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Maximising neuromuscular performance in people with pain and injury: moving beyond reps and sets to understand the challenges and embrace the complexity

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ABSTRACT

Rehabilitative practice is often criticised for being non-individualised, monotonous and not well aligned with foundational principles that drive continued physiological adaptation(s). However, our understanding of neuromuscular physiology is rapidly increasing and the way we programme rehabilitation is improving. This viewpoint highlights some of the potential considerations around why the adaptations achieved during rehabilitation programmes may be suboptimal. We provide basic, clinician-focused discussion about potential confounding physiological factors, and put forward several exercise-based programming recommendations and novel approaches to consider in contemporary rehabilitative practice. Specifically, we outline several potential mechanisms contributing to poor muscle activation and function that might be present following musculoskeletal injury. However, clinicians require strategies capable of attenuating these impairments to restore proper function. Therefore, we also provide an overview of recommended strength and conditioning guidelines, and novel strategies (such as external pacing and electrical stimulation techniques) that clinicians can consider to potentially improve the efficacy of musculoskeletal rehabilitation.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Rehabilitative practice is often criticised for being non-individualised, monotonous and not well aligned with foundational principles that drive continued physiological adaptation(s).

WHAT THIS STUDY ADDS

⇒ This viewpoint focusses on several, often overlooked neurophysiological outcomes that may contribute to impaired function in common musculoskeletal conditions.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ We put forward several exercise-based programming recommendations and novel approaches to consider in contemporary rehabilitative practice to optimise exercise rehabilitation outcomes.

INTRODUCTION

Rehabilitative practice is often criticised for being non-individualised, monotonous and not well aligned with foundational principles that drive continued physiological adaptation(s). One example is the prescription of 3×10 repetitions, which was initially developed for the rehabilitation of soldiers in World War II, and its use has continued in many settings.[1] Despite this approach having merit, the field has advanced with the implementation of more contemporary strength and conditioning principles and adjunct therapies.

In healthy populations, appropriate resistance training facilitates improvements in muscle cross-sectional area, strength, power and function, which are viewed as important modifiable factors for the resolution or prevention of musculoskeletal pain and injury. However, despite the known benefits of resistance training, these are less apparent in many pathological populations.

In a recent systematic review of resistance training protocols for Achilles tendinopathy almost no studies were able to demonstrate a significant improvement in muscle size, endurance or strength after months of resistance training.[2] While it would be simple to say the programmes were poorly prescribed, some trials used what would be considered 'good' strength and conditioning principles.[2] Further, in a separate longitudinal meta-analysis where all included studies reported improvements in pain during the rehabilitation period, a rehabilitative plateau (whereby symptoms tend to stop improving after 12 weeks of resistance training) has been

observed.[3] This was even seen in studies where progressive overload was employed.

This viewpoint focusses on several, often overlooked neurophysiological outcomes that may contribute to impaired function in common musculoskeletal conditions and how these may potentially be addressed during rehabilitation programmes to overcome neuromuscular deficits. We provide basic, clinician-focused discussion about potential contributing neurophysiological factors, and put forward several exercise-based programming recommendations and novel approaches to consider in contemporary rehabilitative practice to optimise exercise rehabilitation outcomes.

Impaired voluntary activation

Musculoskeletal conditions are often associated with muscle weakness and impaired voluntary activation (ie, reduced ability of the nervous system to drive the muscle to produce maximal force). For reference, quadriceps voluntary activation in healthy people is typically >90.0%.[4] Impaired voluntary activation is present in many musculoskeletal conditions including ACL rupture[5] and knee osteoarthritis (mean voluntary activation as low as 66.0% in some studies).[4] Generally speaking, impaired voluntary activation likely results from various mechanisms including altered sensory afferent/nociceptive feedback, or descending drive that impacts the ability to activate motor units (see the following sections).

Impaired voluntary activation presents a rehabilitative problem due to difficulty recruiting muscle fibres during training. Although voluntary activation may improve over time with appropriate training, impairments are often still observed in populations who have undergone long periods of rehabilitation (eg, ACL reconstruction).[5] Further, in the presence of a voluntary activation deficit lower mechanical force can be produced during contraction and minimal physiological stress of non-activated fibres will likely occur. We suggest that these effects contribute to a suboptimal adaptive stimulus. Specifically, the inability to adequately activate the entire motor unit pool may mean that higher threshold units (those responsible for high force production and those with most the hypertrophic potential (ie, type II fibres)) are not well activated during exercise. Additionally, poor activation and/or tissue deconditioning may result in greater 'fatigability', meaning that the overall training stimulus (eg, volume-load) achieved is likely to be lower than optimal and/or less tolerable. The subsequent sections discuss some factors that may contribute to impaired voluntary activation.

