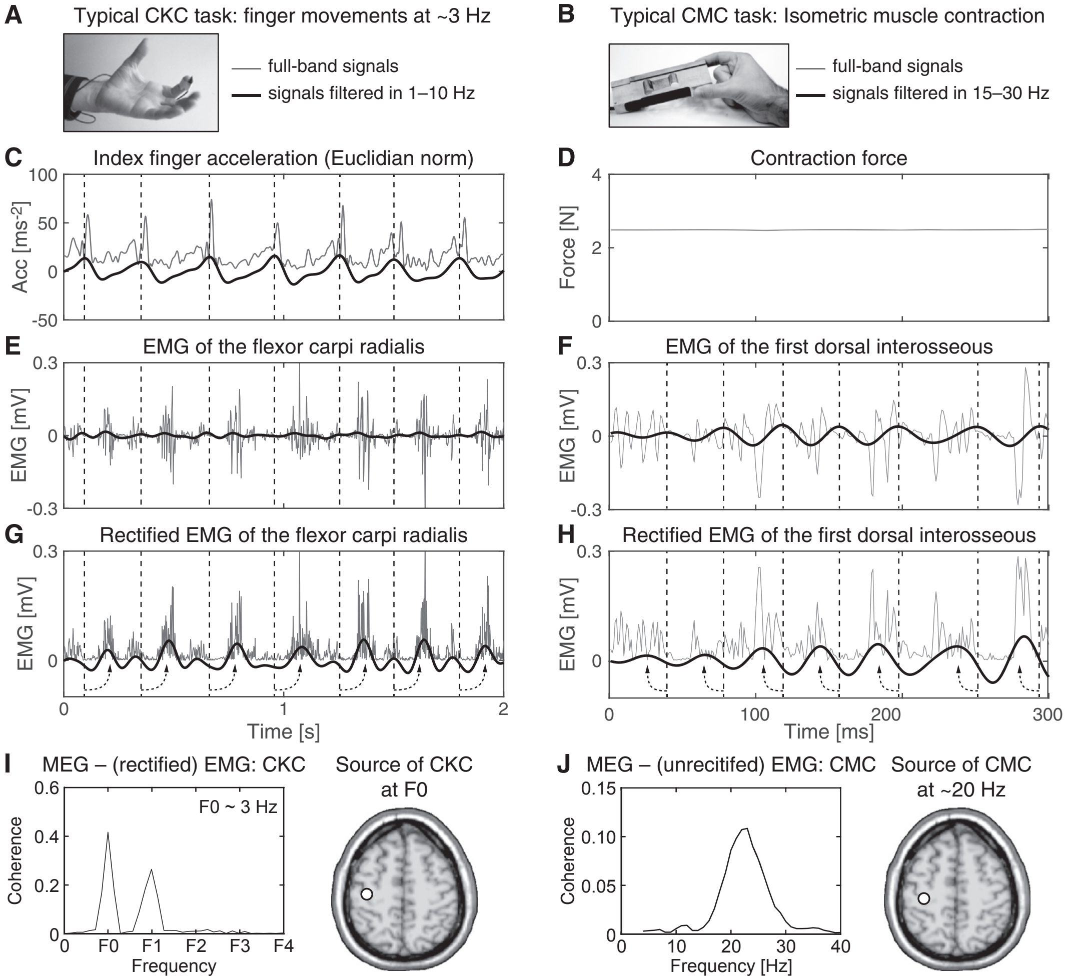

Fig. 2. Corticokinematic coupling (CKC, ***Left***) and corticomuscular coupling (CMC, ***Right***) based on surface electromyographic (EMG) recordings. CKC data are from a subject included in Piitulainen et al. (2013a) who performed ~3-Hz repetitive right hand movements. CMC data are from a subject included in Bourguignon et al. (2017) who performed an isometric pinch contraction of 2–4 N with the right hand. **A & B** — Typical experimental tasks to uncover CKC (A) and CMC (B). Through the figure, gray traces indicate full-band signals and black traces signals filtered through 1–10 Hz (CKC) or 15–30 Hz (CMC). **C & D** — Task monitoring with non-EMG recordings: acceleration (CKC; C) and force (CMC; D). **E–H** — Unrectified (E & F) and rectified (G & H) EMG signals from a muscle involved in the task. It is evident that unrectified and rectified EMG signals are phase-locked in both tasks, and also phase-locked with acceleration in the CKC task. For this reason, both CKC and CMC can be derived with rectified and unrectified EMG. Note, however, that in the case of CKC, rectification is highly recommended since slow EMG fluctuations are nothing more than movement-related artifacts. **I & J** — Coherence spectra with EMG and location of the dominant underlying cortical source at the primary sensorimotor cortex contralateral to hand action.

