Neurophysiological Bases of Human Movement 2025
King's College London, UK | 16 – 17 December 2025

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SA01

Modelling human movement neuromechanics for generalisable human-robot interfaces

Massimo Sartori[1]

[1]University of Twente, Netherlands

Movement is critical for human wellbeing. Therefore, developing robotic technologies that can preserve our ability of moving as we age or restore it following an injury is a key necessity. Wearable robots, such as exoskeletons and exosuits, are rapidly evolving from assistive tools into intelligent platforms that can influence human musculoskeltal health.

Recent advancements in wearable exoskeletons have demonstrated the ability to reduce metabolic cost and neuromechanical effort during locomotion. Emerging improvements in actuation, sensing, and form factor now enable lighter, softer systems—bringing us closer to devices that can be worn continuously and unobtrusively, much like a "second skin." As these systems become integrated into daily life across rehabilitation, industrial, and occupational domains, a critical paradigm shift is needed. Rather than only assisting movement in the short term, next-generation wearable robots must interface with the human body over extended periods to influence long-term musculoskeletal adaptation.

This raises fundamental new questions: How do neuromuscular tissues respond to robot-induced mechanical stimuli? Can we harness these responses to restore neuromuscular function for movement ? Answering these questions requires bridging knowledge gaps at the intersection of biomechanics, neuromechanics, and robotics.

This talk will outline ongoing work aimed at addressing these questions. Specifically, the talk will outline how we can use bioelectrical recording and numerical modelling to decode the activity of spinal motor neurons and concurrently derive the resulting force and stiffness-generating musculoskeletal function in the intact moving human *in vivo*. The second part of the talk will outline how the proposed approach can be extended to develop robotic technologies that could assist and potentially reshape the human neuromuscular system via targeted electro-mechanical stimuli deliverered at extreme ends of the spatio-temporal scale, *e.g.*, cell-to-organ growth over weeks.

Over the next decade, this framework may transform wearable robots into proactive, adaptive tools for preventing chronic musculoskeletal conditions, promoting recovery, and maintaining physical independence—ultimately improving healthspan and quality of life.

SA02

Early development of stepping and locomotor control

Yury Ivanenko[1]

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This presentation will discuss recent advances in our understanding of how motor commands are expressed across early stages of human development. In neonates, alternating spinal motor output reflects a simpler organization of neuronal locomotor networks. Basic motor modules are present at birth but are progressively reconfigured through continuous interactions among the brain, body, and environment. The complexity of motor modularity increases from the neonatal period to adulthood at multiple hierarchical levels of the motor system, from the intrinsic rhythmicity of individual muscle activity to the structure of muscle synergies and bilateral intermuscular connectivity. Particular attention will be given to mechanisms underlying impaired locomotor development, with a focus on spinal cord function. The first two years of life represent a critical window for the maturation of locomotor circuits. The emergence of independent walking during this period offers a unique opportunity to assess how neural circuitry develops in children with cerebral palsy. Recent findings indicate that achieving independent walking requires differentiation of proximal and distal extensor activity and an increase in the number of neuromuscular modules under corticospinal influence. While low complexity and high variability of neuromuscular signals reflect neonatal immaturity, they may also provide an adaptive substrate for learning locomotor skills.

SA03

Combining imaging and deep brain recordings in humans to understand the role of the brainstem in Parkinson's disease

Ben Clennell[1]

[1]King's College London UK

In Parkinson's disease (PD), degeneration in subcortical structures and changes in neuronal activity in the subthalamic nucleus (STN) are implicated in motor symptoms. The development of new imaging techniques provides the opportunity to explore the role of both for the first time. Particularly, deep drain stimulation (DBS) sensing technology allows for the measurement of local field potentials in the STN whilst the person with PD performs activities such as walking. In addition, measures of free water and magnetic susceptibility allow for the identification and assessment of neurodegeneration in small subcortical structures.

We are currently investigating how degeneration in subcortical structures such as the substantia nigra (SN) and the pedunculopontine nucleus (PPN) is associated with Parkinson's-related pathological brain activity measured through LFP in the STN. We have undertaken multimodal MRI pre DBS surgery to extract structural metrics that index degeneration in subcortical structures. Post-surgery, we are recording on and off medication walking, resting and standing (with motion tracking) with mobile EEG and LFP.

Importantly, this work will help us understand how structures outside the basal ganglia, like the PPN, are influencing Parkinson's symptoms, and how they could be targeted in new treatment strategies.

SA04

The borderlands of motor control and behaviour: movement disorders in neuropsychiatric conditions

Mark Edwards[1]

[1]KCL, UK

In this talk I will discuss disorders of movement control that occur in neuropsychiatric conditions. In some of these conditions, for example schizophrenia, disorders of movement are common, but are often overshadowed by the psychiatric symptoms. Other conditions, for example Tourette's syndrome and particular functional neurological disorder, challenge our often quite rigid conceptions of voluntary and involuntary movement. I will present a general overview of these common clinical conditions and consider their pathophysiology and clinical management.

SA05

The common basis of movement decisions and movement adjustments.

Jeroen Smeets[1], Eli Brenner[1]

[1]Vrije Universiteit Amsterdam, The Netherlands

It is textbook knowledge that motor decisions take an amount of time that is proportional to the logarithm of the number of possibilities. We live in a world with an unlimited number of possibilities, so deciding what to do would take an enormous amount of time. In contrast to decisions, adjusting one's movements can start in about 100 ms after the information becomes available. What distinguishes adjustments from decisions? I will present evidence that they don't differ at all. For this, we have to rethink the notion of the motor decision. A movement is not the result of a single decision on where to go, but the decision-making continues during the movement. Adjustments are the consequence of this continuous decision process in the case of a target jump.

SA06

The vestibular system: A fundamental reference for human behaviour

Elisa Raffaella Ferre[1]

[1]Birkbeck University of London, UK

The vestibular system arises from a complex set of sensory transducer organs in the inner ear. It comprises three orthogonal semicircular canals that detect rotational acceleration of the head in three-dimensional space, alongside two otolith organs - the utricle and saccule - that sense translational acceleration and encode head orientation relative to gravity.

Gravity is a constant and fundamental force that shapes the development, physiology and behaviour of all organisms on Earth. Vestibular organs are finely tuned to detect the gravitational vector, providing the brain with essential information to maintain balance, coordinate movement and control posture. When gravity is altered - such as during weightlessness or under the partial gravity of the Moon or Mars - this precisely calibrated system is disrupted, highlighting the extent to which vestibular processing is adapted to terrestrial conditions. Understanding these responses is critical for defining the limits of neural plasticity and behavioural adaptation in novel environmental contexts.

In this talk, I will present our research integrating psychophysics, neuroimaging, computational modelling, and space science studies to investigate how vestibular signals are represented in the human brain and how these representations shape behaviour. I will examine their contributions not only to reflexive motor control and postural stability but also to perception, multisensory integration and higher-level cognition. Finally, I will discuss the implications of these findings for neural plasticity and human adaptation beyond Earth, offering insight into the challenges of living in space.

SA07

How action prediction shapes perception

Clare Press[1]

[1]University College London, London

It has long been appreciated that learning about the probabilistic structure of our sensorimotor environment shapes perception in important ways. However, it is less clear how. For example, we must balance demands of representing the perceptual world accurately while effectively using perception to update our models when the world changes.

Bayesian accounts propose that predictions are integrated with sensory inputs to determine what we perceive. Because the prediction has been a source of information that feeds into the percept, we are more likely to perceive what we expect and we perceive it more intensely. Accounts propose that neurally this is achieved by amplifying the sensory gain of expected channels. This mechanism would render our percepts more accurate in a noisy sensory world, because what we predicted to be there is more likely to be there. We can also generate these best guesses more rapidly than if relying solely on sensory input, given the neural transmission delays associated with its processing. In contrast, attenuation accounts propose the opposite. They claim that we reduce the sensory gain of expected channels, which reduces perception of the expected – rendering us less likely to perceive it and perceiving it less intensely. These accounts highlight a mechanism that would render our percepts more informative in a limited capacity processing system – directing resources to events that tell us more and thereby likely require model-updating. These accounts both seem sensible and are supported by a host of data but are mutually incompatible.

I will ask how we overcome the challenge of optimising sensorimotor perception such that it is accurate, rapid and informative, with accounts that can serve all functions rather than optimising for one at the direct cost to another. I will present work that tests these possibilities by asking what we perceive alongside neural processing across time (EEG) and space (7T fMRI). I hope to convince the audience that our models of action-perception interdependences should move on from some currently-popular monolithic accounts and stimulate discussion concerning how best to conceptualise these synergistic relationships.

SA08

Cortical activity signatures of perceptual-cognitive-motor interactions in health and disease

Lena Ting[1]

[1]Emory University US

Engagement of cortical resources in balance control is an indicator of fall risk in older adults where people cannot "walk and talk" at the same time. However, there are few direct measures of cortical activity during balance control, and their relationship to balance and other brain functions is unclear. I will show that various electroencephalography (EEG) measures of cortical activity during reactive balance stratify healthy young and older individuals without clinically identifiable impairment across perceptual, cognitive and motor function, identifying possible mechanisms of individual differences in motor ability. Specifically, beta oscillations prior to balance perturbations are strongly associated with perceptual function and associated with motor capacity. Further, evoked cortical potentials due to balance perturbations localized to supplementary motor area are highly stable electrophysiological signatures across individuals that are associated with cognitive-motor interactions necessary for mobility. Further, these relationships are altered with training, aging, and neurological disorders such as stroke and Parkinson's disease. The intersections across perceptual, cognitive, and motor domains may help identify complex mechanisms underlying balance function and enable development of mechanistic, precision-medicine efforts aimed at fall prevention.

SA09

Neurophysiology based closed-loop deep brain stimulation for movement disorders

Huiling Tan[1]

[1]University of Oxford, United Kingdom

Deep brain stimulation (DBS) is an established therapy for advanced Parkinson's disease and holds promise for a range of other neuropsychiatric disorders. Conventional DBS (cDBS) delivers continuous electrical stimulation with fixed parameters, disrupting neural circuits regardless of fluctuations in patients' symptoms or brain states. This lack of specificity can result in side effects and diminished therapeutic efficacy over time. Recent advances highlight the potential of adaptive DBS (aDBS), which dynamically adjusts stimulation parameters in real time based on peripheral and/or neural feedback signals, providing more precise and state-dependent modulation of brain activity.

In this talk, I will present several projects demonstrating that aDBS guided by neural recordings can achieve equal or superior improvements in motor control compared to continuous high-frequency stimulation. Specifically, aDBS enhances hand-reaching performance by precisely modulating neural dynamics—suppressing pathological brain activity while preserving neural patterns essential for normal function. In contrast, cDBS indiscriminately suppresses subthalamic nucleus (STN) activity across frequency bands, even in the absence of pathological oscillations. I will also discuss tailored aDBS protocols designed to restore physiological neural functions by selectively suppressing pathological oscillations while maintaining healthy rhythms during walking and sleep. Such approaches show promise in improving gait and sleep disturbances—symptoms that remain inadequately addressed by current therapies, including dopaminergic medications and standard DBS.

SA10

Sensing the self: Psychophysics and neuroimaging of somatosensory processing in voluntary movements

Konstantina Kilteni[1, 2] [1]Karolinska Institutet Sweden [2]Donders Institute for Brain, Cognition, and Behaviour The Netherlands

Self-touch is perceived as weaker and rarely tickles compared to external touch. Using psychophysical and neuroimaging approaches, I will present evidence supporting an efference copy–based mechanism that predicts the sensory consequences of voluntary movements to attenuate self-generated tactile sensations and engages interactions between the cerebellum and somatosensory cortex.

C01

Investigating the role of intrinsic plasticity in the human motor cortex during motor skill learning

Benjamin Clennell[1], Binyu Luo[1], Sophie Lam[1], Yu Hu[1], Angel V Peterchev[2], Ricci Hannah[1]

[1]King's College London, United Kingdom, [2]Duke University, USA

Background

The brain's ability to change in response to experience, known as plasticity, is crucial for learning, memory, and recovery from injury. Most research focuses on synaptic plasticity: changes at the connections between neurons. However, the brain also adapts through intrinsic plasticity, in which changes occur to the excitability of axons thereby regulating action potential firing[1,2]. Intrinsic plasticity is poorly understood in humans due to a lack of specific, non-invasive measurement tools. Controllable pulse transcranial magnetic stimulation (cTMS) allows for the systematic variation of pulse shape to directly measure changes in axonal excitability[3] thereby dissociating synaptic and intrinsic effects. We sought to determine if motor skill learning in humans is accompanied by changes to the strength-duration time constant (SDTC), a biophysical property reflecting voltage-gated sodium channel activity in the axonal membrane[4], supporting a role for intrinsic plasticity in the human motor cortex.

Methods

11 healthy adults completed a ballistic thumb abduction task known to drive plasticity[5]. Task performance kinematics were recorded by accelerometery. Motor-evoked potentials (MEPs) were recorded by electromyography (EMG) in response to cTMS stimulation across a range of pulse widths (30, 60, 90, 120 μs) and intensities (90–120%RMT) to derive input-output curves and SDTC before and after practice (5-min, 30-min, 60-min) from the trained APB and a control muscle (ADM). Procedures adhered to the Declaration of Helsinki and were approved by the King's College London Research Ethics Committee (HR-24/25-39903). All participants were free from neurological or psychiatric disorders and not taking neuroactive medications.

Results

Our **interim results** (n=11) confirm that motor practice leads to performance gains measured as increased acceleration during thumb abduction (main effect of time: $F(4,40)=16.4$, $p<0.0001$, $\eta^2=0.62$; baseline vs 1-hr retention: 14.2±2.8 vs 35.5±5.5 m/s^2; $t10=5.3$, $p$holm=0.002). We find MEP amplitudes for the APB are increased following motor practice with a clear trend towards a sustained elevation up to 60 min (5-min: $t10=2.4$, $p$holm=0.07, $d=0.73$; 30-min: $t10=2.7$, $p$holm=0.06, $d=0.83$; 60-min: $t10=1.8$, $p$holm=0.10, $d=0.53$). Crucially, we observe a pattern of transient increase in SDTC for the APB (5-min: 9±2%; 30-min: 7±2%) that appears to return to baseline levels over the same period (60-min: -3±1%) and is not apparent in the ADM (5-min: -2±2%). Whilst these trends do not reach statistical significance (muscle x time interaction effect: $F(4,40)=1.47$, $p=0.22$) a power analysis indicates that n=26 will be sufficient to detect a significant muscle x time interaction ($\alpha=0.05$, $1-\beta=0.8$) which we will target with ongoing data collection.

Conclusions

Whilst preliminary, these findings hint towards a dissociation between synaptic and intrinsic effects: MEPs increase after training and remain elevated, whereas SDTC rises immediately but returns to baseline within 60 min. This pattern raises the possibility for interactions between the two mechanisms, where transient intrinsic changes facilitate (metaplasticity) or maintain balance (homeostasis) after synaptic facilitation; interactions not previously demonstrated in humans. Irrespective of the outcome, these findings will establish a role for intrinsic plasticity in the human motor cortex during motor skill

learning, with clinical relevance to the development of neurorehabilitation strategies for patients recovering from stroke, brain injury, or other neurological conditions.

C02

Does a global suppression mechanism for suppressing voluntary movements extend to involuntary movements?

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Introduction: Humans are able to voluntarily suppress unnecessary or unwanted actions. The neural mechanism of such suppression is thought to rely on a prefrontal–basal ganglia network. A key neurophysiological marker of this network is a global motor system suppression, characterized by a widespread, transient downregulation of corticospinal excitability during reactive stopping of voluntary actions. However, it remains unclear whether this mechanism extends to the voluntary suppression of involuntary movements.

Aims: The present study aimed to determine whether global suppression is recruited when individuals suppress a sustained involuntary movement and to further characterize the temporal dynamics of this process.

Methods: Healthy adults were recruited in two experiments: Experiment 1 (N=20) and Experiment 2 (N=18). The Kohnstamm phenomenon, defined as involuntary arm elevation following sustained deltoid contraction, was used as a model of involuntary movement. In a within-subject design, participants either allowed the involuntary drift to occur naturally (Allow condition) or voluntarily suppressed it following a stopping cue (Suppress condition). To probe global suppression, motor-evoked potentials (MEPs) were recorded from a contralateral, task-unrelated muscle (first dorsal interosseous, FDI) using single-pulse transcranial magnetic stimulation (TMS). Two stimulation timings were employed: early TMS, delivered shortly after the arm crossed a threshold angle (ACT) (500 ms in Experiment 1; 150 ms in Experiment 2, the latter consistent with the latency of global suppression in reactive-stopping paradigms)[1,2], and late TMS, delivered 4.5 s after ACT. Arm kinematics were continuously tracked and extracted at ACT, early TMS, and late TMS. Electromyography (EMG) was recorded from the deltoid and latissimus dorsi; root mean square (RMS) amplitudes were calculated and compared across the induction phase and three critical epochs (500 ms before ACT, ACT–early TMS, 500 ms before late TMS). ANOVA was conducted to analyse condition by time interactions for arm position, EMG and MEP amplitudes.