Altered motor drive

In musculoskeletal conditions where weakness, deconditioning or pain exists motor cortex excitability and descending corticospinal drive often differ from healthy controls. At the cortical level, altered intracortical inhibition or facilitation may impact descending motor drive. Such alterations have been observed in ACL rupture,[6 7] tendinopathy[8] and hamstring muscle strain injury.[9] However, observed changes appear non-uniform across studies and the direct impacts on muscle force production are difficult to decipher. Comparisons to healthy individuals suggest that altered excitability is either a potential mediating factor in, or consequence of poor muscle function and activation given the differences between groups. Importantly, increased excitatory descending drive correlates with strength adaptation after specific types of strength training[10] and so interventions that improve motor drive may be beneficial.

Further along, altered synaptic drive from descending tracts (primarily corticospinal) or afferent pathways can impact motoneuron firing. In a non-neurological disease model where muscle weakness exists and deconditioning is prevalent (eg, ageing), reduced motoneuron firing rates are reportedly due, at least somewhat, to poorer function of the motoneurons themselves.[11] Ultimately, this can make maximal effort contractions produce less force. Evidence also suggests muscle atrophy and fibre type shifts occur rapidly in response to deconditioning.[12] Again, motor units having the most force production capacity (ie, those innervating type II fibres) are more susceptible to such negative effects.[13] Unfortunately, most research evidence only considers components of the motor pathway in isolation, limiting insight into mechanisms contributing to poor motor function.

Increased pain-related muscle inhibition

It is well accepted that musculoskeletal pain[14] (and other potential drivers such as kinesiophobia) can impact descending motor drive and muscle activation. Specifically, afferent feedback can alter motor drive at spinal and supraspinal levels.[15] Experimental studies show reduced voluntary activation (even of homologous muscles), reduced motor unit firing[16] and altered intracortical network excitability (due to changes in both inhibition and facilitation) during experimentally induced muscle pain. Any subsequent decrease in muscle activation could limit force production, and hence training efficacy. This suggests that non-individualised, monotonous programmes that do not consider physiological underpinnings and appropriate training principles may prescribe suboptimal loads (eg, under or even potentially overdose) which can be problematic over the longer term resulting in poor outcomes. This will not be news to clinicians, but it does emphasise the importance of ensuring sports medicine practitioners consider how they can best facilitate resistance training while ensuring that pain with exercise is minimal.

MANAGEMENT

We have outlined 'some' potential mechanisms of poor muscle activation and function that might be present in musculoskeletal injury. However, clinicians need strategies capable of attenuating these impairments to restore proper function. In the following sections we have highlighted recommended strength and conditioning