2.2. Neural basis of CKC

CKC has also been observed during passive movements of the fingers and toes either produced by an investigator or an MEG-compatible device based on elastic "pneumatic artificial muscles" (PAM) (Bourguignon et al., 2015; Piitulainen et al., 2013b). For more details about the PAM stimulator that predominantly elicits proprioceptive pathways stimulation, see Lolli et al. (2019) and Piitulainen et al. (2015b). The main findings of these studies were that repetitive passive movements led to strong CKC (coherence levels up to 0.8) with underlying sources located in a somatotopic manner at the contralateral SM1 hand or foot areas. The location of these sources was not affected by movement frequency. More importantly, CKC levels were similar or higher during passive movements compared with active movements with similar CKC brain sources (Bourguignon et al., 2015; Piitulainen et al., 2013b). This latter finding is of critical importance as it demonstrates that the absence of efferent signals in the passive condition does not affect CKC level, which highly suggests a negligible contribution of efferent motor processes in CKC. Also, CKC brain sources were significantly different in terms of location from those of evoked magnetic fields elicited by pneumatic tactile stimulation at the tip of the right index finger (Bourguignon et al., 2015; Piitulainen et al., 2013b). This finding provides support for the limited involvement of movement-related tactile information processing in CKC.

CKC was initially thought to be an efferent phenomenon presumably reflecting the encoding of kinematic plans at the level of SM1 cortex used to generate appropriate muscle forces via kinematics-dynamics transformation (Bourguignon et al., 2012, 2011; Jerbi et al., 2007). This hypothesis naturally followed from the knowledge that in non-human primates, some M1 cortex neurons encode numerous movement kinematic parameters (Ashe and Georgopoulos, 1994; Caminiti et al., 1990; Carmena et al., 2003; Kettner et al., 1988; Mehring et al., 2003; Moran and Schwartz, 1999; Reina et al., 2001). However, results obtained in the context of passive movements suggested that CKC is predominantly driven by proprioceptive inputs to contralateral SM1 cortex (Bourguignon et al., 2015; Piitulainen et al., 2013b). This hypothesis was subsequently supported by directionality analyses, which showed that the coupling was dominated by an afferent contribution (Bourguignon et al., 2015; Piitulainen et al., 2013b). As a final support to the dominant proprioceptive contribution to CKC, the level of CKC elicited by active and passive finger movements was reduced by 60–70% at contralateral

SM1 cortex in patients with Friedreich ataxia, which is a genetically-determined ataxic disorder mainly characterized by spino-cortical proprioceptive afferent and cerebellar pathways degeneration (Marty et al., 2019). These findings are in agreement with the fact that both the primary somatosensory (S1; Brodmann areas 3a and 2) and M1 cortices (Brodmann area 4) receive proprioceptive feedback during both active and passive hand movements (Goldring and Ratcheson, 1972).

CKC is therefore likely driven by somatosensory proprioceptive signals generated by muscle spindles, Golgi tendon organs, and possibly some mechanoreceptors of the skin activated by skin motion (*e.g.*, Pacinian corpuscles). These receptors indeed play a crucial role in monitoring movements of even a few millimeters in amplitude (Bourguignon et al., 2015; Marty et al., 2019; Piitulainen et al., 2013b). Movement rhythmicity activates extremely sensitive proprioceptors sensing the internal state of the moving musculoskeletal system, which in turn send synchronous afferent volleys up to SM1 neocortical areas contralateral to movements via spinocortical proprioceptive pathways (Piitulainen et al., 2013b).

The neural basis of CKC at F0 vs. F1 is still debated (Bourguignon et al., 2012; Marty et al., 2019). The fact that CKC peaks at both F0 and F1 may non-exclusively reflect cortical processing of different movement-related proprioceptive features, or follow from the non-sinusoidality of the brain and kinematic signals underpinning CKC (Bourguignon et al., 2012; Marty et al., 2019). In repetitive movements such as those used in previous studies, F0 likely reflects cycles of movements and corresponding afferent proprioceptive signals, while F1 might reflect the afferent proprioceptive signals (e.g., from muscle spindles) associated with contraction/relaxation of agonist and antagonist muscles during both flexions and extensions associated with one movement cycle of various body parts (*e.g.*, fingers, toes) (Marty et al., 2019).

Of note, similar dominant proprioceptive contribution to CKC has been demonstrated between movement kinematics and activity of cerebellar lobule VIII during repetitive finger movements (Marty et al., 2018). Furthermore, CKC was also found during non-goal-directed (Bourguignon et al., 2013a) and goal-directed (Marty et al., 2015a) observed movements. These findings suggested that observing others' motor actions actually engages some of the viewer's brain areas, and particularly the SM1 cortex, in a similar kinematics-related manner as during own action execution. Such mirroring driven by action kinematics and—presumably—proprioceptive information might represent a prerequisite for human brain exploitation of visual kinematics of others' motor actions to understand *how* observed actions are actually performed (Marty et al., 2015a).