Results: Behavioural and EMG analyses confirmed that participants successfully suppressed involuntary arm elevation (Condition × Time interaction: Exp.1, $F(2,38)=31.87$, $p<.001$; Exp.2, $F(2,34)=38.80$, $p<.001$), primarily through control of the agonist deltoid – preventing the rise in agonist activity (Condition × Time interaction: Exp.1, $F(2,38)=4.03$, $p=.028$; Exp.2, $F(2,34)=25.10$, $p<.001$), Condition × Time interaction: Exp.1, $F(2,38)=1.67$, $p=.202$; Exp.2, $F(2,34)=3.87$, $p=.058$). However, MEP analyses revealed no evidence of global suppression: at no timepoint were MEP amplitudes lower in Suppress than in Allow. A significant main effect of Condition was observed in both experiments (Exp.1: $F(1,19)=13.73$, $p=.002$; Exp.2: $F(1,17)=21.21$, $p<.001$), whereas the Condition × Time interaction reached significance only in Exp.1 ($F(1,19)=7.30$, $p=.014$). At early timepoints, no significant differences appeared (Exp.1: $t(19)=-1.47$, $p=.158$; Exp.2: $t(17)=-1.94$, $p=.068$). Indeed, at the late timepoint, MEPs in Suppress were consistently greater than in Allow (Exp.1: $t(19)=-4.04$, $p<.001$; Exp.2: $t(17)=-2.52$, $p=.022$), indicating that sustained suppression was not accompanied by widespread cortical downregulation.

Conclusions: Across two independent experiments, we consistently found that voluntary behavioural suppression of sustained involuntary movement does not depend on neural global suppression

mechanism. Instead, behavioural suppression may be achieved through selective neural suppression of the primary agonist muscle representation.

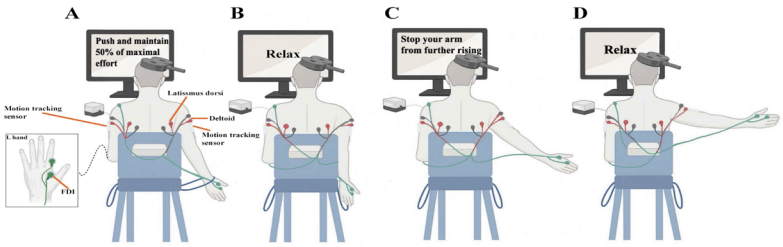
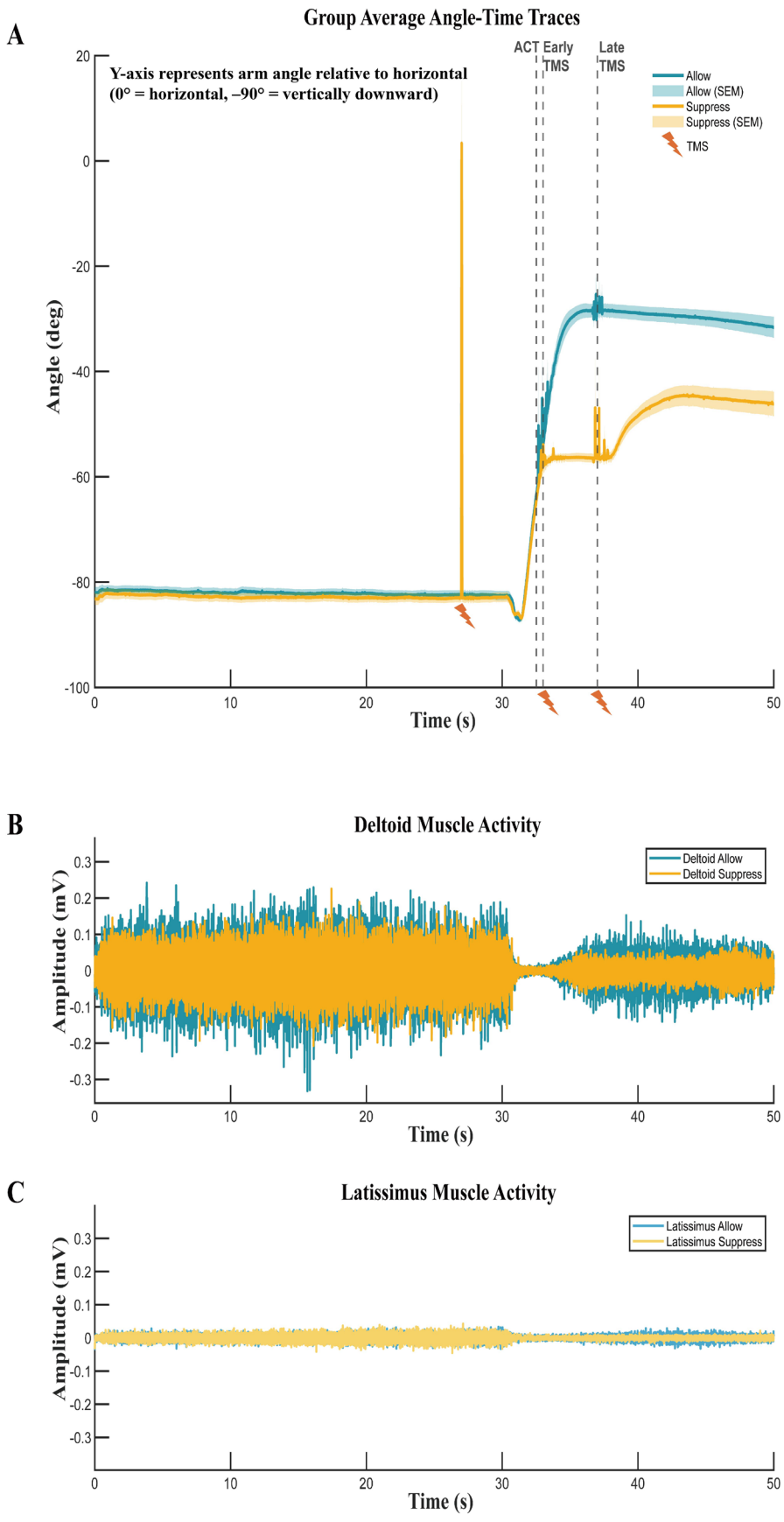


Figure 1. Experimental setup and task procedure (example from the right-arm Suppress condition). This figure illustrates a representative trial from the Suppress condition using the right arm.
(1A) Electromyography (EMG) was recorded from the deltoid (agonist) and latissimus dorsi (antagonist) muscles throughout each. Motion tracking sensors were placed on the right arm to record kinematic data from the arm. Trials began with an induction contraction, in which participants abducted their right arm against a rigid strap positioned just above the wrist, maintaining a strong voluntary contraction (~50% of their maximal EMG) for 30 seconds.
(1B) A visual cue instructed participants to relax. After 1–2 seconds, the right arm involuntarily rises due to the Kohnstamm phenomenon.
(1C) When the arm crossed a threshold (ACT) angle (25 degrees from vertically downward), a cue appeared instructing participants to stop further elevation (Suppress condition). The early TMS pulse was delivered 500 ms after the ACT was reached in Experiment 1, and 150 ms after the ACT in Experiment 2.
(1D) At 4.5 seconds after the suppression cue, participants were instructed to relax, which often resulted in the arm resuming the elevation. The late TMS pulse was delivered at this 4.5-second time point. EMG and motion data were continuously recorded throughout the trial. The inset shows the location of the left first dorsal interosseous (FDI) muscle, from which MEPs were recorded to assess corticospinal excitability from the stimulated (right hemisphere) motor cortex.


A
Group Average Angle-Time Traces
Y-axis represents arm angle relative to horizontal
(0° = horizontal, –90° = vertically downward)
ACT Early TMS
Late TMS
Allow
Allow (SEM)
Suppress
Suppress (SEM)
TMS
Angle (deg)
Time (s)
B
Deltoid Muscle Activity
Deltoid Allow
Deltoid Suppress
Amplitude (mV)
Time (s)
C
Latissimus Muscle Activity
Latissimus Allow
Latissimus Suppress
Amplitude (mV)
Time (s)

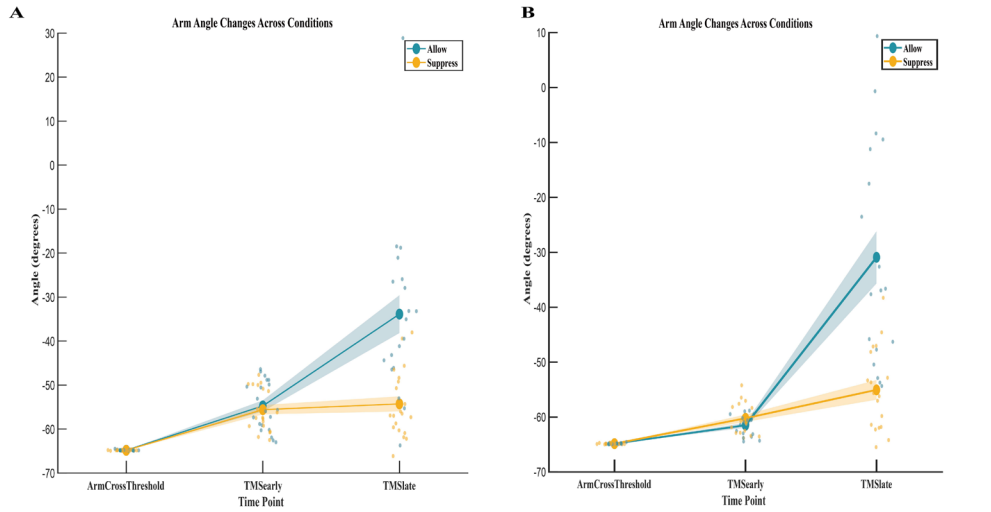


Figure 3. Time course of arm angle across experimental conditions. Figure 3A is for Experiment 1 and Figure 3B is for Experimen 2.
Mean shoulder angle traces are shown for the Allow (blue) and Suppress (orange) conditions at three key time points: ACT (when the arm crossed a threshold), early TMS (500 ms after ACT in Experiment 1 and 150ms after ACT in experiment 2), and late TMS (4.5 s after ACT). Individual data points from all participants are overlaid. In the Allow condition, the arm continued to rise following the ACT, whereas in the Suppress condition, arm elevation rapidly plateaued, confirming successful modulation of involuntary movement in response to the stop cue.

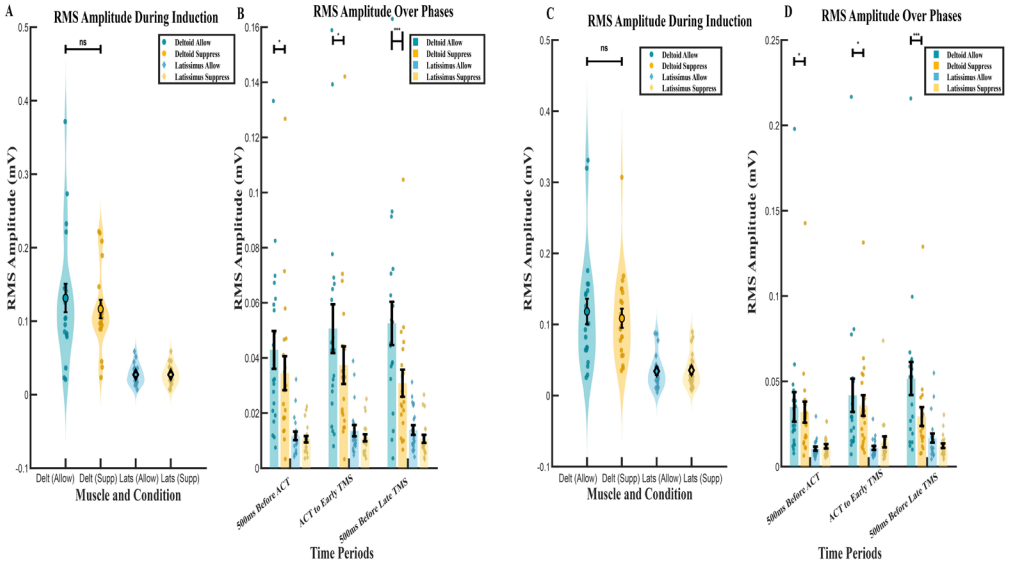


Figure 4. RMS amplitudes of deltoid and latissimus dorsi muscles during the induction and over phases. Figures 4A and 4B are for Experiment 1 and Figures 4C and 4D are for Experiment 2.
Figures 4A and 4C:Mean RMS amplitudes during the induction phase for deltoid (agonist) and latissimus (antagonist) muscles under Allow and Suppress conditions. No significant differences were observed between conditions for either muscle.
Figures 4Band 4D: RMS amplitudes across three time periods during the suppression phase: 500 ms before ACT, ACT to early TMS, and 500 ms before late TMS. A significant Condition × Muscle × Time interaction was observed. Deltoid RMS amplitudes were significantly higher in the Allow condition than in the Suppress condition at all three-time windows, with the difference increasing over time as EMG continued to rise in Allow but remained relatively stable in the Suppress condition. No significant differences were found in latissimus activity. Bars represent means ± SEM; individual data points are shown. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns, not significant.

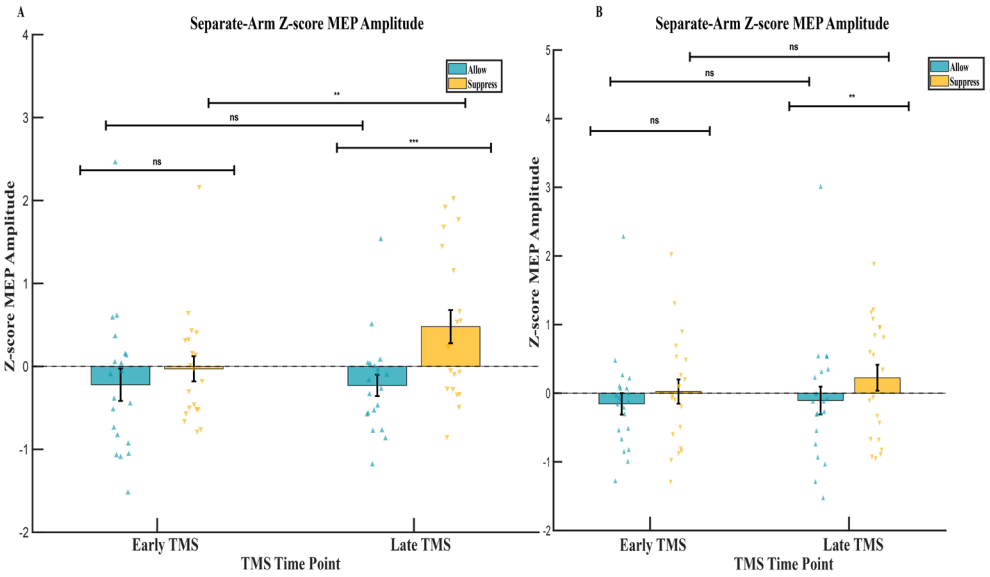


Fig. 5. Corticospinal excitability indexed by z-scored MEP amplitude across conditions and time points. Figure 5A is for Experiment 1 and Figure 5B is for Experiment 2.
Group-level z-scores of MEP amplitudes (normalized separately within each arm) are plotted for each experimentalcondition and TMS time point. A 2×2 repeated-measures ANOVA revealed a significant main effect of Condition (Suppress vs Allow), Time (Early vs Late TMS), and a significant Condition × Time interaction . Pairwise comparisons indicated no difference between conditions at Early TMS, but significantly higher MEP amplitudes in the Suppress condition at Late TMS. MEP amplitudes increased from Early to Late only in the Suppress condition. Individual subject values are shown as triangles, with bars indicating group means ± SEM. * p < 0.05; ** p < 0.01; *** p < 0.001; ns, not significant.

C03

Does passive blood flow restriction affect central neuromodulation and motoneuron excitability?

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Introduction and Aim: Blood flow restriction (BFR) is a method that is used in training and rehabilitation settings to improve functional outcomes. Although widely and successfully applied, the underlying neuromuscular mechanisms are not yet fully understood. By tracking the same motor units (MU) active before and after BFR (trapezoid ramp contractions), we showed an increased MU discharge rate after BFR. Persistent inward currents (PICs) are known to impact synaptic inputs to motoneurons by providing additional excitatory current. Thus, this study aimed at investigating the acute passive BFR effects on the central neuromodulation of PICs (Gorassini et al. 2002).

Methods: Thirteen uninjured male handball players (age: 24 [SD 3] years, body mass index: 24 [2] kg/m^2) participated in the study after providing written informed consent (IRB approval number: 2024-167). High-Density surface EMG (HDsEMG) grids (HD08MM1305) were placed on participants' dominant lower limbs over the vastus medialis (VM) and lateralis (VL) muscles. Signals were recorded before and after a 5-minute passive BFR intervention at 80% of maximal arterial occlusion pressure (AOP: 157 [14] mmHg). Maximal AOP was defined using a mobile Doppler ultrasound in the popliteal fossa. Isometric force-matching triangular contractions (duration 20 s) at 20% maximal voluntary contraction (4.06 [0.84] Nm/kg) were performed before and immediately after BFR. HDsEMG signals recorded before BFR were decomposed (Negro et al. 2016) into firing instances of MU action potentials. To compare the properties of the same MU, the MU identified before BFR were tracked after BFR using their MU filters (Francic & Holobar 2021). Analyses were performed in Python with the openhdemg V0.1.1 library (Valli et al. 2024) by applying the paired MU analysis technique (Gorassini et al. 2002). Thereby, low recruitment threshold MU (control units) were paired with higher recruitment threshold MU (test units). Delta frequency (F) values of each test-control unit pair were estimated and served as the outcome. Effects were assessed utilizing the linear mixed effects model.