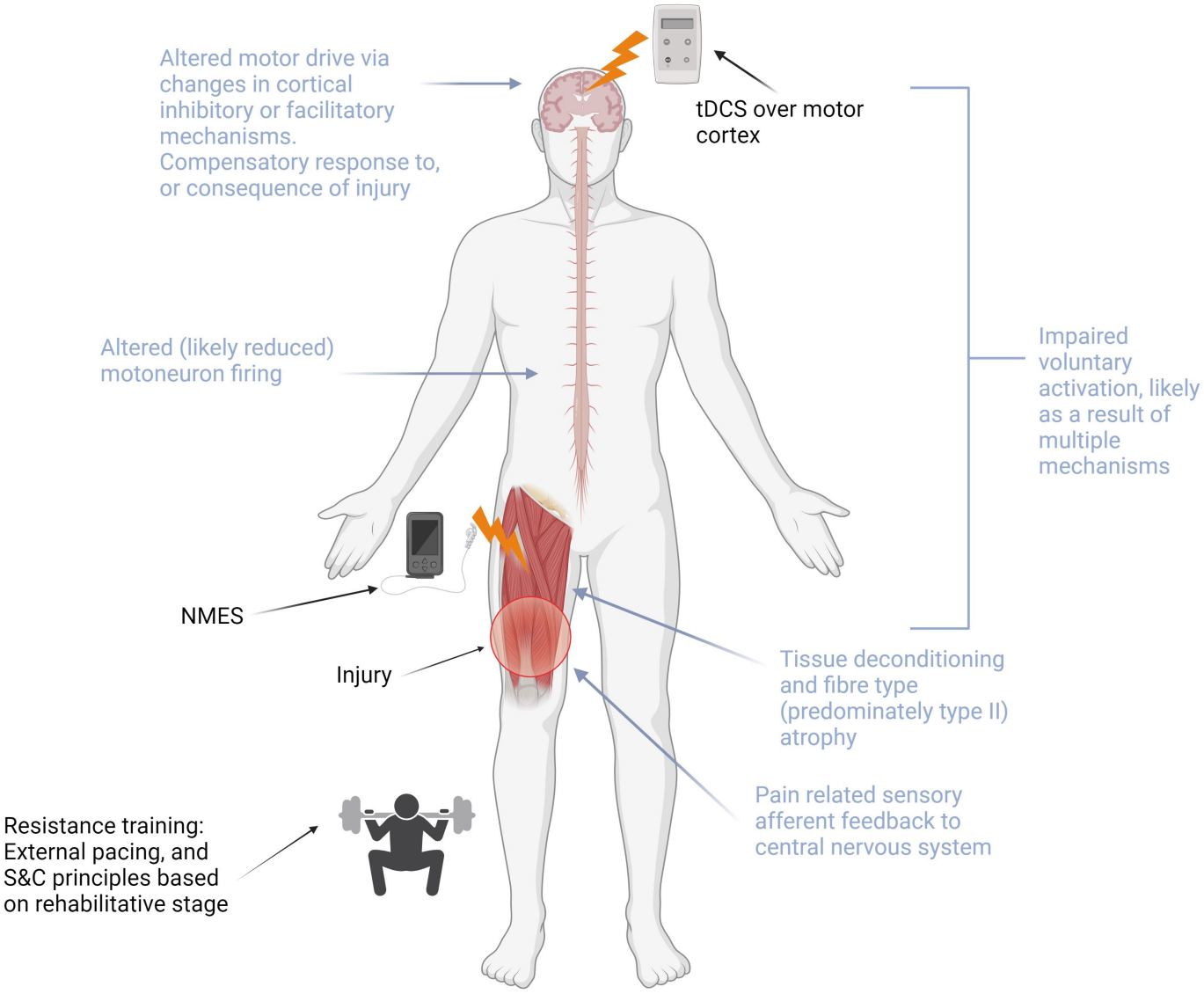

Figure 1 Example of the different mechanisms potentially contributing to suboptimal motor output in injury and the potential role of adjuncts in optimising neuromuscular function. NMES, neuromuscular electrical stimulation; S&C, strength and conditioning; tDCS, transcranial direct current stimulation.

guidelines, and some novel strategies that clinicians can consider to potentially improve the efficacy of musculoskeletal rehabilitation (figure 1).

Considerations for rehabilitative exercise programming and periodisation

Strength and conditioning following injury should include resistance training, which we know can improve motor function and reduce motor inhibition (even in the untrained limb with unilateral training[17] and may be applicable when the injured limb is immobilised). Following this, guidelines include progression and periodisation of training based on the rehabilitative stage of the injured tissue.[18] The duration of each stage and progression criteria will be somewhat individualised and based on the person's goals, external and internal factors that dictate the strength activities and loads employed. In a more generalist training sense, we could consider these as specific blocks/phases (eg, mesocycles typically 2–8 weeks in duration). After the initial 'inflammatory response phase' (sometimes less than 1 week in duration), the fibroblastic repair phase (often ≤8 weeks duration) and then finally the maturation-remodelling phase (8 weeks plus) represent each progressive stage.

Recommended loads used in the fibroblastic repair phase may look similar between athletes of different sports. For example, for both a powerlifter and triathlete, isometric contractions at various joint angles and submaximal dynamic exercises with loads ≤50% of maximum are warranted with some variation in sets and repetition volume. Progression to the maturation-remodelling stage should see a sport specific differentiation towards the required demands (eg, specific loads, energy systems and contraction characteristics). To exemplify, a powerlifter needs to be able to perform multi-joint squat, hinge and

pressing movements under maximal loads, hence the programme should consider what is required to re-develop this ability. In contrast, a sprinter would need to begin implementing contraction speeds more akin to that encountered in their sporting discipline.

Examples of adjunct therapies to support rehabilitation

External pacing

Although contraction speed-specific exercises are necessary during later rehabilitation, external pacing (eg, contraction speed guided by a metronome) can positively alter corticospinal excitability and inhibition in healthy populations,[19] with emerging research in some injured populations (eg, tendinopathy).[20] These changes also result in greater strength improvements compared with self-paced strength training, likely due to both controlled time under tension, and possibly due to improvements in corticospinal excitability.