2.3. Perspectives

CKC might be useful to probe the integrity of spinocortical—and possibly spinocerebellar—proprioceptive pathways in humans and to gain novel information about brain disorders affecting those pathways (see, e.g., Marty et al., 2019). CKC is also an interesting and robust method for non-invasive functional sensorimotor mapping in neurosurgical patients (Bourguignon et al., 2013b, 2011). Of note, the ability to probe CKC with surface EMG (Piitulainen et al., 2013a) is of particular interest in the clinical context as EMG electrodes are widely available and at reduced cost, which should ease the dissemination of the method in clinical centres.

3. Corticomuscular coupling

3.1. Coupling frequency and cortical generators

CMC was first reported in 1995 (Conway et al., 1995). Studies investigating CMC have been reviewed previously (Mima and Hallett, 1999; Salenius and Hari, 2003). CMC is the coupling occurring between sensorimotor cortical rhythms and muscular activity as measured with surface EMG mainly during steady isometric muscle contraction (Conway et al., 1995; Kilner et al., 2000). Here "isometric contraction" refers to muscle contraction with stable muscle length and no change in joint angle.

CMC can be estimated with both unrectified and rectified EMG since both pick up the rhythmicity of muscle action potentials (see Fig. 2, ***right*** part). Whether it is preferable to rectify EMG signals to uncover CMC is still a matter of debate (Boonstra and Breakspear, 2012; Farina et al., 2013; Halliday and Farmer, 2010; McClelland et al., 2014, 2012; Myers et al., 2003; Negro et al., 2015; Neto and Christou, 2010; Ward et al., 2013; Yao et al., 2007).

CMC occurs mainly at ~20 Hz (range: 15–35 Hz) during weak contraction (see Fig. 2, *right* part) in about 60–80% of the individuals based on ~5-min long recordings (Conway et al., 1995; Mendez-Balbuena et al., 2012; Pohja et al., 2005; Salenius et al., 1997; van de Steeg et al., 2014), with a jump to ~40 Hz (range: 30–60 Hz) at maximum force (Brown et al., 1998; Mima et al., 1999; Salenius et al., 1996). It is characterized by rather weaker coupling (typically about 0.05–0.3 coherence level) compared with CKC (coherence level up to 0.8), and coupling level in a given individual may be influenced by motor learning (Mendez-Balbuena et al., 2012). Still, several studies have reported CMC at lower frequencies <15 Hz (Bourguignon et al., 2017; Marsden et al., 2001; Ohara et al., 2000; Raethjen et al., 2002; Salenius et al., 1997) (see Section 4.2. for further discussion) or in the low gamma-range (30–45 Hz) during selective movement preparation (see, *e.g.*, Schoffelen et al., 2011) or isotonic (*i.e.*, constant muscle tension but with muscle length changes) contractions (see, *e.g.*, Gwin and Ferris, 2012). For the purpose of conciseness, we will henceforth focus on the ~20 Hz CMC, and in the following, the term CMC implicitly refers to this specific coupling phenomenon (except if explicitly stated), without claims of generalization to CMC measured at other frequencies.

CMC originates mainly from M1 cortex contralateral to the contracted muscle and is somatotopically organized (Brown et al., 1998; Maezawa et al., 2014; Murayama et al., 2001; Salenius et al., 1997). Indeed, source reconstruction localizes CMC (i) with upper limb muscles to the hand area (Brown et al., 1998; Salenius et al., 1997), *i.e.*, at the hand knob (Yousry et al., 1997), (ii) with lower limb muscles to the paracentral lobule at the foot area (Brown et al., 1998; Hari and Salenius, 1999; Salenius et al., 1997), and (iii) with tongue muscles (during tongue protrusion) more laterally on the convexity (Maezawa et al., 2014). CMC magnitude also appears to scale with the size of the cortical representation of the muscles, as CMC to trunk muscles (paraspinal and abdominal) is weaker than CMC to hand (first dorsal interosseous) and foot muscles (tibialis anterior) (Murayama et al., 2001). Also, CMC is weaker for proximal than distal lower limb muscles (Ushiyama et al., 2010). This is in line with results that show monosynaptic cortico-motoneuronal inputs form weaker connections with proximal than distal muscle motoneurons (Farmer et al., 1993a, 1993b; Murayama et al., 2001).

Electrocorticographic recordings have confirmed that CMC is strongest at M1 cortex, although it is also present in other neocortical areas contralateral to the contracted muscles such as S1 cortex, the supplementary motor area, the cingulate gyrus, and the lateral premotor cortex (Ohara et al., 2000). According to non-human primate data, all these brain areas share the commonality of sending efferent axons to the spinal cord (Galea and Darian-Smith, 1994; Rizzolatti et al., 1998). Similar findings were obtained from cortical field potential recordings in monkeys, *i.e.*, CMC was dominant in the anterior bank of the precentral sulcus (Oya et al., 2019; Tsujimoto et al., 2009).