Results: In total, 97 (96%) out of 101 identified MUs were successfully tracked after BFR for VL and 56 (71%) out of 79 for VM. The linear mixed effects model revealed a significant main effect of Time ($p < 0.001$) on the estimated Delta F values. Specifically, the Delta F values increased by 20.3% after BFR (from 2.63 [0.24] to 3.17 [0.24] pps). No relevant effects for the main effect of Muscle ($p = 0.65$) or the Time*Muscle interaction ($p = 0.49$) were observed.

Conclusions: This study analyzed the effects of a passive BFR intervention on central neuromodulation indirectly by estimating the PICs of thigh muscles. The absence of an effect of Muscle underscores the underlying central mechanisms caused by local ischaemic interventions at the extremities (McNulty et al. 2002). Since individuals with knee joint injuries suffer from altered motor system excitability and reduced volitional muscle activation BFR, particularly the passive mode, may be used to counteract these neuromuscular alterations by enhancing excitatory plasticity.

C04

Entrainment of motor unit firing by deep brain stimulation for Parkinson's disease

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[1]University College Dublin, Ireland, [2]Mater Misericordiae University Hospital, Ireland

Motivation: Deep brain stimulation (DBS) is a well-established therapy for reducing the motor symptoms of Parkison's disease. The mechanisms by which it affects motor function and more specifically motor unit activity and recruitment patterns, however, are not well understood. It has been established that DBS antidromically stimulates pyramidal tract neurons projecting from the cortex to the subthalamic nucleus (STN) (Li *et al.*, 2012). Motor evoked potentials may also be observed during low frequency, high amplitude stimulation. The effect of DBS at the motor unit level at clinically relevant frequencies and amplitudes, however, has not yet been investigated. To investigate this, high-density EMG was used to examine the effect of DBS on motor unit firing patterns during isometric contraction of the first dorsal interosseous (FDI) muscle.

Methods: Following institutional ethical approval, 17 (4 females, aged 60.8 ± 7.4 years, Time since Diagnosis: 11.3 ± 3.7 years) participants volunteered for the study. Isometric index finger abduction was performed at 10%-30% of maximum voluntary contraction (MVC) in 4 conditions: DBS ON and OFF, with and without medication. High-density EMG was recorded from the FDI using a 126-electrode array and the data were decomposed into the constituent motor unit firing trains. Power spectra of the EMG signals, individual motor unit spike trains and cumulative spike trains were computed using Welsh's method. Synchronization was estimated from the cross-correlogram between pairs of motor unit discharges, with short- and long-term synchrony defined as an increased probability of motor units firing within 10ms or greater than 10 ms of each other, respectively.

Results: DBS led to entrainment of a subset of motor unit firing times at the stimulation. This was observed as a significant increase in power in the EMG power spectra at the DBS frequency and higher harmonics during DBS ON ($p < 0.001$). A significant increase was also observed in the power spectra of the cumulative spike trains of the decomposed EMG data during DBS ON ($p < 0.001$). Eight participants exhibited a peak in the power spectra of both EMG and cumulative spike trains. In four participants, peaks in the power spectrum of the spike trains of individual motor units were observed, with secondary peaks consistent with pulse frequency modulation of the motor unit mean firing rate at the DBS frequency. In these participants, motor unit synchronization of units was increased ($p = 0.038$) during DBS ON with cross-correlogram peaks at multiples of the DBS interpulse interval.

Conclusion: The presence of peaks in the EMG and motor unit power spectra, and increased motor unit synchronization, are consistent with entrainment of motor unit firing during high frequency DBS (79-130 Hz). The results provide evidence of orthodromic activation of pyramidal tract neurons during STNS DBS at clinically relevant amplitudes and frequencies. In addition to providing insight into changes in motor unit firing during DBS, the findings suggest potential new biomarkers for optimization of DBS parameters based on motor unit activity.

C05

Exploring the role of vestibular input in muscle reflex function

Rhys Manning Garrero[1], Matteo Ciocca[1], Bradley Lonergan[1], Toby Ellmers[1]

[1]Imperial College London, United Kingdom

Introduction

Balance control is a complex, multisensory process that depends on the integration of visual, proprioceptive, and vestibular inputs to generate appropriate reflexive responses and adaptive motor behaviours that maintain postural stability. Balance impairments are common in older adults and various clinical populations, leading to increased risk for falls. Emerging research has highlighted the potential clinical utility of noisy subthreshold vestibular stimulation (termed, 'noisy galvanic vestibular stimulation') applied to the mastoids to reduce imbalance. However, our understanding of the mechanisms underpinning these effects remains poor. This study tested the hypothesis that noisy galvanic vestibular stimulation enhances balance by modulating spinal reflexes.

Methods

19 healthy young adults took part in our study. Participants received noisy galvanic vestibular stimulation (± 0.35 mA) during standing whilst H-reflexes were measured. H-reflex was elicited through stimulation in the popliteal fossa and the EMG was measured across the right soleus. Standing H-reflex amplitude was measured during standing balance (eyes closed), whilst receiving either active or sham vestibular stimulation. The protocol was conducted on both a foam and a solid surface to compare across different levels of vestibular dependence: standing on foam with eyes closed results in the CNS relying predominately on vestibular input to regular balance. Average normalised H-reflex amplitudes were calculated for each condition. All participants provided informed consent and the study was approved by the local ethics committee.

Results

In total we analysed 1520 H-reflexes (n=80 per participant). There was no significant change in normalised H-reflex amplitude between sham and active stimulation on the solid surface ($t(18)=-0.481$, $p=0.636$). However, on the foam surface we found a significant increase in H-reflex amplitude during active stimulation ($t(18)=2.149$, $p=0.046$).

Discussion

Our results indicate that noisy galvanic vestibular stimulation modulates spinal reflexes during challenging, vestibular-dominant balance conditions (standing on a foam surface). Our findings support spinal reflex modulation as a possible mechanism for the improvements in balance seen with noisy galvanic vestibular stimulation. The contrasting results on the foam and solid surfaces are a likely result of sensory reweighting, whereby the body up-regulates vestibular contributions to balance and down regulates the visual and somatosensory inputs which are inherently less reliable under these conditions (eyes closed and foam). This increased reliance on vestibular inputs likely potentiates the effect of vestibular stimulation. Our results provide new evidence for the possible mechanism/s behind noisy galvanic vestibular stimulation's effect on balance, which may help further enhance future clinical use.

C06

High-density electrospinography reveals altered high-frequency oscillations at cervical spinal levels in Multiple Sclerosis: Towards a marker of spinal dysfunction

Prabhav Mehra[1], Helene Arnold[1], Saroj Bista[1], Marjorie Metzger[1], Rosie Giglia[1], Serena Plaitano[1], Matthew Mitchell[1], Peter Bede[1], Muthuraman Muthuraman[2], Madeleine Lowery[3], Orla Hardiman[4], Hugh Kearney[5], Bahman Nasseroleslami[1]

[1]Trinity College Dublin, Ireland, [2]University of Augsburg, Germany, [3]University College Dublin, Ireland, [4]Beaumont Hospital, Dublin, Ireland, [5]St James's Hospital, Dublin, Ireland

Background:

Electrophysiological techniques have been widely used to study the functional mechanisms of the brain's sensory-motor networks in both health and neurodegeneration. However, such assessments of spinal cord activity remain highly challenging and underexplored. This gap is concerning, considering that cervical spinal degeneration[1] is a key clinical indicator of disease severity and future disability in neurological conditions such as Multiple Sclerosis (MS)[2].

To address this gap, our team recently developed a standardised high-density electrospinography (HD-ESG) system (SC10X/U)[3], an up to 76-channel platform designed to reliably record spinal cord electrical activity and enable assessment of spinal function. This is especially relevant in conditions like MS, which features a clinico-radiological paradox, i.e., changes in structural spinal MRI measures, such as atrophy, fail to predict or correlate with disability progression despite strong clinical evidence of spinal involvement.

Objective:

Here, we used HD-ESG to quantify signatures of spinal High-frequency oscillations (HFOs) in response to the median nerve stimulation (MNS), in MS. HFOs are a measure of localised synchronous presynaptic neural activity[4], [5], reflective of functional integrity. To our knowledge, this is the first study to investigate spinal-HFO responses in people with MS (pwMS).

Methodology:

64-channel ESG[3] was recorded in response to the stimulation (right wrist, 1.5 X Motor Threshold, 2Hz) from 14 pwMS and 18 controls. A total of 1400 evoked responses were recorded per participant. Signals were pre-processed and bandpass filtered between 350- 1400 Hz. The evoked HFO (phase-locked) responses were obtained by averaging the resulting signals across all responses.

Temporal HFO characteristics were compared between groups using the Mann-Whitney U test with adaptive false discovery rate (q = 0.05). Characteristics include: onset latency, peak latency, and normalised peak amplitude (Peakamp).

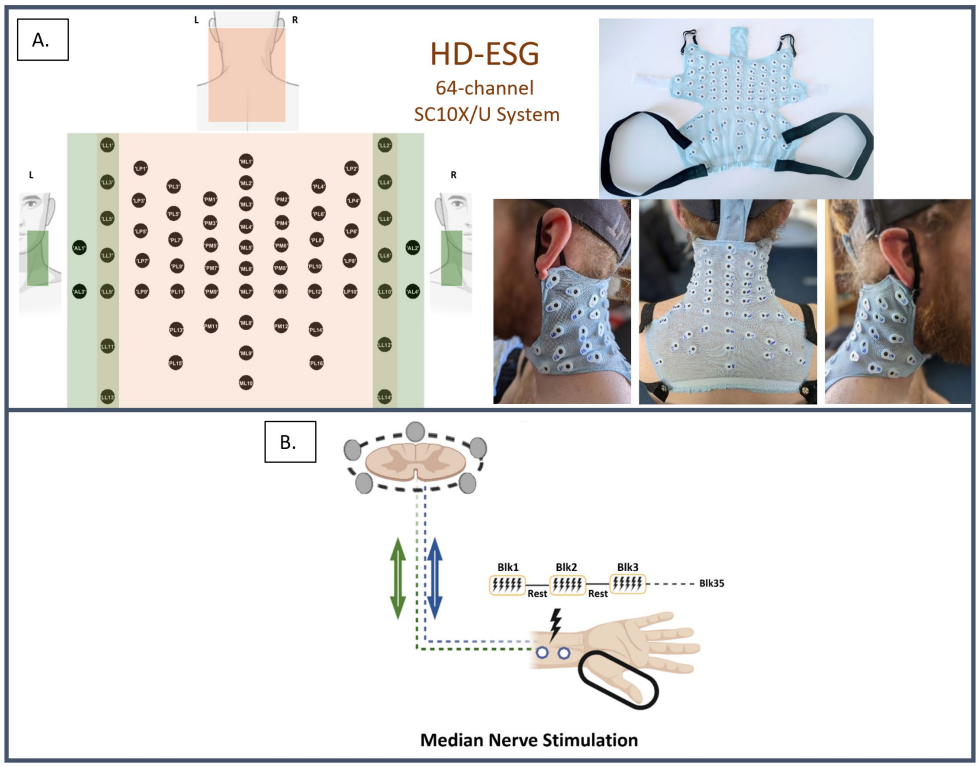
Results:

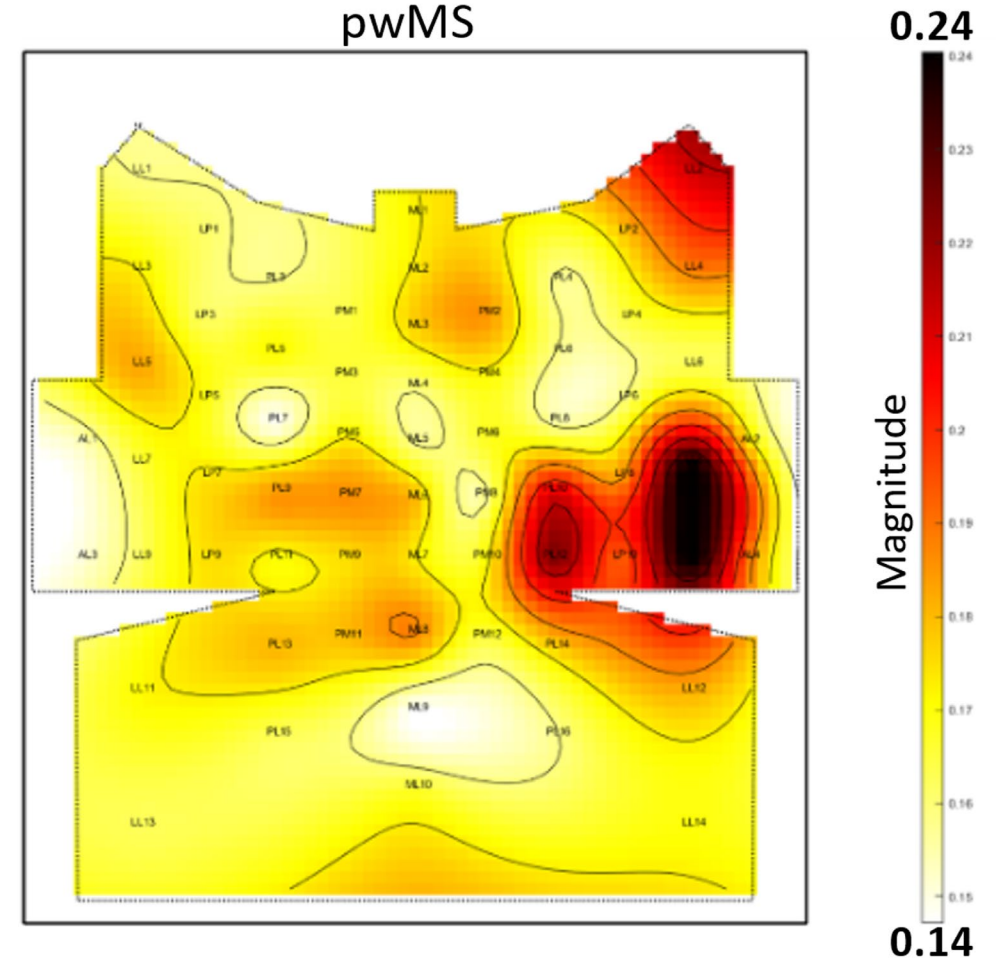
A highly lateralised ($p < 0.05$) evoked HFOs were observed at lower cervical levels, with the maximum response observed at the LL8 electrode (C6 vertebral level (Cv6): right-lateral channel) for both groups *[pwMS: 0.26 ± 0.11, controls: 0.4 ± 0.2]*. The observed Peakamp at the LL8 electrode was significantly lower in pwMS ($p = 0.021$). No significant differences were observed for onset or peak latency.

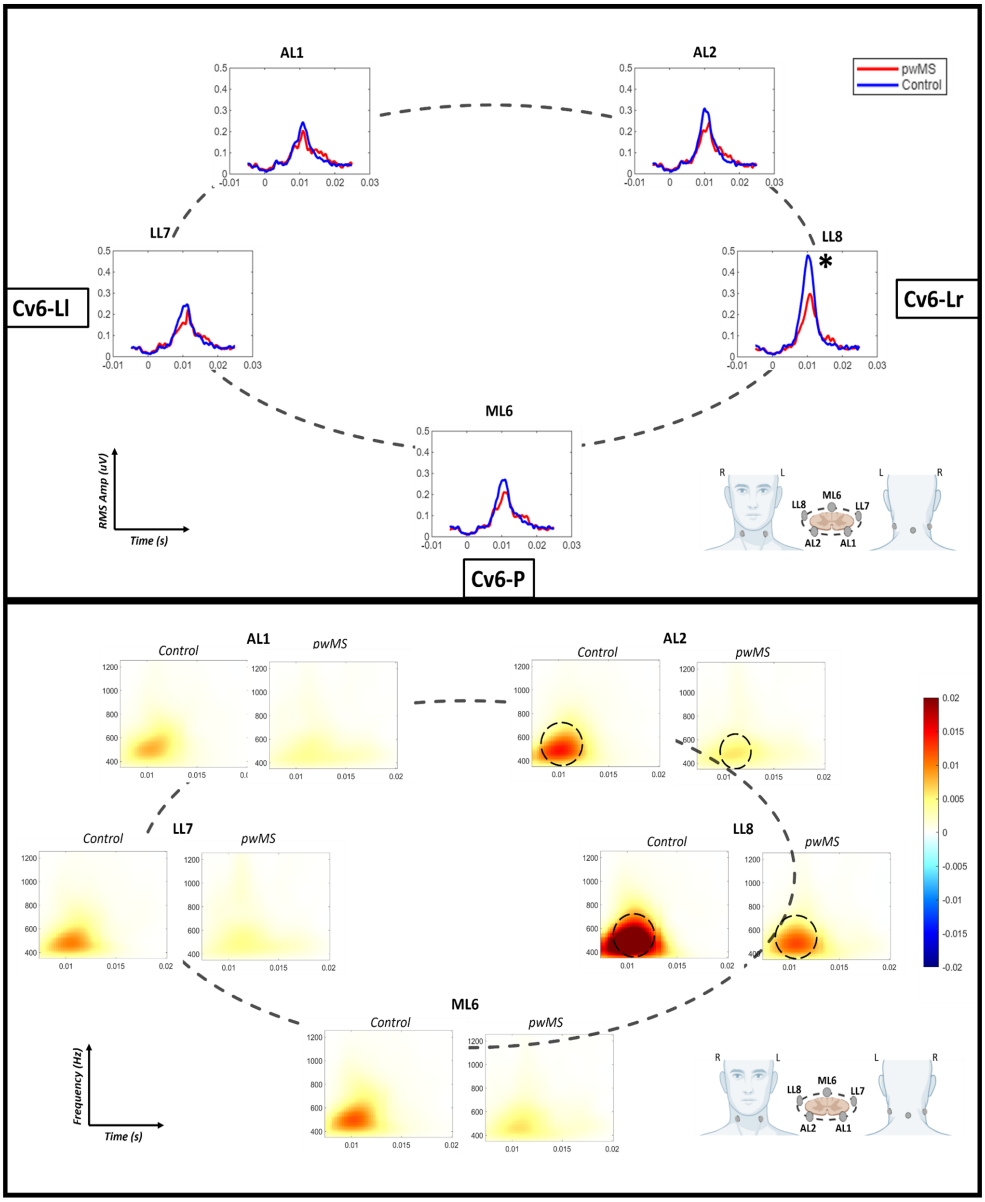
During the 8-15 ms post-stimulation period, both groups demonstrated broadband activity between 400-600 Hz at lower cervical levels. However, this activity was significantly reduced in the pwMS.