3.2. Neural basis of CMC

Initial reports concurred on the view that CMC is in essence driven by cortico-spinal efference, *i.e.*, that motor cortical oscillatory activity drives spinal motoneuronal pool (Brown et al., 1998; Gross et al., 2000; Murayama et al., 2001; Salenius et al., 1997). The efferent origin of CMC is supported by several findings. For example, ischemia-induced deafferentation dampens CMC but does not shift its peak frequency, which would be expected in case of strong contribution of afferent signals to CMC as a result of the decrease in sensory feedback and of the ischemia-induced prolongation of neural conduction times (Pohja and Salenius, 2003). Also, individuals in whom motor but not somatosensory functions have been relocated to the ipsilateral hemisphere due to pre- or perinatal damage to the pyramidal tract do show CMC in M1 but not S1 cortex (Gerloff et al., 2006; Marsden et al., 2001). Finally, in the framework of coherence analysis, it is possible to estimate the time delay between brain and muscle signals since it is proportional to the slope in the frequency-phase plots of the cross-spectrum (Halliday et al., 1995), the sign of the delay indicates which signal drives the other. Delays estimated that way implied that M1 cortex drives muscles and, according to some reports, were remarkably faithful to the conduction time from M1 cortex to EMG signals reported in transcranial magnetic stimulation studies, *i.e.*, from ~15 ms for the muscle *extensor indicis* to ~40 ms for muscle *flexor hallucis brevis* (Gross et al., 2000; Salenius et al., 1997).

Some authors have, however, objected to the view that CMC solely involves a cortical drive of the spinal motoneuron pools. Central to their claim was the observation that some individuals do not display the canonical phase–frequency relationship described above (Baker, 2007). Instead the phase remained essentially constant over the ~20-Hz frequencies, or implied delays shorter than known conduction delays (Riddle and Baker, 2005). It was argued that such phase–frequency behavior is easily explained if somatosensory afference plays a role in maintaining CMC (Baker, 2007). This was supported by computational modeling showing that two reciprocally coupled oscillators can phase-lock with zero phase-lag (Baker, 2007; Gerstner et al., 1996). Accordingly, methods based on the concept of Granger causality were used to disentangle the efferent and afferent contribution to CMC (Lim et al., 2014; Tsujimoto et al., 2009; Witham et al., 2011, 2010). Such directionality analyses demonstrated that both efferent and afferent signals contribute to CMC—although the efferent contribution was clearly dominant—with a similar delay of 25–30 ms in both directions for hand muscles (Witham et al., 2011). This finding suggested the importance of the closed sensorimotor loop in generating CMC—in line with a report of almost absent CMC in a deafferented subject (Kilner et al., 2004)—and provided an explanation for the previous inconsistencies in time-delays estimated from the phase of the cross-spectrum: there is inter-individual variability in the relative level of afferent and efferent contributions to the coupling (Riddle and Baker, 2005; Witham et al., 2011).

The finding that CMC receives a contribution from both efferent and afferent signaling has led to speculation on the functional role of CMC (Baker, 2007). Briefly, CMC was suggested to reflect the integration of afferent signaling into motor commands to promote a stable motor state (Androulidakis et al., 2007, 2006; Baker, 2007; Gilbertson et al., 2005), or the mechanism by which the sensorimotor system sends pulses at ~20 Hz and monitors the resulting afferent signal to probe the state of the periphery for continuous sensorimotor recalibration (Baker, 2007; Mackay, 1997; Witham et al., 2011).

Other findings however suggest that, although the cortex and periphery are coupled at ~20-Hz, such coupling might not be engaged in motor control *per se*. This alternative hypothesis comes from the fact that the main frequency of appearance of CMC (*i.e.*, ~20 Hz) during isometric contraction is closely linked to the ~20-Hz component of the sensorimotor mu rhythm that mainly reflects motor processes (for more details about the mu rhythm, see Fig. 3, Démas et al., 2019, and Hari and Puce, 2017). Specifically, the ~20-Hz mu rhythm would be involved in maintaining the current motor state (Engel and Fries, 2010) or in predictive coding (Tan et al., 2016). As for the ~20-Hz mu rhythm (Gastaut, 1952; Jasper and Penfield, 1949; Pfurtscheller and Neuper, 1997; Schnitzler et al., 1997), CMC is abolished during movements, and at its maximum right after movement stabilisation (Kilner et al., 2003, 2000). The modulation of ~20-Hz mu power and CMC also follow a similar trajectory in response to distracting auditory and visual stimulations (Hari et al., 2014; Piitulainen et al., 2015c).

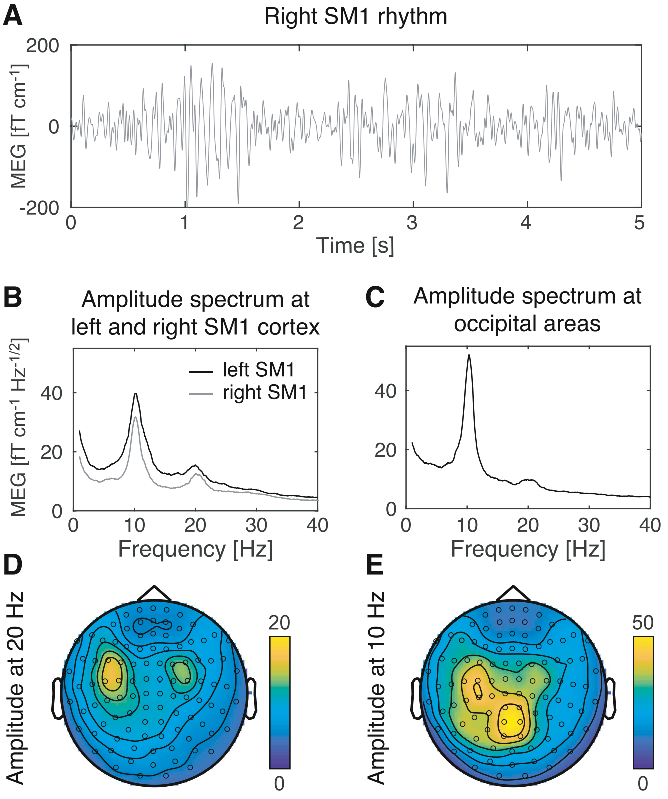

Fig. 3. Sensorimotor rhythm during isometric muscle contraction in a subject in whom it is prominent. Data are from a subject included in Bourguignon et al. (2017) who performed an isometric pinch contraction of 2–4 N with the right hand. **A** — Time course in the 5–45-Hz band of MEG signals above the left primary sensorimotor (SM1) cortex. Bursts of mu rhythm are evident, especially at 1–1.5 s. **B–C** — Amplitude spectrum of MEG signals measured above the left SM1 cortex (B; black trace), the right SM1 cortex (B; gray trace), and the occipital cortex (C). **D–E** — Spatial distribution of MEG amplitude at 20 Hz (D) and 10 Hz (E). Peaks of sensorimotor rhythm are clearly visible at 10 and 20 Hz, although 10-Hz amplitude is dominated by the occipital alpha rhythm. Also note that ~20 Hz power peaks at slightly more anterior sensors than ~10 Hz power.

Still, under some experimental conditions, CMC and mu rhythm follow different trajectories (see, *e.g.*, Hari et al., 2014; Stancak et al., 2005; Vigneswaran et al., 2013), which suggests that only a subset of the mu rhythm components underpin CMC (for a review, see, e.g., Kilavik et al., 2013). Furthermore, the mu rhythm typically appears in bursts separated by silent periods of ~1 s (Baker et al., 1997; Feingold et al., 2015; Gilbertson et al., 2005; Jasper and Penfield, 1949; Murthy and Fetz, 1996, 1992), and detailed analyses demonstrated that these bursts at ~20 Hz reach the periphery with a subject-dependent efficiency, giving rise to CMC (Bourguignon et al., 2017). Of note, high-density EMG only mildly increases CMC levels relative to standard EMG (Piitulainen et al., 2015a; van de Steeg et al., 2014), suggesting that inter-individual variability in CMC indeed relates to differences in a transmission mechanism.

But do individuals with nearly absent CMC (and ~20-Hz bursts in EMG) perform any poorer than their peers? Although within subjects, the magnitude of the CMC is higher for stable than unstable contractions (Kristeva-Feige et al., 2002; Kristeva et al., 2007; Witte et al., 2007), such a relation is not seen between subjects (Bourguignon et al., 2017). Moreover, the absence of CMC can be ascribed to the absence of ~20-Hz bursts in EMG or contraction force (i.e., the trace of CMC) (Bourguignon et al., 2017), rather than to an inability of MEG or EEG to record related brain signals due to technical or anatomical reasons. All this tends to favor the view that CMC might not be directly involved in motor control *per se*. CMC would rather reflect modulation of the motor command by the ∼20-Hz mu rhythm. That is, the ∼20-Hz mu rhythm causes rhythmic changes in M1 neuron excitability, leading these neurons to discharge in synchrony. Therefore, at the population level, the motor command tends to structure according to the mu rhythm, inducing similar oscillations in EMG or contraction force.

As far as we know, the view that CMC is not directly involved in motor control is compatible with the fact that its disruption or enhancement has little to no impact on contraction force (Hari et al., 2014; Piitulainen et al., 2015c; Tecchio et al., 2006).