Discussion:

This first-of-its-kind study reveals specific disease-relevant alterations in pwMS, highlighting the potential of HD-ESG-based investigations towards a viable marker of spinal dysfunction. The reduction in peak HFO amplitude likely reflects temporal dispersion due to demyelination in MS[6], indicative of impaired cervical spinal integrity. A similar decrease in HFO amplitude has previously been reported at the somatosensory cortex in pwMS[6]. Our findings indicate that these alterations are detectable even at the cervical spinal level, suggesting that HFO-alterations may represent a signature of network-level dysfunction.




pwMS
0.24
Magnitude
0.14


AL1
AL2
pwMS
Control
LL7
LL8
*
Cv6-Ll
Cv6-Lr
ML6
RMS Amp (uV)
Time (s)
Cv6-P
Control
pwMS
Frequency (Hz)
R
L
LL8
ML6
LL7
AL2
AL1

C07

Superior neuromuscular recovery during high-intensity resistance exercise using five-minute versus two-minute rest intervals between sets.

Amy Neeson[1], Rui Wu[2], Jeremy Liegey[2], Rodney Kennedy[1], Conor McClean[1], Gerard McMahon[1]

[1]Ulster University, United Kingdom, [2]University College Dublin, Ireland

Introduction: It is well established that manipulating rest intervals (RIs) between sets can affect acute performance of resistance exercise (RE) such as training volume and intensity (Grgic et al., 2017), which can modulate development of muscular characteristics such as hypertrophy, strength and power. Previous research has highlighted maintenance of greater torque, muscle activity, and volume over multiple sets with five-minute RIs compared to two-minutes (McMahon et al., 2024). However, it has never been shown how RIs modulate specific neuromuscular strategies during RE. This study aims to investigate how neuromuscular behaviour is impacted by a moderate and longer RI duration using high-density electromyography (HDEMG).

Methods: Ten (n=10) participants (male; 29 ± 5 years; 12 ± 6 years RE experience) completed two randomly assigned acute bouts of RE; four-sets of five-second isometric knee extensions to failure at 80% maximal voluntary isometric contractions (MVIC) with two-minute (REST-2) or five-minute (REST-5) RIs.Participants had ≥4-days' rest between sessions. Neuromuscular characteristics were measured using HD-EMG (Quattrocentro EMG, Bio Elettronica) on the vastus lateralis. Muscle fibre conduction velocity (MFCV) was measured during MVICs pre-, 0-MINS-POST-, 5-MINS-POST-, and 10-MINS-POST-exercise. Recruitment threshold (relative to MVIC), firing rates (PPS Mean), coefficient of variation of inter spike interval (CVISI), and motor unit conduction velocity (MUCV) were measured during RAMP contractions at 20%MVIC (increasing 5%MVIC per second until 20% [RAMP-Up] and holding for 10-seconds [RAMP Plateau]) pre-exercise, at the end of RIs between sets, 0-MINS-POST-, 5-MINS-POST-, and 10-MINS-POST-exercise. A linear mixed model was used to examine the effects of rest interval and time on EMG variables.

Results:

MVICs

MFCV, there were no effects (p>0.05).

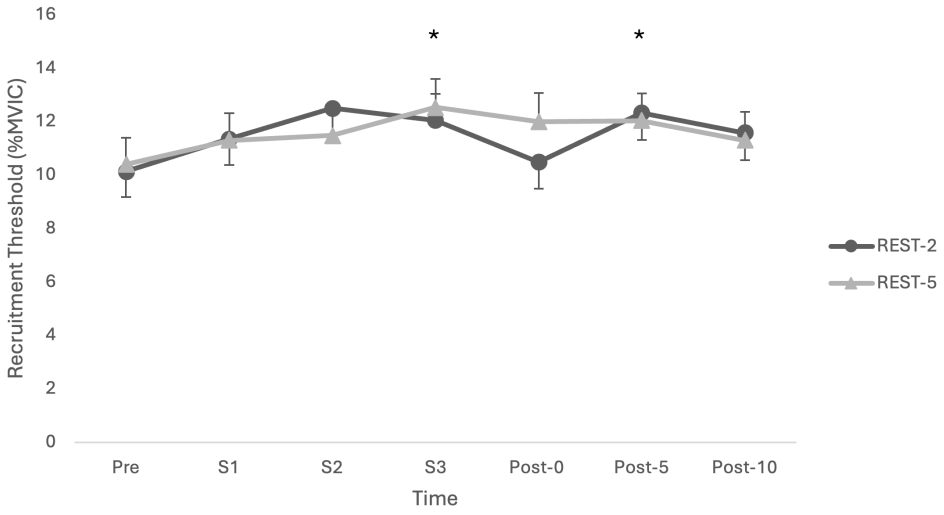
RAMPs

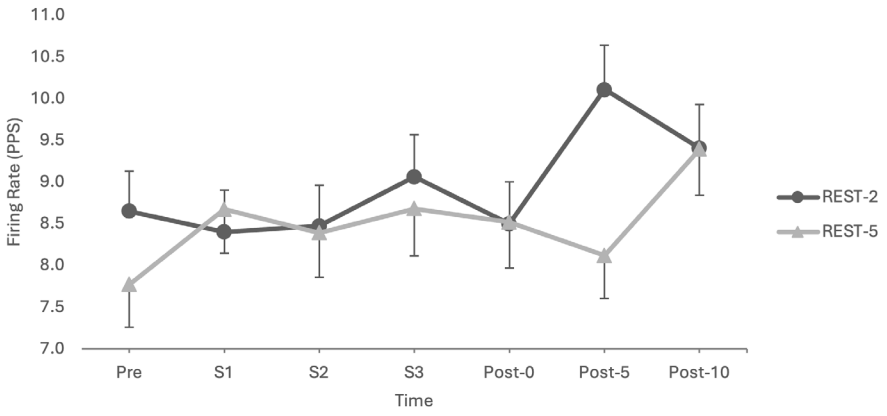
Recruitment threshold showed a main effect for time (p<0.05) but not rest, nor rest × time interaction

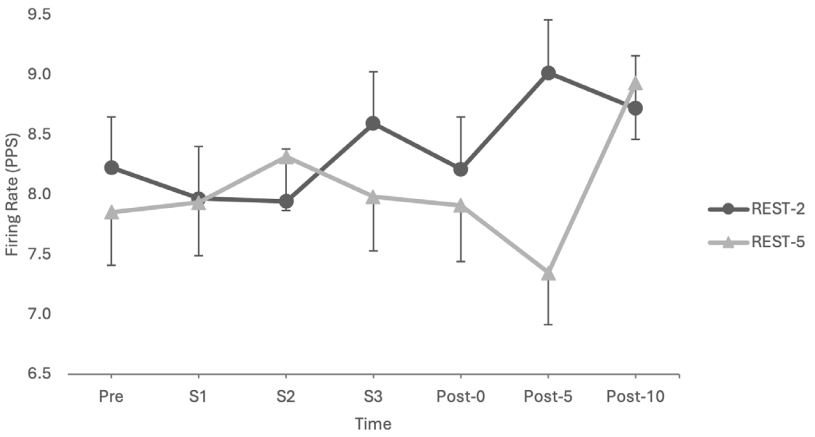
During RAMP-Up, mean firing rates showed a main effect for rest, time, and rest × time interaction (p<0.05) with REST-2 significantly higher than REST-5(Figure 2). For CVISI, there was a main effect for rest (p<0.05).

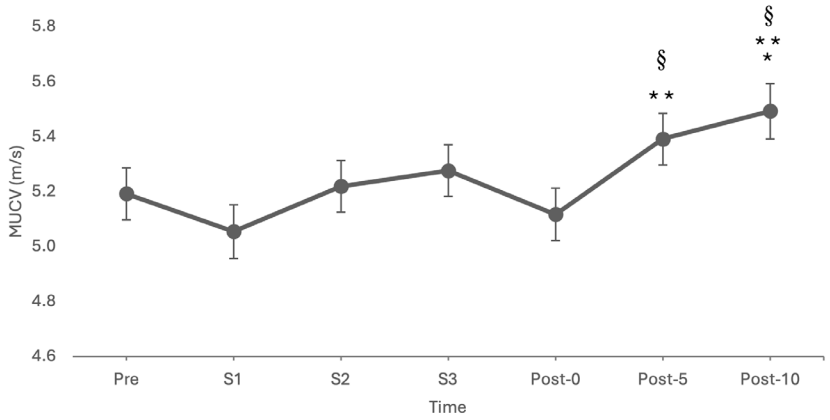
During RAMP-Plateau, mean firing rates showed a main effect for rest and rest × time interaction (p<0.05) REST-2 significantly higher than REST-5 (figure 3). There were no effects for CVISI. For MUCV, there was a main effect for time (p<0.05) (Figure 4), but not rest or rest × time interaction. For MUCV correlation coefficient, there were no effects (p>0.05).

Conclusion: The current findings suggest that five-minute RIs facilitate superior neuromuscular performance compared to two-minutes. The greater firing rates suggests the central nervous system compensating for greater residual fatigue to maintain force output for two-minute RIs. In contrast, lower firing rates shown for five-minute RIs indicate superior recovery going into subsequent sets, and throughout the recovery period.








5.8
5.6
5.4
5.2
5.0
4.8
4.6
MUCV (m/s)
§
* *
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* *
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Pre
S1
S2
S3
Post-0
Post-5
Post-10
Time

C08

The combined contraceptive pill facilitates spike-timing-dependent plasticity in long-term users

Pádraig Spillane[1], Keela Cross[1], Elisa Nédélec[1], Stuart Goodall[1], Kirsty M Hicks[1], Paul Ansdell[1]

[1]Northumbria University, United Kingdom

Introduction: Endogenous ovarian hormones have been shown to contribute to modulations in cortical excitability (1) inhibition (2), and neuroplasticity (3). However, little is known about the physiological effects of the synthetic versions of these hormones (e.g., ethinyl oestradiol), despite morphological changes in brain grey matter volume occurring with long-term use (4). The contraceptive pill is used by 151 million people world-wide and creates a daily micro cycle of synthetic hormones (~ 4-fold changes, 5), but the neurophysiological effects of these acute changes are yet to be investigated. Accordingly, the aim of this study was to determine the effect of the combined monophasic contraceptive pill on corticospinal excitability and plasticity.

Methods: The study received institutional ethical approval. Seventeen female participants (age: 23 ± 5 years) volunteered to take part, all were long term (45 ± 46 months) combined contraceptive pill users. Participants visited the lab in two pill cycle phases: withdrawal and active. Visits comprised of baseline assessments of corticospinal excitability (motor evoked potential [MEP]/Mmax), short-intracortical inhibition (SICI), and intracortical facilitation (ICF) measures, recorded in the resting *first dorsal interosseous*. Participants then took their pill and after 90 minutes rest assessments were repeated. This was followed by a paired associative stimulation (PAS) protocol, utilising ulnar nerve and transcranial magnetic stimulation (25 ms interstimulus interval) to induce spike-timing-dependent plasticity. To assess the time course of spike-timing-dependent plasticity, measurements were repeated at 15 and 30-minutes post PAS.

Results: Prior to taking the pill, corticospinal excitability (MEP/Mmax) was 12 ± 9% lower in the active phase (p <0.001) compared to withdrawal, with no differences observed for SICI or ICF ($p \geq 0.203$). 90 minutes after taking the pill, a further reduction in MEP/Mmax was observed in the active phase (86 ± 3% pre-pill, $p < 0.001$), with no change in the withdrawal phase (98 ± 3% pre-pill, $p = 0.526$). ICF increased pre-to-post regardless of phase ($p = 0.007$), but there were no changes in SICI ($p = 0.347$). PAS elicited an increase in MEP/Mmax in active only, whereby at 15-minutes (118 ± 5% pre-PAS, $p < 0.001$), and at 30-minutes responses were facilitated (119 ± 5% pre-PAS, $p < 0.001$). PAS failed to elicit any facilitation in the withdrawal phase at either 15 minutes (101 ± 5% pre-PAS) or 30 minutes (106 ± 5% pre-PAS, $p \geq 0.424$).

Conclusion: The present data contains two novel findings. Firstly, the oral contraceptive has acute neuroactive effects, reducing corticospinal excitability. Secondly, that oral contraceptive users are only able to experience synaptic plasticity during the active pill consumption phase, with blunted responses when not taking the pill. This has potential implications for neurorehabilitation, with individuals potentially needing to consider timing their pill consumption around motor performance.

C09

Deep brain stimulation in Parkinson's: A rapid review of motor and non-motor outcomes in relation to subtypes and symptom domains

Jakhongir Abdullaev[1], Rhian Thomas[1]

[1]Swansea University, United Kingdom

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterised by both motor and non-motor symptoms, significantly impacting quality of life. Deep brain stimulation (DBS) has emerged as an established therapeutic intervention for advanced PD, particularly in patients with motor complications refractory to pharmacological treatment. However, heterogeneity in clinical presentation across PD subtypes, such as tremor-dominant (TD), postural instability and gait difficulty (PIGD), akinetic-rigid (AR), and mixed (MX), suggests that outcomes may not be uniform. The majority of studies confirmed the efficacy of DBS in a general way, but they do not shed much light on which subtype gains the most, nor on which symptoms should continue to defy treatment (Eghlidos et al., 2022). This rapid review evaluated outcomes of deep brain stimulation in Parkinson's disease, comparing motor, non-motor, and quality-of-life outcomes across established motor subtypes. In addition, this review offers a specific analysis of outcomes by motor subtypes and by item scores derived from the UPDRS.

A structured review protocol guided the study to ensure transparency and best practices. The review followed PRISMA 2020 guidelines (Page et al., 2021). Researchers searched PubMed, Scopus, Web of Science, and Embase. Eligibility criteria followed the PICO model and informed inclusion and exclusion rules. Risk of bias was assessed in all ten studies using the ROBINS-I tool. A systematic search found ten eligible studies; five reported outcomes by subtype and were narratively synthesized. The other five did not stratify by subtype, but gave UPDRS item subscores for tremor (items 20–21), rigidity (item 22), bradykinesia (items 23–26, 31), and axial symptoms (items 27–30); these formed the basis for meta-analysis.

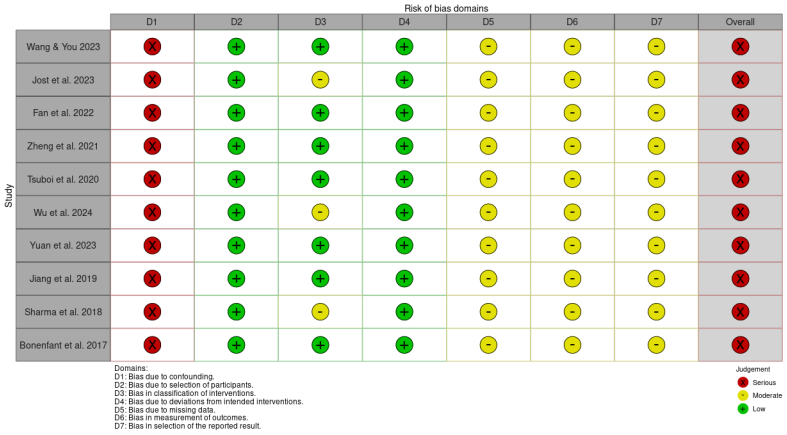
Findings demonstrated that DBS provides substantial global motor improvement, with tremor showing the most robust and consistent response across all subtypes. TD patients achieved the greatest overall benefit, with improvements exceeding 60% in some cohorts. AR and MX patients experienced intermediate benefits, while PIGD patients showed the least favourable outcomes, primarily due to the axial symptoms such as gait and postural instability. Subscore symptom domain meta-analysis also supported the highest improvements in tremor scores, with a pooled mean difference of −3.01 (95% CI −5.44 to −0.59; $Z = 2.44$, $p = 0.01$), whereas axial improvements did not reach statistical significance. Non-motor outcomes were more variable: TD patients tended to maintain cognitive stability, whereas PIGD patients were more vulnerable to postoperative cognitive decline. Quality-of-life improvements were evident across subtypes but differed in magnitude, reflecting baseline disparities.