3.3. Perspectives

CMC might not be directly involved in motor control *per se*. Still, in individuals in whom ∼it is not too low, studying this coupling (*i.e.*, level and modulation by experimental conditions) might provide precious information on motor cortical dynamics. Indeed, a disruption of CMC with a given muscle implies the absence of rhythmic bursts of ∼20-Hz mu rhythm within the ensemble of cortical motor neurons connected to this muscle, or in other words, that such ∼20-Hz activity remains desynchronized. Accordingly, CMC with a given muscle should inform on the state of the ensemble of cortical motor neurons that project to the motor pool of this muscle, rather than on corticospinal interaction. By "*state*", we here mean whether or not (or to which extent) the local ∼20-Hz mu rhythm undergoes rhythmic fluctuations in amplitude. Such information can hardly be obtained directly from power spectra, simply because—due to field spread—mu rhythms and activity from distinct but nearby neuronal populations cannot be separated based on their sensor topography. Considering this should help make sense of past and future research in which, *e.g.*, CMC is measured with several agonist and antagonist muscles with the endeavour to unravel cortical motor control of skilled motor actions in health and impairment (Cremoux et al., 2017; Dal Maso et al., 2017; Desmyttere et al., 2018). Note also that based on MEG or EEG recordings, CMC estimated with a given muscle is affected by the ∼20-Hz mu activity in surrounding regions, and that a measure free from such contamination is the burstiness of ∼20-Hz EMG/force fluctuations (Bourguignon et al., 2017).

4. Corticokinematic and corticomuscular couplings index two different neural processes

CMC and CKC have different neural bases. CKC is predominantly driven by the processing of proprioceptive feedback occurring during movements. CMC occuring at ∼20 Hz is a form of coupling with muscular activity that implicates the ∼20-Hz mu rhythm. It is maximal during weak isometric contraction and vanishes during movements. In some instances, however, the distinction between CMC and CKC may not be that clear. This is because (i) CKC and CMC can both be uncovered with EMG measures, (ii) CKC can occur during isometric muscle contraction, and (iii) CMC can occur during brief periods of isometric contractions within movements.

4.1. Corticokinematic coupling uncovered with EMG measures

As described in Section 2.1., CKC can be properly estimated using *rectified* electromyographic (EMG) signals (Piitulainen et al., 2013a). Based on this finding, previous studies (Pollok et al., 2005, 2004a, 2004b) that identified coupling at movement frequency (typically below <10 Hz) between brain activity and surface EMG signals during various upper limb movement tasks and that used the CMC terminology to refer to the coupling, actually identified CKC rather than CMC *per se*. Similar conclusions can be drawn for the coupling reported between ventral SM1 cortex (*i.e.*, mouth area) and orbicularis oris muscle activities during silent mouthing of a syllable (/pa/) periodically repeated at different frequencies (*i.e.*, 0.8–5 Hz) (Ruspantini et al., 2012). A more detailed discussion on these latter aspects is provided in Bourguignon et al. (2019).

Accordingly, the terminology used to refer to such "*cortex–kinematic*" interaction observed during movements should emphasize the nature of the coupling (*i.e.*, a coupling driven by movement rhythmicity) rather than the method (*e.g.*, coherence with finger acceleration or surface EMG) used to investigate it.

4.2. Corticokinematic coupling during isometric muscle contractions

CKC is not only seen during large-amplitude movements. Slow movements are typically accompanied by weak fluctuations in movement kinematics at 1–9 Hz (Gilbertson et al., 2005; Kakuda et al., 1999; Marshall and Walsh, 1956; McAuley et al., 1999, 1997; Vallbo et al., 1993). SM1 oscillations were found to be coherent with these kinematic fluctuations (Dipietro et al., 2011; Gross et al., 2002; Hall et al., 2014). Moreover, during isometric muscle contraction, SM1 oscillations are also coupled with the unavoidable fluctuations in the contraction force occuring at frequencies <3 Hz (see Fig. 4 A–C) that translate into tiny—sub-millimeter—movements (Bourguignon et al., 2017). The dominant afferent contribution to this coupling corroborates the idea that it should be seen as a form of CKC (Bourguignon et al., 2017). This finding suggested a simple mechanism to explain motor control of isometric muscle contractions, *i.e.*, the cortex sends a population-level motor command that is modulated by the ∼20-Hz sensorimotor rhythm, and it dynamically adapts these commands based on the <3-Hz fluctuations of proprioceptive feedback.

During isometric muscle contraction, the ∼20-Hz component of the mu rhythm is not only phase-coupled with EMG, but also with finger tremor at ∼20 Hz recorded with an accelerometer (Airaksinen et al., 2015) or with a force transducer (Bourguignon et al., 2017). This is likely because ∼20-Hz CMC entails rhythmic fluctuations in muscle activity, which in turn induce subtle force fluctuations or tremor at ∼20-Hz (see Fig. 4 D & E). Although the amplitude of this tremor is extremely low in healthy individuals, it is still high enough to activate proprioceptors such as muscle spindles or Golgi tendon organs (Bourguignon et al., 2017). We suggest that this ∼20-Hz tremor might be at the origin of the afferent contribution to CMC, and propose that such contribution should be linked to CKC rather than to CMC *per se*.

CMC is occasionally seen at ∼10 Hz with weaker coupling levels than at ∼20 Hz (Bourguignon et al., 2017; Marsden et al., 2001; Piitulainen et al., 2015a; Salenius et al., 1997), supposedly corresponding to the coupling with the ∼10-Hz component of the mu rhythm (for details about this mu rhythm component, see Démas et al., 2019, and Hari and Puce, 2017). The fact that this ∼10-Hz coupling has a small amplitude was also attributed to a possible specific blocking mechanism that would prevent the motor pool from synchronizing with descending inputs at ∼10 Hz (Baker et al., 2003). Such a blocking mechanism could be in place to prevent excess physiological tremor at ∼10 Hz (Baker et al., 2003; Raethjen et al., 2000), a type of non-clinical tremor present in all individuals (Gilbertson et al., 2005; Marshall and Walsh, 1956; McAuley et al., 1997). But whether such tremor has a cortical origin remains debated (see, *e.g.*, Raethjen et al., 2002 for positive evidence in epilepsy patients). Other more probable generators are spinal interneuronal systems (Allum et al., 1978; Elble and Koller, 1990) and subcortical oscillating structures (Elble, 1996). Regardless of its origin, such tremor should generate repetitive proprioceptive feedback that would result in CKC-like coupling with EMG. In line with that, ∼10-Hz movement discontinuities occurring during slow finger tracking movements produce strong sensory feedback that lead to an afferent-driven coupling with M1 activity (Williams et al., 2009). In sum, it is unclear to what extent the ∼10-Hz mu rhythm contributes to motor processes, and the origin of the ∼10-Hz physiological tremor is likely multifactorial (McAuley and Marsden, 2000). Accordingly, ∼10-Hz CMC could reflect efferent-driven coupling with the ∼10-Hz component of the mu rhythm (*i.e.*, a form of CMC), afferent-driven coupling with ∼10-Hz physiological tremor (*i.e.*, a

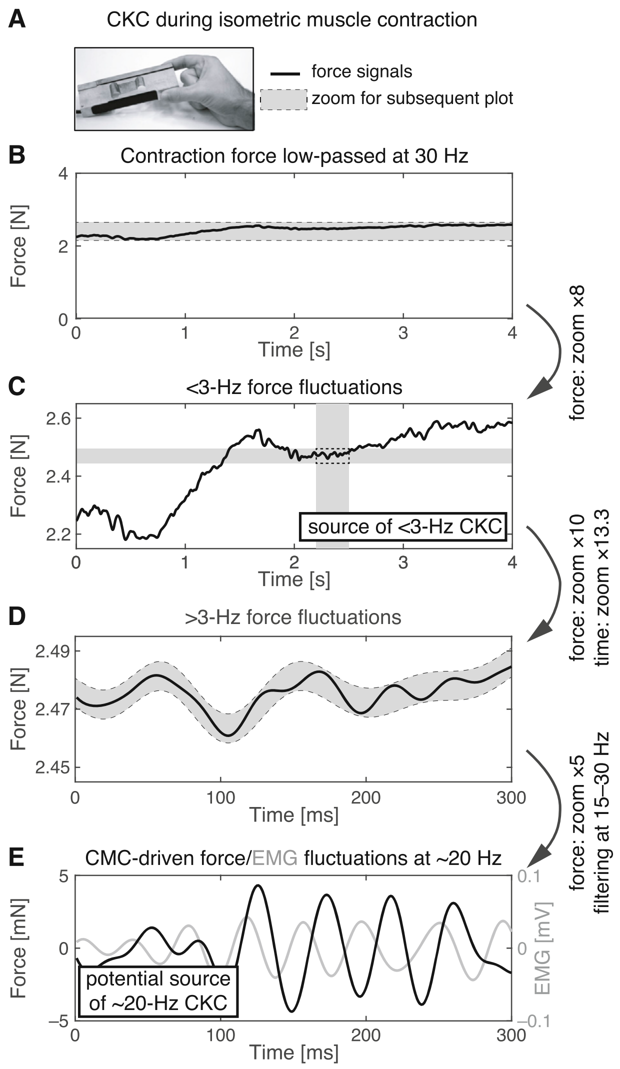

Fig. 4. Corticokinematic coupling (CKC) during isometric contraction. Data are from a subject included in Bourguignon et al. (2017), who performed an isometric pinch contraction of 2–4 N with the right hand. **A** — Isometric muscle contraction typically used to uncover cortex–muscle coupling (CMC). Through the figure, black traces show the force signal and gray frames indicate the area illustrated in the subsequent plot. **B** — Force signal low-pass filtered at 30 Hz in a 4-s time window indicating that the contraction force is relatively stable. **C** — Zoom on the force signal showing typical fluctuations dominant by frequencies <3 Hz. These fluctuations generate sensory feedback at the origin of a CKC-type of coupling. **D** — Zoom on force fluctuations at a shorter time-scale. **E** — Force signal and corresponding EMG signal (gray trace) filtered through 15–30 Hz. Fluctuations in EMG activity at ~20-Hz—which are mainly driven by the sensorimotor rhythm at ~20 Hz—induce subtle force fluctuations which could be a source of CKC.

form of CKC), or a combination of both. Further empirical studies are needed to clarify the involved mechanisms.