In conclusion, DBS is an effective but selective intervention in PD, with outcomes strongly influenced by motor subtype and symptom domain. These findings highlight the importance of subtype-specific patient selection, careful preoperative counselling, and multidisciplinary management. Future research should prioritise standardised subtype definitions, systematic assessment of non-motor domains, and long-term follow-up to better inform personalised DBS strategies.

Figure 3.

Traffic light plot for the assessment of the risk of bias of each included study A and weighted plot for the assessment of the overall risk of bias B via the Cochrane RoB 1 tool (B) (n =10 studies).

A



B

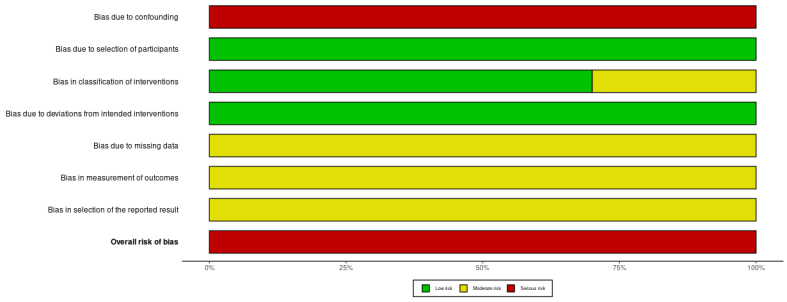
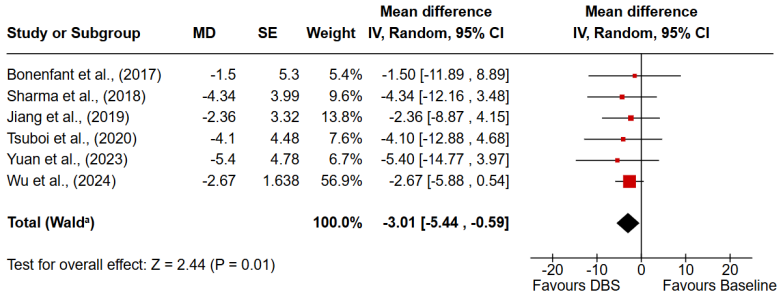


Figure 4A.
Tremor score forest plot



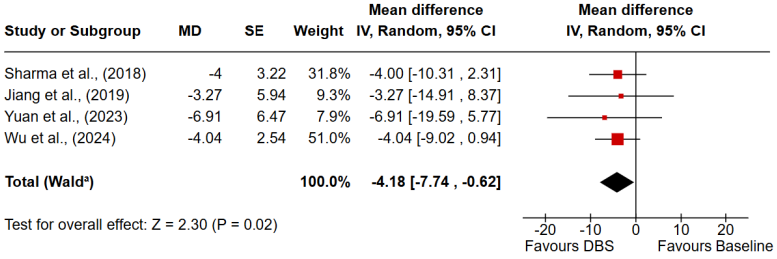
Test for overall effect: Z = 2.44 (P = 0.01)

Heterogeneity: Tau² (REML[b]) = 0.00; Chi² = 0.58, df = 5 (P = 0.99); I² = 0%

Footnotes
[a]CI calculated by Wald-type method.
[b]Tau² calculated by Restricted Maximum-Likelihood method.

Figure 4B.

Bradykinesia score forest plot

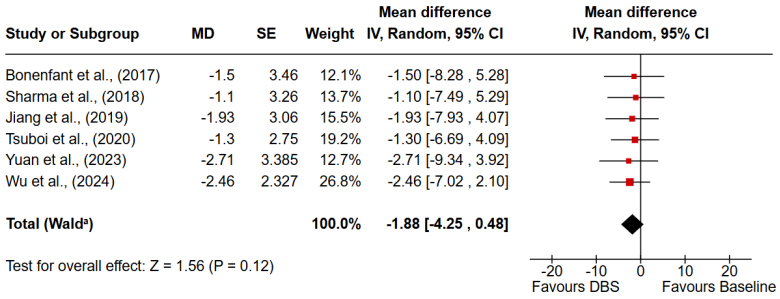


Test for overall effect: Z = 2.30 (P = 0.02)

Heterogeneity: Tau² (REML[b]) = 0.00; Chi² = 0.21, df = 3 (P = 0.98); I² = 0%

Footnotes
[a]CI calculated by Wald-type method.
[b]Tau² calculated by Restricted Maximum-Likelihood method.

Figure 4C.
Rigidity score forest plot

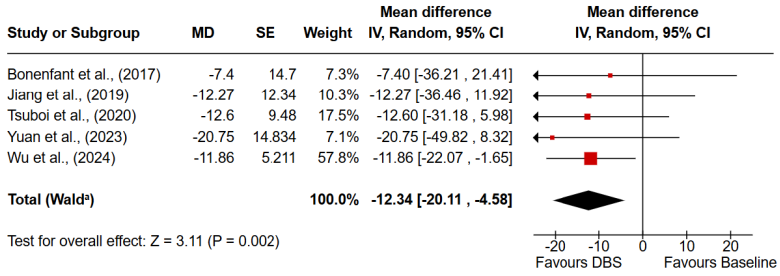


Heterogeneity: Tau² (REML[b]) = 0.00; Chi² = 0.24, df = 5 (P = 1.00); I² = 0%

Footnotes
[a]CI calculated by Wald-type method.
[b]Tau² calculated by Restricted Maximum-Likelihood method.

Figure 4D.

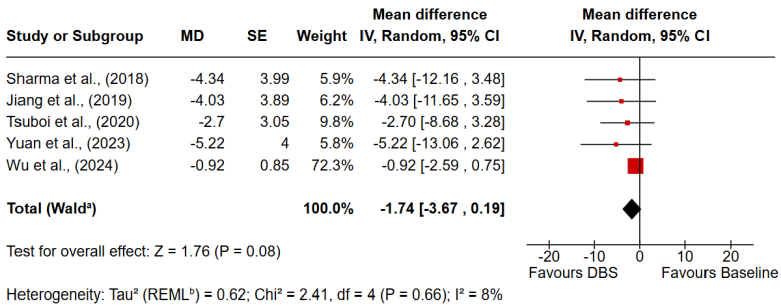
Axial score forest plot



Heterogeneity: Tau² (REML[b]) = 0.00; Chi² = 0.44, df = 4 (P = 0.98); I² = 0%

Footnotes
[a]CI calculated by Wald-type method.
[b]Tau² calculated by Restricted Maximum-Likelihood method.

Figure 4A.
UPDRS III total score forest plot



Footnotes
[a]CI calculated by Wald-type method.
[b]Tau² calculated by Restricted Maximum-Likelihood method.

C10

Age-related changes in sensory reweighting and perceptual delays: The role of adaptation length

Holly Adams[1], Olivia Blundell[1], Anna Truzzi[1], Andrew Monaghan[1], Mihalis Doumas[1]

[1]Queen's University Belfast, United Kingdom

A prevalent issue facing older adults is the increased risk of falling. It is estimated that between 30-60% of older adults will fall each year, having a detrimental impact on quality of life (Rubenstein, 2006). Ability to maintain postural stability is reliant on a complex and dynamic interaction between cognitive, motor, and sensory resources (Horak, 2006). The likelihood of falls or loss of everyday functionality can be attributed to the age-related decline in accuracy of the sensory integration process. Previous studies (Doumas & Krampe, 2010; Craig & Doumas, 2019) suggest that older adults show equally efficient adaptation to environments with inaccurate proprioceptive information about body sway as younger adults, however, de-adaptation seems to be impacted by age-related decline. This study aimed to replicate previously observed effects and explore their link to a common-mechanism of age-related decline in sensory processing. The present study predicted that older adults would demonstrate a longer aftereffect following a balance disturbance as well as take longer to detect the platform stopping. Participants included 45 young adults (YA) (aged 18-35, M = 17, F = 28) and 33 older adults (OA) (aged 70+, M = 15, F = 18). Baseline posture was assessed on a fixed platform with eyes closed for two minutes, followed by either a short adaptation (YA *n* = 23, OA *n* = 17) (condition (sway-referenced platform for one minute), or a long adaptation condition (YA *n* = 22, OA *n* = 16) (sway referenced platform for 6 minutes). This was concluded by a three minute reintegration phase on a fixed platform. Gain was set at 1.0 for older adults and 1.6 for young adults, aligning with research from Craig and Doumas (2019), to reflect differences in adaptation sway for the two age groups. Participants were instructed to press a button when they perceived the platform had started or stopped. Data from all measures were analysed using mixed-design ANOVAs with group (Young, Older) and length (Short, Long) as between- and window as within-subjects factors. Postural sway path length was calculated using L5 position-time trajectories in the AP (anterior-posterior) direction. As predicted, results showed that aftereffects were more pronounced in the long adaptation condition for both age groups, and were greater for older adults. In addition, older adults experienced longer perceptual delay, however, in contrast to our predictions, no differences were found between older adult groups in the long and short condition. The findings suggest that older adults show an age-related decline in sensory re-weighting, as shown through greater aftereffects than younger adults after a long adaptation period. In addition, Older adults experience greater perceptual delay than their younger counterparts after a period of instability. However, robust links between greater perceptual delay and greater aftereffects require further study.

C11

Malocclusion, mitochondrial disease and three-dimensional reconstruction of the muscle spindle column in mouse deep masseter muscle.

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Muscle spindles are mechanosensors crucial for proprioception and skeletal alignment. Previous studies have shown a column of more than 20 muscle spindles within the rodent deep masseter muscle, including rats and mice, suggesting a specialised role in sensory feedback during jaw movement. Mitochondrial defects can cause malocclusion, a congenital misalignment of the jaws. Interestingly, we found that muscle spindle sensory terminals are densely packed with mitochondria, occupying more than 50% of the terminal volume. To investigate how the spindle column arises and is affected by mitochondrial defects, we are determining its precise location and structure throughout development, then in a mouse model of malocclusion.

This initial study aims to develop a technique for creating a 3D model of the spindle column in adult mouse muscle. The right deep masseter muscle was dissected, fixed in 4% paraformaldehyde overnight, before dehydration and wax embedding. The sample was serially sectioned transversely at 5 µm thickness, then stained with haematoxylin and eosin. Sections were scanned, and images were manually aligned using *GIMP* image processing software. *Reconstruct* software was used to trace manually the muscle and muscle spindle outlines within each section, then compiled to provide the 3D visualisation of their spatial distribution.

Preliminary findings confirmed the spindle column's presence near the masseter's anteromedial margin and provided the first comprehensive histological map. Future work will determine the column's location throughout development, and whether malfunctioning mitochondria disrupt its alignment and contribute to malocclusion.

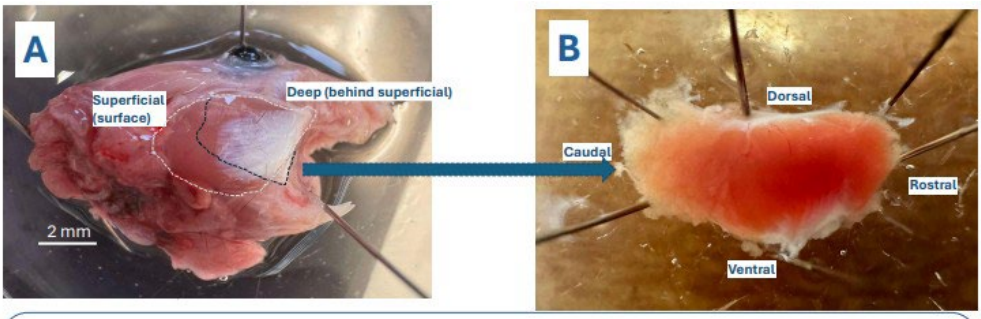

Figure 1
A) Mouse head showing superficial masseter (white line), which will be reflected from the nose towards the posterior of the head, to expose the deep masseter (blue line) underneath.
B) Deep masseter dissected free, from the zygomatic arch (dorsal) and mandible (ventral and caudal). Muscle fibres and muscle spindles run dorsoventrally.

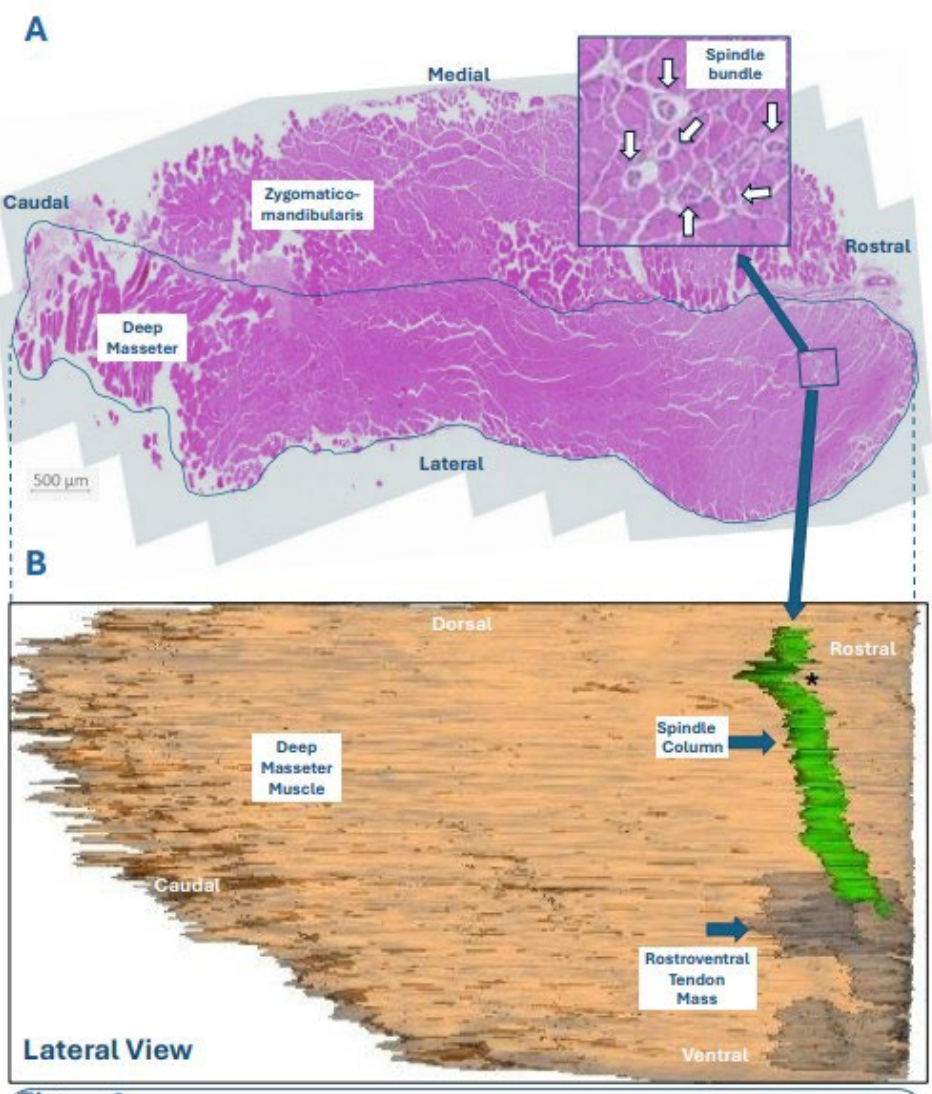

Figure 2

A) Whole masseter (blue outline) and deeper zygomaticomandibularis in transverse section, showing position of masseter spindle column, and (inset) ~8 adjacent individual spindles.
B) 3D reconstruction of deep masseter muscle viewed from lateral aspect, showing rostral position of the spindle column (green) inserting into a thick tendon (grey) at the rostroventral margin of the muscle.
*alignment artefact

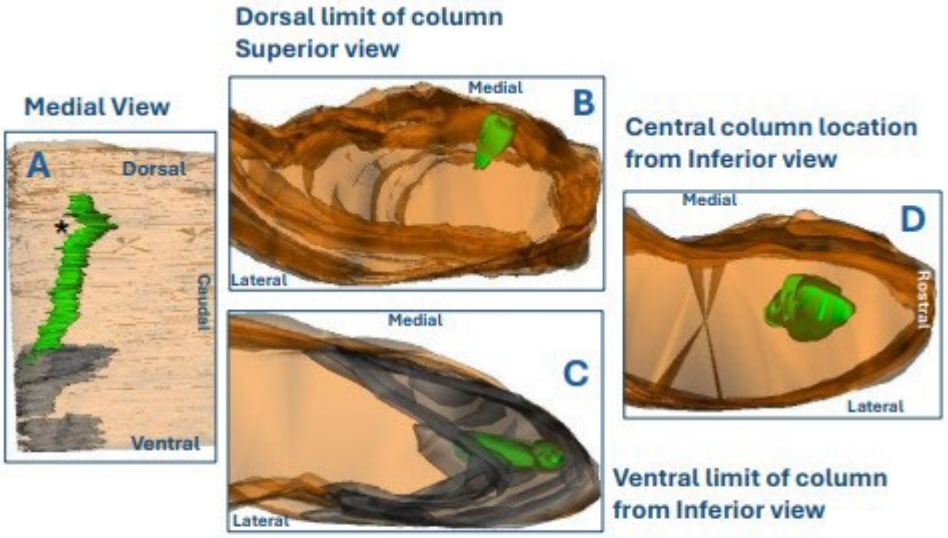

Figure 3

A) Spindle column (green) viewed from medial aspect. Note strong insertion into rostroventral tendon (grey).
B) Dorsal limit of column viewed from superior viewpoint. Note the extreme medial position.
C) Ventral limit of column viewed from inferior position. Note more lateral position of column.
D) Transverse section of column (green) in centre of muscle block, showing it lies in a rostromedial location for most of its length

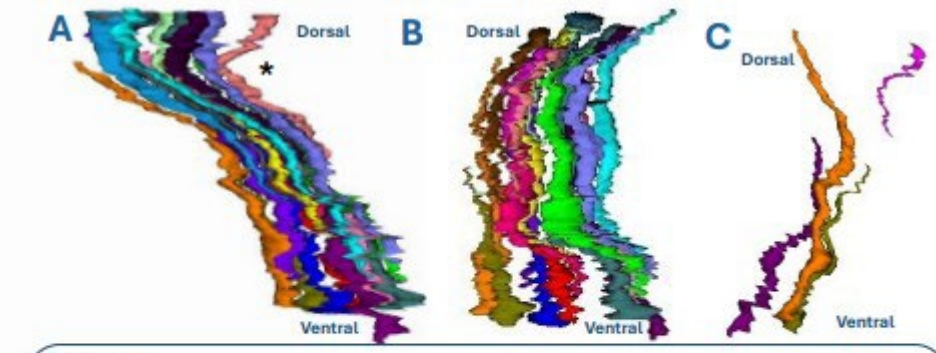

Figure 4

A) Individual muscle spindles (20 spindles in total) viewed from the lateral aspect.
B) Individual muscle spindles as in A) but viewed from the frontal aspect.
C) Example spindles. Note how the spindles run in parallel but are different in length and slightly offset dorsoventrally in origin and insertion. Length: range 620-1240 µm, mean 931.0 ± 41.0 µm.

* column shifts due to alignment artifact.

C12

Evaluating the accuracy of markerless motion capture for upper-limb assessments in stroke rehabilitation.

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Introduction

Arm weakness is the most common impairment affecting the quality of life in stroke survivors [1]. Current clinical interventions do not improve outcomes, and gold-standard motion capture methods for motion analysis are costly and require expertise for setup and analysis, limiting their use in clinical settings. Markerless motion capture (MMC), derived through smartphone and/or tablet cameras and AI pose estimation models, could be a promising, accessible alternative to marker-based systems. However, its accuracy for quantifying upper limb movements commonly assessed in stroke rehabilitation remains unclear.

Aims

We therefore aim to initially investigate the accuracy of MMC for common upper limb assessments in non-hemiplegic upper limbs, before evaluating the performance of the method in affected arms.

Methods

Movement data during a simple functional task of drinking from a cup [2] were recorded concurrently using a gold-standard eight-camera marker-based capture (MBC) system (Vicon, recording at 120 Hz) and two RealSense D435 RGB-depth cameras (30 Hz). MMC data were derived from RGB footage from the RealSense cameras by employing a pose estimation model, RTMPose [3], to extract 2D keypoints, which were then triangulated across camera views to produce 3D coordinates [4].

To establish the accuracy of the MMC system, marker-based data were downsampled to allow data overlay (Fig 1A–E). For each joint movement, the Mean Absolute Error (MAE) for the data aggregate and the Coefficient of Multiple Correlation (CMC) for within-subject agreement were calculated.

Results

The MAE of joint angles and tangential velocity (Figure 1 A-E) between the MMC and MBC systems (n = 4, age: 65.25 ± 6.18 years) when performing the drinking task were measured at key joints and movemnts of teh upper lim (Shoulder flexion-extension MAE=16.08°, 95%CI=4.46; Shoulder adduction-abduction MAE=5.92°, 95% CI=2.43; Elbow flexion-extension MAE=12.23°, 95% CI=6.63; Wrist flexion-extension MAE=9.49°, 95% CI=0.86; Wrist TV MAE=0.16 m/s, 95% CI=0.04). Overall the agreement was good for joint ranges and excellent for tangential velocity.

Intra-individual variability was extremely low between the systems for all movements (Shoulder flexion-extension CMC=0.88, SD=0.05; Shoulder adduction-abduction CMC=0.80, SD=0.08; Elbow flexion-extension CMC=0.97, SD=0.03; Wrist flexion-extension CMC=0.80, SD=0.09; Wrist TV CMC=0.98, SD=0.01; Elbow TV CMC=0.97, SD=0.03). These results demonstrate very good to excellent agreement between the systems.

Discussion and Conclusion

Overall, MMC kinematic measures demonstrated good to excellent agreement with MBC data. However, an offset for the starting position was evident between the systems for shoulder flexion joint angles, and greater variability in wrist movements was noted for MMC. Occlusion, as well as concurrent trunk movements, are likely to contribute to these discrepancies and should be explored with a larger dataset.

Nevertheless, the current findings suggest that MMC can be a promising tool to measure outcome measures of arm impairment after stroke. Future work will investigate the system's ability to accurately measure arm movement in the affected upper limbs of stroke survivors.

Ethical consideration:

The research project has been approved by teh King's College London Research Ethical Committee.

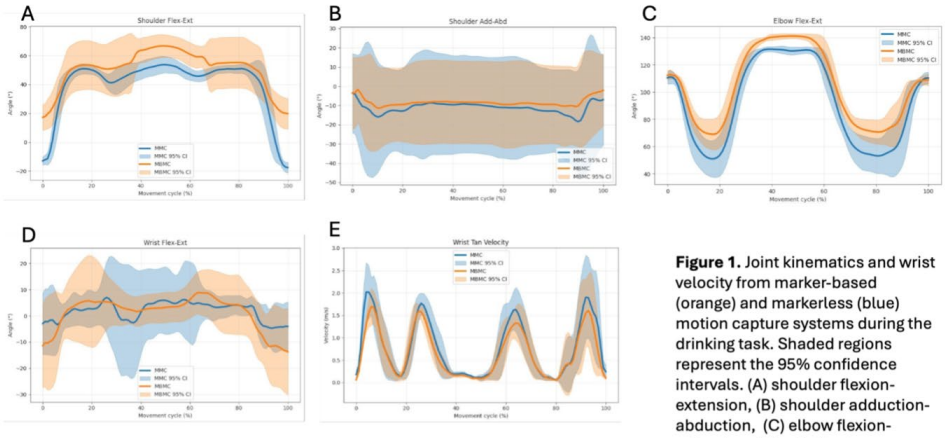

Figure 1. Joint kinematics and wrist velocity from marker-based (orange) and markerless (blue) motion capture systems during the drinking task. Shaded regions represent the 95% confidence intervals. (A) shoulder flexion-extension, (B) shoulder adduction-abduction, (C) elbow flexion-extension, (D) wrist flexion-extension, (E) wrist tangential velocity

C13

Haptic feedback is a key driver of corticospinal excitability in virtual reality settings

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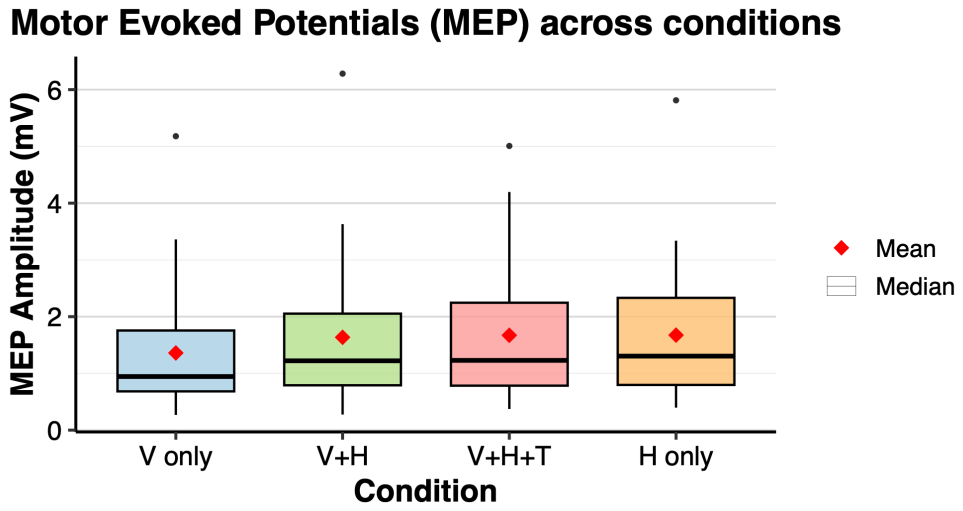
Virtual reality (VR) setups are a promising tool for neurorehabilitation. However, most existing VR therapeutic protocols offer environments exclusively relying on vision, without rendering haptic feedback to users for their interactions with virtual objects. Haptic feedback, including tactile and proprioceptive inputs, is fundamental for sensorimotor function and in particular for skilled hand-object interactions (see Wolpert and Flanagan, 2001). In addition, emerging evidence indicates that the incorporation of multisensory stimuli augments virtual reality experience (Melo et al., 2022) and promotes neuroplasticity (Laver et al., 2017). Here we investigated whether the presence of haptics during hand-object interactions in VR can modulate corticospinal excitability (CSE) at point of object contact.

The experimental design comprised four combinations of sensory feedback conditions: (1) vision only, (2) vision combined with force feedback rendered by a haptic robot, (3) vision combined with force feedback rendered by a haptic robot, and with the actual touch of a real object, and (4) haptic feedback only. Participants (n=26) were instructed to perform a brisk movement with their right index finger to touch either the virtual or real object. A head-mounted display (Meta Quest Pro headset, Meta) was used to generate an immersive virtual environment (controlled by a custom-made Unity platform, Unity Technologies) to present the visual conditions, showing a cube and a stylus, which were calibrated and co-located to correspond to a real physical cube and the participants' index finger, positioned at reachable distance on a table in front of them. A Phantom Touch X robot (3D systems) was used to provide force-feedback haptics.
To assess CSE, we applied single pulse transcranial magnetic stimulation (TMS) using a figure-of-eight coil positioned over the left primary motor cortex. TMS was triggered at the time point the stylus contacted the virtual object. We measured the peak-to-peak amplitude of motor-evoked potentials (MEP) induced in the right first dorsal interosseous (FDI) muscle and the root-mean-square (RMS) electromyographic activity in the same muscle over a period covering 200 ms prior to TMS. Statistical analyses were conducted using non-parametric Friedman's ANOVAs, with further post hoc Bonferroni-corrected pairwise comparisons. These experiments were approved by King's College London ethical committee.

We found a significant main effect of sensory feedback on MEP amplitudes ($chi^2(3)=22.48$, $p<0.001$), suggesting that CSE is differentially modulated depending on the sensory modalities involved in object contact. Post hoc analyses revealed that the 'vision only' condition elicited significantly lower CSE compared to the other three conditions (all $p<0.001$, see figure). These results suggest that CSE is lower when hand-object contact is perceived exclusively through visual input, compared to conditions where any type of haptic feedback is present, irrespective of whether it is combined with visual information. We found no effect of sensory modality on muscle activity RMS ($chi^2(3)=4.02$, $p=0.259$), ruling out any low-level effect of background muscle activity on CSE.

We conclude that the presence of haptics in VR settings is a key driver of motor cortical activity, an important factor to consider for VR protocols aimed at promoting neuroplasticity.


Motor Evoked Potentials (MEP) across conditions
MEP Amplitude (mV)
0
2
4
6
V only
V+H
V+H+T
H only
Condition
Mean
Median

C14

Neuromotor adaptations to the quadriceps muscles following a traumatic injury: A case study

James Forsyth[1], Jonathan Shemmell[2], Deirdre McGhee[1], Christopher Richards[1], Joshua Mattock[1], Julie Steele[1]

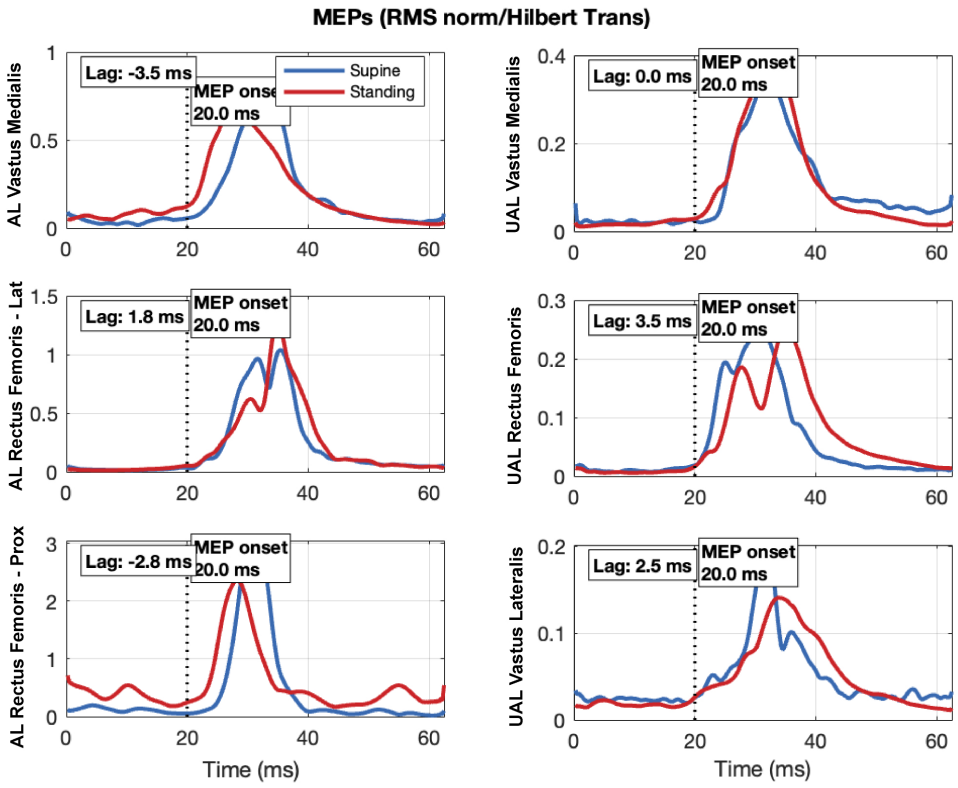
[1]Biomechanics Research Laboratory, University of Wollongong, Australia, [2]Neuromotor Adaptations Laboratory, University of Wollongong, Australia

Background: Traumatic injuries are known to modulate the neuromuscular system and adapt to the feedback processes interrupted, altered or removed by injury (1). Here, we present a case-study of a 26-year-old male (height 178.4cm, mass 67.3kg) who was bitten by a shark on his left lateral thigh whilst surfing. To restore function, a free muscle flap from the latissimus dorsi was inset to the remaining rectus femoris (RF). Following intensive rehabilitation, this surfer returned to surfing at a high level. Using transcranial magnetic stimulation (TMS), we explored how the supraspinal pathways to the lower limbs were affected.

Methods: The protocols were approved by the institution's Human Research Ethics Committee (16/133). Surface electromyography (sEMG) recordings were captured bilaterally from the vastus lateralis (VL), vastus medialis (VM) and RF using bipolar electrodes. On the left, or affected limb (AL), sEMG could not be placed in a similar position to the right, or unaffected limb (UAL), for both VL and RF. The VL sensor was therefore placed over the free muscle flap for the injured RF (RF-Lat), while RF sensor was placed over the proximal, remaining portion of RF (RF-Prox). TMS-induced motor evoked potentials (MEP) were generated at 120% of the active motor threshold, with the participant receiving 20 stimuli in both a standing and supine condition. During the supine condition, sEMG activity was matched to standing for VM of the AL. Waveforms were normalised to their root mean square amplitude. MEP area and cross-correlation lag were compared between conditions and limbs for each muscle, with the waveform differences compared using Statistical Parametric Mapping (SPM; 2).

Results: The transition from supine to standing significantly increased MEP area ($p < 0.05$), including for the VM where the matched sEMG activity would equalise the state of the motoneuron pool. In the AL, cross-correlation lag was -3.5 and -2.8 ms during standing in the VM and RF-Prox, respectively (Figure 1). This was supported by the SPM analyses, which indicated VM and RF-Prox had an earlier onset in the AL when comparing standing to supine. Importantly, the RF-Lat demonstrated no change in MEP onset with the transition to standing, following similar results to the UAL muscles.