Finally, a form of CKC can be seen during isometric muscle contractions at the transition between two different force levels. In this situation, significant ~9-Hz and 33–39-Hz coupling has been reported, but only when there was an overshoot in contraction force (Mehrkanoon et al., 2014). Accordingly, the ~9-Hz coupling is most likely an evoked response or what we describe as CKC. Indeed, phase–frequency plots suggested it entailed an afferent-driven coupling with a scalp distribution more widespread than that at ~20 Hz during isometric muscle contraction, in line with reports of multiple cortical generators of CKC (Bourguignon et al., 2012).

4.3. *Corticomuscular coupling during movements*

Past research has clearly demonstrated that CMC is abolished during movements and maximal right after movement stabilisation (Kilner et al., 2003, 2000). This opened the possibility of measuring CMC during phasic contraction.

The recent development of ambulatory EEG has made it possible to investigate CMC and cortical oscillatory dynamics during walking (Artoni et al., 2017; Boonstra et al., 2009; Bradford et al., 2016; Gwin et al., 2011; Petersen et al., 2012; Roeder et al., 2018; Severens et al., 2012; Sipp et al., 2013) and bicycling (Storzer et al., 2017, 2016), leading to improved knowledge about cortical processes involved in walking or cycling locomotion. Overall, these studies have demonstrated that cortical power and CMC with low limb muscles increase during the double stance period of the gait cycle in a wide frequency range (4–45 Hz) (Artoni et al., 2017; Bradford et al., 2016; Gwin et al., 2011; Petersen et al., 2012; Roeder et al., 2018; Severens et al., 2012; Sipp et al., 2013). Again, delay estimation implied that the SM1 cortex drives the periphery at frequencies >8 Hz (Artoni et al., 2017; Roeder et al., 2018). Also, left and right mu power increases were shown to alternate along the gait cycle (Bradford et al., 2016; Gwin et al., 2011; Severens et al., 2012; Sipp et al., 2013). These results were taken as evidence that the cortex is involved in gait control in humans.

In the walking studies, heel strike is expected to generate significant tactile and proprioceptive responses phase locked with EMG activity. Accordingly, the strong coherent responses at frequencies <8 Hz should probably be considered to actually arise from CKC.

5. Limitations and perspectives

Most studies reviewed here rely on non-invasive human brain recordings such as MEG or scalp EEG that are characterized by a low spatial resolution that induces some confounding effects such as, *e.g.*, linear mixing of closely located neural sources. This is especially a problem for studies focusing on the sensorimotor system, given the proximity of M1 and S1 cortices. This issue indeed complicates the proper assessment of the respective sensory and motor contributions to CMC and CKC. It is also a specific problem for directionality analyses. Further studies relying on intracranial recordings should therefore be performed to bring more definite data supporting the respective functional roles of CMC and CKC emerging from this review, and to confirm some of the hypotheses developed in Section 4.

Pioneering works have started looking at the interplay between multiple muscle activity for postural control (Kerkman et al., 2018). Building on this, future research should strive to extent such work to integrate brain, muscle and kinematic signals recorded from multiple muscles/effectors in ecological motor actions such as locomotion or skilled hand (*e.g.*, writing, drawing, knitting) actions.

Finally, more studies bridging theoretical, modeling and empirical research are needed in order to achieve a holistic view of the underlying principles that govern brain-muscle and brain-movement interactions (see, *e.g.*, Todorov, 2000).

6. Conclusions

This review has highlighted that CKC and CMC are two clearly distinct forms of brain–body interactions. CKC is the coupling between activity of sensorimotor network nodes and various movement-related signals driven by movement rhythmicity. It predominantly reflects the cortical processing of proprioceptive feedback. It is especially salient during dynamic motor actions, but also detectable during subtle and unavoidable movements/tremors present during slow movements or steady isometric contractions. Empirical findings suggest that ~20 Hz CMC occuring during isometric contraction is linked to the modulation of the descending motor command by the ~20-Hz sensorimotor rhythm. Finally, this review emphasizes that the study of brain-body interactions during various motor actions should attempt to be more explicit about the nature of the underlying central–peripheral coupling they capture (*e.g.,* coupling driven by movement rhythmicity or by the mu rhythm) rather than focus on the method used to investigate these brain-body interactions.

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Appendix A. Supplementary data

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