Conclusion: The results provide a unique appreciation for how the neuromuscular system adapts to postural challenges following traumatic injury. The earlier MEP onset of the AL VM and RF-Prox suggest a different innervation pattern to the RF-Lat and UAL muscles, which may be explained by two potential mechanisms. This shift in MEP timing may be driven by an increase in excitability of the motoneuron pool for VM and RF-Prox, which is plausible considering that for VM this was matched between conditions to equalise the state of the motoneuron pool. Alternatively, this may be due to changes in neuromuscular state that preferentially increased the excitability of direct corticomotoneuronal cells during standing. In both cases, this may facilitate faster signal transmission to the knee of the AL during dynamic tasks to account for its functional limitations in strength.


MEPs (RMS norm/Hilbert Trans)
AL Vastus Medialis
Lag: -3.5 ms
MEP onset
20.0 ms
Supine
Standing
UAL Vastus Medialis
Lag: 0.0 ms
MEP onset
20.0 ms
AL Rectus Femoris - Lat
Lag: 1.8 ms
MEP onset
20.0 ms
UAL Rectus Femoris
Lag: 3.5 ms
MEP onset
20.0 ms
AL Rectus Femoris - Prox
Lag: -2.8 ms
MEP onset
20.0 ms
UAL Vastus Lateralis
Lag: 2.5 ms
MEP onset
20.0 ms
Time (ms)
Time (ms)

C15

Stopping a Voluntary Sway Action Depends on the Degree of Forward Leaning at the Time of the Stop

Panagiotis Kitsikoudis[1], Irene Di Giulio[2], Vassileios Konstantakos[1], Vassilia Hatzitaki[1]

[1]Aristotle University of Thessaloniki, Greece, [2]King's College London, UK

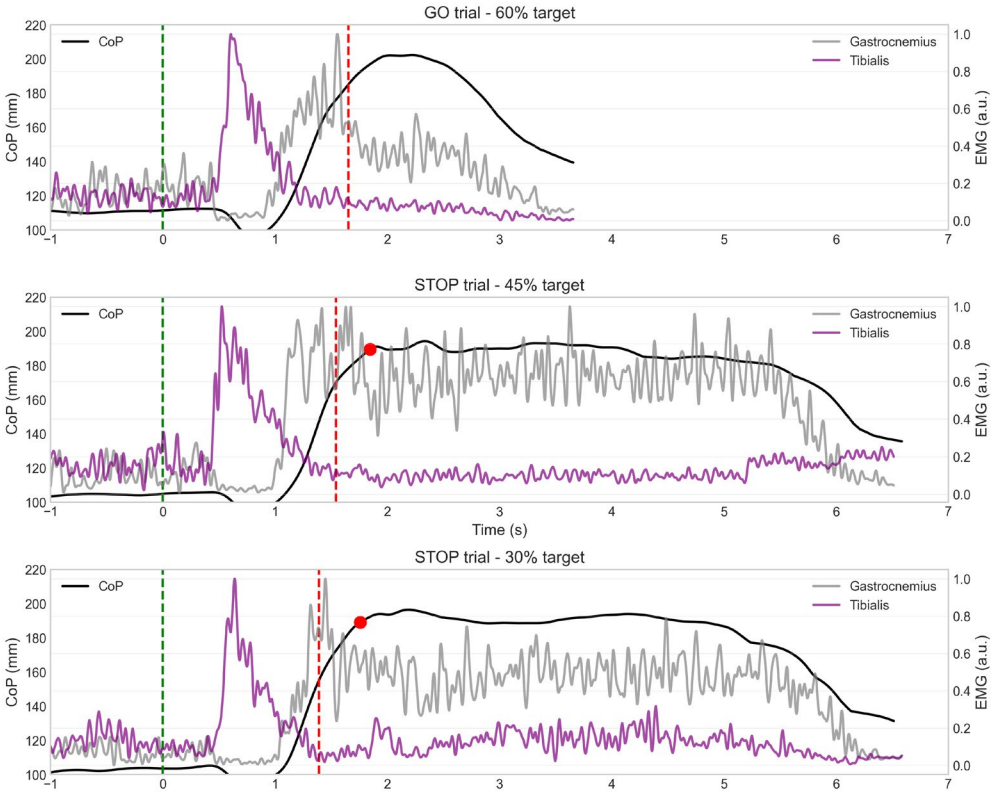
The process of stopping an ongoing action could either originate from the cortex, as global suppression of the motor command regardless of the task-relevant muscles or can be triggered at subcortical levels by activation of the movement antagonists (1). Whether the action is inhibited from the start (no movement at all) or is suppressed at a later stage (stop an ongoing movement) depends on the time of the stop (2).

Objective: In this preliminary study, we investigated the neuromuscular underpinnings of stopping a forward voluntary postural sway action. We predicted that the ability to inhibit forward sway is not only related to time but also to the degree of body incline at the time of the stop.

Method: The study was conducted at King's College London in the Biomechanics Laboratory (ethics MRA-24/25-48273). Ten healthy young adults (6 males, 4 females; mean ± SD age: 30.0 ± 9.80 years; height: 172.83 ± 8.20 cm; body mass: 66.8 ± 13.03 kg; foot length: 256.67 ± 17.08 mm) stood barefooted on a force platform (AMTI OR6-7, MA, USA, 1000Hz) facing a computer monitor (1.5 m) displaying a GO (green target) and a STOP (red target) signal. Participants were fitted with surface electromyography (EMG) electrodes (Delsys Trigno Delsys Inc.,MA, USA, 2000Hz) placed over the gastrocnemius, soleus, tibialis anterior and the deltoid (reference) muscles. EMG and CoP data were synchronously sampled (Vicon Nexus, version 2.12, Vicon Motion Systems Ltd., Oxford, UK). At the onset of the GO signal, participants performed 100 forward sway trials until the CoP reached a target corresponding to 60% of maximum leaning distance. At random trials (28%), the GO target disappeared signaling a STOP, a command to halt forward sway and maintain this posture for 3s. The STOP appeared at two positions, when CoP reached 30% and 45% of the maximum leaning CoP distance. Representative CoP and EMG traces for one participant are shown in Figure 1. Stop signal distance (SSD) and stop signal reaction time (SSRT) were calculated as the distance and time between the STOP trigger and the maximum forward CoP velocity respectively. Outcome measures were compared between the two STOP positions using paired samples t-tests.

Results and conclusions: SSD was greater when the STOP was signaled at the short (30%) than longer (45%) CoP position [$t(9) = 2.8$, $p<.05$]. However, SSRT was not different between the two STOP distances ($p>.05$). Sway velocity at the time of the STOP was also not different between the two STOP distances ($p>.05$). Results suggest that the distance needed to halt voluntary forward sway depends on the degree of body incline at the time of the stop onset highlighting the importance of considering whole-body mechanics in action stopping (3) when compared to upper limb stopping paradigms.

Figure 1: Representative CoP (black) and EMG (gastrocnemius, tibialis) signals during the GO(1st row), the 45% (2nd row) and the 30%(3rd row) STOP trials. The green and red vertical dotted lines indicate the time of the GO and STOP signals respectively whereas the red dot indicates the behavioral STOP.


GO trial - 60% target
STOP trial - 45% target
STOP trial - 30% target
CoP
Gastrocnemius
Tibialis
CoP (mm)
EMG (a.u.)
Time (s)

C16

Age-Related Neuromuscular Junction Dysfunction: Electrophysiological Evidence of Accelerated Decline Beyond 70 Years

Oliver Hayman[1], Abdulrahman Alsowail[1], Mathew Piasecki[2], Stuart Gray[1]

[1]University of Glasgow, United Kingdom, [2]University of Nottingham, United Kingdom

Introduction: Neuromuscular junction (NMJ) degeneration is a key contributor to age-related declines in muscle strength and function. While neuromuscular deterioration with age is well-documented, it remains unclear whether declines accelerate beyond the age of 70.

Aim: To determine whether neuromuscular function declines with age in older adults, and to identify electrophysiological markers sensitive to early neuromuscular ageing.

Methods: Forty healthy older adults (21 males, 19 females; mean age 66.9 ± 5.1 years) were stratified into younger-old (60–69 years; n = 29) and older-old (≥70 years; n = 11) groups. Intramuscular electromyography assessed motor unit potential (MUP) duration, area, phases, firing rate (FR), firing rate variability (FRV), and near-fiber jiggle (NF Jiggle) during knee extensor contractions at 10% and 25% maximal voluntary contraction (MVC). Groups were compared with independent-sample t-tests, and associations between age and NMJ parameters were examined with sex-adjusted regression models.

Results: The ≥70 years group had longer MUP duration (mean difference = 0.84 ms, 95% CI: −1.38 to −0.27, P = 0.004), greater MUP area (mean difference = 57.7 μV·ms, 95% CI: −111.29 to −4.11, P = 0.036), and more MUP phases (mean difference = 0.41, 95% CI: −0.81 to −0.01, P = 0.043) at 10% MVC, with no significant group differences at 25% MVC. Regression showed age was associated with longer MUP duration at 10% (β = 0.065 s/year, 95% CI: 0.014–0.117, P = 0.014) and 25% MVC (β = 0.057 s/year, 95% CI: 0.003–0.111, P = 0.039), higher NF Jiggle at 25% MVC (β = 0.446 units/year, 95% CI: 0.041–0.851, P = 0.033), and lower FRV at 10% MVC (β = −0.001, 95% CI: −0.002 to −0.000, P = 0.044). Muscle thickness, grip strength, and torque were not associated with age (*all* P > 0.05).

Conclusions: NMJ electrophysiological parameters, particularly MUP duration, NF Jiggle, and FRV, appear more sensitive than structural or functional measures for detecting early neuromuscular ageing. These measures may serve as valuable markers for identifying older adults at risk of accelerated decline before clinical weakness or atrophy emerges.

Ethics: The study was approved by the University of Glasgow, College of Medical, Veterinary and Life Sciences Research Ethics Committee and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

C17

From Muscle to Mind: Link Between Handgrip Strength and Mental Health

Liyan Khadeeja[1]

[1]RAKMHSU, UAE

Introduction:
Hand Grip strength (HGS) is a reliable and valid objective parameter to evaluate the functional integrity of the musculoskeletal system.

It can be quantified by measuring the amount of isometric force generated by the hand around a dynamometer.

The physiological parameters of age, body mass, height and gender influence the hand grip strength along with one's occupation, leisure activities, and hand span. Recently studies have reported an association between handgrip and the mental health of the population.

Aims/Objectives:
To investigate the **association between HGS and symptoms of anxiety and depression** among university students, faculty, and staff.

Methods:
A cross-sectional study was conducted at RAKMHSU with **216 participants** (138 females, 78 males; mean age 20.6 ± 2.3 years). Following informed consent, demographic data were collected. Mental health was assessed using the **Generalized Anxiety Disorder Scale (GAD-7)** and the **Patient Health Questionnaire (PHQ-9).** HGS was measured bilaterally using a **Jamar Plus dynamometer** according to standard protocols. Statistical analyses included **Pearson's correlation** to examine relationships between HGS and mental health scores, and independent **t-tests** to compare grip strength between high and normal anxiety/depression groups.

Results:
A **statistically significant negative correlation was found between HGS and both GAD-7 and PHQ-9 scores ($p < 0.05$),** indicating that participants with higher grip strength had lower anxiety and depression levels. This association was more pronounced in females than in males.

Conclusions:
Higher handgrip strength is associated with reduced risk of anxiety and depression, supporting its potential as a simple, non-invasive screening tool for mental health in young adults. Future longitudinal studies are recommended to explore causal relationships and underlying neurophysiological mechanisms.

Ethical Standards:
The study was approved by the institutional ethics committee of RAKMHSU. All participants provided written informed consent.

Keywords: handgrip strength, anxiety, depression, neurophysiology, mental health

Table 1: The scores of various parameters in study subjects

	N	Mean	SD
Age	216	20.62	4.66
JPP LEFT	216	26.77	10.33
JPP RIGHT	216	28.47	11.58
GAD	216	6.38	4.88
PHQ	216	7.44	5.75

JPP: Jamar Plus Plus hand dynamometer,
GAD: Generalized Anxiety Disorder
PHQ: Patient Health Questionnaire

Table 1 shows the overall parameters of the results. The study involved 216 subjects (N) of both genders. The mean scores and respective standard deviation for each parameter is shown.

Table 2

I. GAD	Mean±SD **Group 1** (162)	Mean±SD **Group 2** (53)	P value
JPP LEFT	28.07 ±10.29	22.93 ± 9.57	**<.001**
JPP RIGHT	29.62 ± 11.73	24.96 ± 10.59	**.006**
GAD SCORES	4.14 ± 2.90	13.21 ± 3.07	**<.001**
II. PHQ	**Group 1** (157)	**Group 2** (59)	
JPP LEFT	27.76 ± 9.95	24.13 ±10.92	**.038**
JPP RIGHT	29.14 ± 11.06	26.68 ±12.80	.237
PHQ SCORES	4.63 ± 2.97	14.93 ± 4.50	**<.001**

JPP: Jamar Plus Plus hand dynamometer,
GAD: Generalized Anxiety Disorder
PHQ: Patient Health Questionnaire, SD: Standard Deviation
Group 1: scores 0-9 in both scales; Group 2: scores 10-15+

Table 2 shows the values of various parameters in normal and high scores of GAD & PHQ. A statistically significant decrease in JPP left in both GAD & PHQ high scores are obtained when compared to normal scores.

Correlations of GAD (Generalized Anxiety Disorder)				
		JPP LEFT	JPP RIGHT	GAD
JPP LEFT	Pearson Correlation	1	.944**	**-.058**
	Sig. (2-tailed)		<.001	.682
JPP RIGHT	Pearson Correlation	.944**	1	**-.035**
	Sig. (2-tailed)	<.001		.805
GAD	Pearson Correlation	-.058	-.035	1
	Sig. (2-tailed)	.682	.805	

JPP: Jamar Plus Plus hand dynamometer,
GAD: Generalized Anxiety Disorder
PHQ: Patient Health Questionnaire

Table 3 shows Pearson Correlation of parameters among GAD high scores (Scores >10 in GAD scale). A negative correlation is obtained in both left and right hand of JPP values with the GAD high score cases.

Correlations of PHQ (Patient Health Questionnaire)				
		JPP LEFT	JPP RIGHT	PHQ
JPP LEFT	Pearson Correlation	1	.952**	**-.153**
	Sig. (2-tailed)		<.001	.246
JPP RIGHT	Pearson Correlation	.952**	1	**-.177**
	Sig. (2-tailed)	<.001		.179
PHQ	Pearson Correlation	-.153	-.177	1
	Sig. (2-tailed)	.246	.179	

JPP: Jamar Plus Plus hand dynamometer,
GAD: Generalized Anxiety Disorder
PHQ: Patient Health Questionnaire

Table 4 shows Pearson Correlation of parameters among PHQ high scores (Scores >10 in PHQ scale). A negative correlation is obtained in both left and right hand JPP values for the PHQ high score cases.

C18

Neural mechanisms of behavioral asymmetry in motor control: A systematic review

Taewon Kim[1], Hakjoo Kim[2], Samah Gassass[3], Namarta Kapil[3], Kim Lipsey[3], Benjamin A Philip[3]

[1]The Pennsylvania State University, USA, [2]McLean Hospital, USA, [3]Washington University in St. Louis, USA

Introduction: Upper limb performance asymmetries between the dominant and non-dominant hands are a critical feature of human motor control. However, the neural mechanisms underlying these differences across a continuum of motor behaviors—from simple tapping to complex grasping—remain unclear. This systematic review synthesizes and characterizes the neural mechanisms of hemisphere-specific lateralization in the context of motor control across tasks.

Methods: This study was registered in the PROSPERO database (CRD42021286264). We conducted a systematic review synthesizing n=40 original neuroimaging studies spanning four categories of manual motor tasks in right-handed adults: 1) Finger Pressing/Tapping (n=20), 2) Continuous Precision (n=12), 3) Aiming (n=4), and 4) Grasping/Gripping (n=4). The analysis focused on key motor-related regions of interest (primary motor M1, supplementary motor, premotor, parietal, cerebellum) to identify the unique contributions of contralateral execution vs. ipsilateral support for dominant right hand (RH) and non-dominant left hand (LH) movement. Data were extracted to characterize ipsilateral vs. contralateral BOLD magnitude, structural asymmetry (fractional anisotropy in cerebello-cortical tract), and changes in effective [BP1] [KT2] connectivity.

Results: The synthesis supported functional asymmetries consistent with the complementary dominance hypothesis. The left hemisphere (contralateral to dominant RH) provided optimal control (speed and efficiency) across all tasks, exhibiting effector-independent dominance in planning areas (intraparietal sulcus, premotor) and specialized control for tasks sensitive to rate or complexity. RH control maximizes efficiency through the left hemisphere inhibiting the right hemisphere, including transcallosal suppression of M1 in the ipsilateral left hemisphere. Conversely, LH control is maintained through dynamics coupling that compensates for the right hemisphere's specialization: LH execution requires enhanced interhemispheric coupling to the ipsilateral left hemisphere, which is evidenced by increased ipsilateral M1 and premotor activity[BP3] [KT4] . This coupling is vital for LH skill acquisition and performance and extends to subcortical loops, where the left hemisphere requires unique ipsilateral cerebello-cortical modulation for LH force precision.

Conclusion: Motor performance asymmetry is driven by the dynamic allocation of the optimal control specialization in the left hemisphere, in right-handed adults. The left hemisphere controls the dominant RH via efficient interhemispheric pathways while simultaneously enforcing ipsilateral inhibition. Conversely, non-dominant LH control is achieved by dynamically transmitting the specialized control signal from the left hemisphere via enhanced interhemispheric connections to the contralateral right hemisphere. This framework provides specific neuroanatomical targets for understanding and manipulating motor control mechanisms in neurorehabilitation.

C19

Endurance training induces different brain oxygenation adaptations in the left and right prefrontal cortex during submaximal exercise

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Intoduction: Sufficient blood and oxygen supply to the brain is crucial for optimal performance and exercise tolerance. This study examined the effects of endurance training on oxygen delivery to the right and left prefrontal cortex, hypothesizing that increased aerobic capacity would enhance brain oxygen delivery.

Methods: Sixteen (n=16) male distance runners (age: 34.1±5.1 yrs, body mass: 70.8±4.1 kg) trained five times per week for eight weeks. Training included two high-intensity interval sessions (90-100% VO2max) and three continuous sessions (70-80% VO2max). Before and after training VO2max was assessed on a treadmill and a cycle ergometer. Deoxyhemoglobin (HHb), oxyhemoglobin (O2Hb), total hemoglobin (tHb), and tissue saturation index (TSI) were recorded using functional multichannel near-infrared spectroscopy to assess local tissue oxygenation in the left and right prefrontal cortex, the vastus lateralis, and the biceps brachialis (inactive muscle) during 10 min of submaximal cycling performed at an intensity 5% below the first ventilatory threshold. Oxygen uptake and hemodynamic data were also collected using respiratory gas exchange and noninvasive photoplethysmography, respectively.

Results: VO2max increased in both running (from 55.2±1.2 to 58.8±1.8 ml/kg/min, $p<0.01$) and cycling (from 51.1±2 to 54.3±2.2 ml/kg/min, $p<0.01$). During the 10-min submaximal exercise, post-training increases were observed in O2Hb (27.7%), HHb (37.9%) and THb (30.6%) in both the left and right prefrontal cortex ($p<0.01$). However, TSI decreased in the left (-9.8%, $p<0.05$) and remained unchanged in the right prefrontal cortex. In the vastus lateralis, O2Hb (-27.1%), THb (-18.7%) and TSI (-10.4%) decreased ($p<0.05$), while HHb remained unchanged. Calculated arterio-venous oxygen difference increased (+5.6%, $p<0.05$) after training. In the inactive biceps brachii muscle, O2Hb and THb increased (by +59% and +44%, respectively; $p<0.05$), while HHb and TSI remained unchanged.

Conclusions: Endurance training increases muscle oxygen extraction and reduces blood flow to working muscles, facilitating blood volume redistribution and augmenting oxygen delivery to the brain, during submaximal exercise. The right and left prefrontal cortex appear to adapt differently to endurance training, suggesting distinct functions in regulating aerobic exercise tolerance.

Ethical standards: the study was approved by the University's Ethical Committee for human experimentation and conformed to the Declaration of Helsinki.

C20

Neurophysiological underpinnings of menstrual cycle-related symptom worsening in multiple sclerosis

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Background

Multiple sclerosis (MS) disproportionately affects females, with prevalence more than three times higher than in males. Despite this marked sex difference, relatively little is known about how reproductive hormones, such as oestrogen and progesterone, modulate disease symptoms. While pregnancy and menopause have been explored in the context of MS, the influence of the menstrual cycle on symptom burden remains poorly understood. Individuals with MS experience variable symptoms including fatigue, weakness, and impaired balance or coordination, which some evidence suggests may worsen during low-hormone phases of the cycle (Schwendimann & Alekseeva, 2007; Zorgdrager & de Keyser, 1997). However, a detailed mechanistic explanation of these symptom fluctuations has yet to be established.

Recent work from our group has shown that in healthy females, variations in oestrogen and progesterone across the menstrual cycle modulate motor system excitability. Elevated hormone concentrations around mid-cycle enhance corticospinal excitability, facilitating neural drive to muscle (Piasecki et al., 2024). Conversely, during low-hormone phases, reduced excitability may impair motor control. In MS, reduced corticospinal excitability and impaired intracortical neurotransmission have been linked to greater fatigue and disability (Coates et al., 2020; Conte et al., 2009). Understanding whether hormone-related modulation of motor pathways contributes to symptom variability in MS could therefore inform new approaches to symptom management.

Aims and Objectives

This study aims to (1) provide a comprehensive neurophysiological characterisation of the brain-to-muscle pathway across the menstrual cycle in individuals with MS, and (2) determine how fluctuations in reproductive hormones influence both functional tasks and symptom experience , with the goal of informing tailored therapeutic and self-management strategies.

Methods

Fifty-four individuals with MS (18 naturally menstruating females, 18 hormonal contraceptive users, 18 males) aged 18-40 years will be recruited. Naturally menstruating participants will attend three laboratory sessions corresponding to distinct hormonal timepoints, verified through serum sample hormone assays. Hormonal contraceptive users and male participants will complete matched sessions across comparable time intervals to control for repeated-measures effects.

At each visit, participants will complete validated questionnaires assessing fatigue, mood, and quality of life, alongside functional motor tests evaluating gait, balance, and manual dexterity. Neurophysiological assessments will include transcranial magnetic stimulation (TMS) to quantify corticospinal excitability and intracortical inhibitory and facilitatory neurotransmission, as well as high-density surface electromyography (HD-sEMG) of the tibialis anterior muscle to characterise motor unit behaviour during

voluntary contractions. Data will be analysed to determine phase-related changes in corticospinal and motoneuronal underpinnings of motor function, and their associations with symptom severity.

Significance

This study will be the first to investigate how menstrual cycle-related hormonal fluctuations influence corticospinal and motor unit function in individuals with MS. By integrating neurophysiological and functional assessments, it aims to uncover mechanistic links between reproductive hormones and symptom variability. The findings will provide critical insight into the timing and physiological basis of symptom exacerbations, supporting the development of evidence-based strategies for optimising function, treatment timing, and quality of life in females with MS.

C21

GABA Levels and Motor Performance in Older Adults: A Systematic Review of Biomarker Potential

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Background:

Age-related motor decline is increasingly linked to neurochemical changes, particularly in γ-aminobutyric acid (GABA) signaling. While individual studies suggest associations between GABA levels and motor performance, a structured synthesis is needed to evaluate its potential as a biomarker in older adults.

Objective:

To systematically review evidence on the relationship between GABA concentrations and motor impairment in aging populations, focusing on studies employing magnetic resonance spectroscopy (MRS), serum assays, and multimodal imaging.

Methods:

A comprehensive literature search was conducted across PubMed, Scopus, and Proquest for studies from the date of inception till present. Citation search of included studies were also done in addition. Inclusion criteria targeted original research assessing GABA levels in adults aged ≥60 years, with corresponding motor performance outcomes. Data extraction included study design, GABA quantification methods, motor metrics, and population characteristics. Risk of bias was assessed using standardized tools.

Results:

Out of 422 studies thirteen studies met inclusion criteria, (10 from database search and 3 fro citation search) after comprehensive screening for title, abstract and full text encompassing diverse methodologies such as MRS-fMRI, TMS-MRS, and serum biomarker analysis. Across studies, reduced GABA levels were consistently associated with impaired motor function, diminished inhibitory control, and attenuated neuroplasticity. Sensorimotor GABA concentrations—both baseline and task-modulated—emerged as potential predictors of motor decline. However, heterogeneity in measurement techniques, outcome definitions, and population sampling limited direct comparability and precluded quantitative synthesis.

Conclusion:

This systematic review highlights a consistent association between lower GABA levels and motor impairment in older adults. GABA may serve as a promising neurochemical biomarker for age-related motor decline. Future research should prioritize harmonized protocols, longitudinal designs, and standardized motor assessments to validate predictive utility and inform targeted interventions.

C22

Effects of a 4-week Electrical Muscle Stimulation programme on neural drive to trunk muscles

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Introduction: Critically ill patients frequently suffer from significant muscle wasting, or sarcopenia. Electrical muscle stimulation (EMS) offers potential as a rehabilitation tool, having been shown to improve muscle strength and neural drive; both commonly reduced in deconditioned patients. However, EMS research has predominantly focused on limb muscles; thus, the effect of EMS on trunk muscles, particularly in relation to neural drive, remains unclear. Given the vital role of the trunk in maintaining stability, balance and posture, this knowledge could be crucial for improving the recovery of such patients. This study represents the first to assess the impact of a 4-week EMS programme on the neural drive to trunk muscles.

Methods: With ethical approval (SETREC No. 6948844), healthy adult participants were randomly assigned to Intervention (n=6, mean±SD age=21.5±0.55 years) or Control (n=6, mean±SD age=21.7±0.52 years) groups. Intervention participants completed a 25-minute, home-based EMS programme (DiPulse) targeting Rectus Abdominis (RA) and Erector Spinae (ES) three times a week, for four weeks. Control participants continued their usual activity levels. Neural drive was assessed via Transcranial Magnetic Stimulation-induced twitch interpolation, pre- and post-intervention in both muscles. Maximum Voluntary Contraction (MVC) torque, Motor Evoked Potential (MEP) amplitudes and time-to-peak (TTP) twitch amplitude were recorded pre- and post-intervention, at three different contraction strengths (50%, 75% and 100% of MVC). Trunk function was also assessed. Differences between parameters were assessed using two-way analysis of variance (ANOVA) with factors group (intervention/control) and time (pre/post) or contraction strength (50%, 75% and 100% MVC). The level of significance was set at $p<0.05$.

Results: Following 4-weeks of EMS, statistically significant increases in the mean ES MEP amplitudes were observed, at all contraction strengths levels (50%-pre:0.679±0.499mV, post: 0.901±0.466mV, $p=0.02$; 75%-pre: 0.870±0.811mV, post: 1.365±0.861mV, $p<0.0001$; MVC-pre: 0.655 ± 0.394mV, post: 1.272 ± 0.589mV, $p<0.0001$). No significant changes were observed in control group. These results imply increased corticospinal excitability to ES, suggestive of EMS-induced neuromodulation. Although no statistically significant improvements in neural drive, trunk muscle strength, function, or TTP amplitude were seen, similar discrepancies have been reported in the literature, where MEP amplitude changes fail to align with functional outcomes. Thus, the increase in corticospinal excitability in ES, whilst significant, may have been inadequate to produce any measurable functional benefits over 4-weeks. This could be attributed to insufficient EMS parameters used, inclusion of healthy participants with limited potential for neurophysiological improvement, or inherent differences in neural control between trunk and limb muscles, where greater effects have previously been demonstrated.

Conclusion: A 4-week EMS programme targeting RA and ES resulted in a significant increase in ES MEP amplitudes but did not alter neural drive or MVC strength in either muscle. These findings indicate that EMS may induce neural adaptations in the trunk musculature, although further investigation with greater sample sizes is needed to determine the underlying neuromodulatory mechanisms. While the utility of this intervention lies with sarcopenic patients, this preliminary study in healthy individuals demonstrates, for the first time, the feasibility of using EMS to increase corticospinal drive to trunk muscles.

C23

Visuomotor Adaptation with Fresnel Prisms: Gender Influence and White Noise

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This study investigated visuomotor adaptation, a neurological process crucial for modulating motor responses based on visual and environmental cues, which involves transforming visual signals into motor commands, error correction, and multimodal recalibrations, with attentional processes playing a significant role. The primary aim was to assess the effects of white noise on visuomotor adaptation accuracy and efficiency in adults, hypothesizing that it would improve performance by enhancing concentration, and to explore gender-related influences. Fifty participants (N=50, 31 women; 19 men) with normal or corrected vision and no motor/hearing limitations engaged in three tasks: a Control (T1) without a controlled auditory stimuli (performed on the ambient sound environment), an experiment with Headphones (T2) without white noise, and an experiment with Headphones and White Noise (T3), all involving a pointing task with 35-diopter Fresnel prisms to induce visual distortion. Data, including maximum/minimum displacement and displacement tendency, were analysed using repeated measures ANOVA. Post-hoc analyses revealed significant differences in maximum displacement values between the first attempt of Task 1 and Task 2 ($p=0.003$), indicating T1 outperformed T2, while no significant differences were observed between Task 1 and Task 3 ($p=0.446$) or Task 2 and Task 3 ($p=0.188$). Regarding minimum displacement values, no significant differences were found between Task 1 and Task 2 ($p=0.446$) or Task 1 and Task 3 ($p=0.188$). A significant difference was found between the maximum and minimum displacement values within each task (T1, T2, T3) from the first to the last attempt ($p<0.001$). For displacement tendency, no statistically significant differences were noted between Task 1 and Task 2 ($p=0.299$) or Task 2 and Task 3 ($p=0.299$), but a significant difference emerged between Task 1 and Task 3 ($p=0.032$), demonstrating that Task 3 (displacement tendency: 0.032±0.09) required more attempts to reach the target compared to Task 1 (displacement tendency: −0.03±0.1).While no significant gender differences were found in maximum or minimum displacement, a notable distinction emerged in displacement tendency: women exhibited to achieve the goal in less attempts in Task 1 ($p < 0.001$), whereas men showed a more efficient displacement tendency in Tasks 2 and 3, adapting in fewer attempts ($p < 0.001$). Further analysis revealed that the commercially used white noise was not fully spectrally balanced (different constant frequencies from 20 Hz to 20,000 Hz.), and a subsequent test with corrected white noise indicated a more efficient displacement tendency. In conclusion, optimal visuomotor adaptation in this context occurred without external auditory stimuli, while headphones alone hindered it. White noise modulated these effects, influencing brain activation and spatial attention, with its efficacy potentially linked to its spectral composition. Crucially, significant gender differences in adaptation strategies were observed, highlighting a gender-dependent effect of white noise, which was not influenced by age, suggesting a critical role for attentional systems in visuomotor performance when controlled external auditory stimuli are present.

C24

Motor unit firing rates of the flexor digitorum brevis muscle in young and older individuals

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Introduction: Deterioration in muscle strength and function is an inevitable consequence of the normal ageing process. The intrinsic foot muscles, which stabilise the medial longitudinal arch and contribute to balance and mobility, are particularly affected. Such declines increase the risk of falls and impair activities of daily living. This phenomenon reflects alterations in motor unit (MU) properties, which may include MU firing. Intrinsic foot muscle strength is important for foot function, with the flexor digitorum brevis (FDB) muscle contributing to postural control (Okai and Kohn, 2015, Fukuyama et al., 2024). To date, no studies have yet investigated the effect of age and sex on MU firing in the FDB muscle. This study aimed to compare the MU firing rate during submaximal isometric toe grips in young and older individuals of both sexes.

Methods: Following institutional ethical approval, fifteen young (6 females, aged 22.8 ± 2.8 years, BMI: 24.12 ± 4.15 kg/m^2) and 14 older (5 females, aged 66.6 ± 3.8 years; BMI: 23.73 ± 2.76 kg/m^2) participants volunteered for the study. Participants were tested for maximal isometric toe grip force (MTGF) and sustained isometric toe grip at 10, 20, 30 and 40%MTGF using a toe grip dynamometer. High-density surface EMG signals from the FDB muscle were simultaneously recorded using a 611 grid of 64 electrodes. Force steadiness was estimated as the coefficient of variation. MU firing rate and firing rate variability (i.e., CoV-ISI) were estimated from decomposed motor units.

Results: No age-related differences in height, weight or BMI were observed. Males exhibited a greater age-related decline in MTGF (170.7 ± 64.9 vs 97.8 ± 34 N; $p < 0.001$), whereas no difference was observed between young and older females (81.24 ± 22.3 vs 81.1 ± 27.4 N). No age-related differences were observed in MU firing rate or its variability during submaximal contractions. However, a main effect of contraction intensity was observed in both MU firing rate ($p < 0.001$) and CoV-ISI ($p = 0.002$). MU firing rate was lowest at 10%MTGF (all $p < 0.05$) when compared to other intensities and was lower at 20%MTGF than at 30 ($p = 0.02$) and 40%MTGF ($p < 0.04$). COV-ISI at 10%MTGF was lower than at 40%MTGF. Similarly, force steadiness was lowest at 10%MTGF when compared to the other intensities (all $p < 0.001$).

Conclusions: This study is the first study to examine the FDB motor unit firing behaviour in young and older individuals of both sexes. During submaximal isometric toe grips, no age-related differences in MU firing rate or variability were observed. However, the firing rate increased with contraction intensity, and CoV-ISI was lowest at 10%MTGF, consistent with the force steadiness pattern. Despite preserved MU firing, older males exhibited a more pronounced reduction in maximal toe grip force, but this was not observed in females. These preliminary results suggest an intensity-dependent modulation of MU behaviour in the FDB muscle and highlight the need for larger cohorts or longitudinal studies to elucidate the neuromuscular determinants of age-related loss of MTGF.