External pacing can be easily implemented in the clinical setting. For example, you may set a metronome at 60 bpm for leg extension and instruct your athlete to complete 4×8 repetitions at the pace of three seconds concentric and four seconds eccentric. This constrains the movement, adds an additional motor skill component to the task, and engages important parts of the brain and neural networks.

Neuromuscular electrical stimulation

Neuromuscular electrical stimulation involves the direct activation of peripheral muscle tissue. This technique is well established in the rehabilitation of certain populations (eg, following ACL reconstruction)[21] but not routinely used. The direct activation of muscle may facilitate tissue adaptation. However, most stimulation protocols likely only recruit lower threshold units as pain and tolerability become a factor with increasing stimulation intensities. Hence, such an approach may be most warranted in the early phases of rehabilitation where the performance of voluntary contractions is severely compromised or not recommended. However, it seems unlikely that such an approach will have significant impacts on higher threshold motor units/type II fibre and maximal voluntary activation.

Transcranial electrical stimulation

An increasingly popular therapeutic tool to improve treatment outcomes via modulations in cortical excitability is transcranial direct current stimulation (tDCS),[22] or alternatives such as transcranial alternating current stimulation. This intervention applies a low-level constant current, typically focused over the motor cortex for musculoskeletal effects. Hence, as opposed to neuromuscular electrical stimulation, tDCS does target the central nervous system.[23] Hendy *et al* showed that the cross education of strength (increased strength of untrained limb in response to unilateral training of the homologous limb) is greater with tDCS application in healthy people[24 25] and hence, may be a promising tool in musculoskeletal rehabilitation. Preliminary studies have also shown benefits of tDCS in pathological conditions such as knee osteoarthritis.[26 27]

Note, there are other potential techniques not discussed that may have some merit. However, we have put forward several that show the most potential for clinical uptake due to accessibility, feasibility of the technique in a clinical patient setting, and skills required for safe implementation.

CONCLUSION

This viewpoint highlights that while the field is beginning to do better than the simple prescription of 3 sets of 10 repetitions during rehabilitation, there are multiple factors and considerations in advancing rehabilitative exercise prescription. For example, identification of specific mechanisms underpinning altered motor function/activation in various injury conditions could allow targeted interventions to be developed to achieve better outcomes. Apart from the implementation of recommended training principles the exploration of suitable, promising adjunct therapies, particularly those that may facilitate motor plasticity and restore voluntary drive to the muscle should also be considered.

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REFERENCES

1 Todd JS, Shurley JP, Todd TC. Delorme and the science of progressive resistance exercise. *Journal of Strength and Conditioning Research* 2012;26:2913–23.
2 Murphy MC, Travers M, Chivers P, *et al*. Can we really say getting stronger makes your tendon feel better? no current evidence of a relationship between change in Achilles Tendinopathy pain or disability and changes in Triceps Surae structure or function when completing rehabilitation: a systematic review. *Journal of Science and Medicine in Sport* 2023;26:253–60.
3 Murphy M, Travers M, Gibson W, *et al*. The rate of improvement of pain and function in mid-portion Achilles Tendinopathy with loading protocols: a systematic review and longitudinal meta-analysis. *Sports Med* 2018;48:1875–91.
4 Murphy MC, Latella C, Rio EK, *et al*. Does lower-limb osteoarthritis alter motor cortex descending drive and voluntary activation? A systematic review and meta-analysis. *EFORT Open Rev* 2023;8:883–94.

5 Lisee C, Lepley AS, Birchmeier T, *et al*. Quadriceps strength and Volitional activation after anterior Cruciate ligament reconstruction: A systematic review and meta-analysis. *Sports Health* 2019;11:163–79.
6 Zarzycki R, Morton SM, Charalambous CC, *et al*. Athletes after anterior Cruciate ligament reconstruction demonstrate asymmetric Intracortical Facilitation early after surgery. *J Orthop Res* 2021;39:147–53.
7 Zarzycki R, Morton SM, Charalambous CC, *et al*. Examination of Corticospinal and spinal Reflexive excitability during the course of postoperative rehabilitation after anterior Cruciate ligament reconstruction. *J Orthop Sports Phys Ther* 2020;50:516–22.
8 Rio E, Kidgell D, Moseley GL, *et al*. Elevated Corticospinal excitability in Patellar Tendinopathy compared with other anterior knee pain or no pain. *Scand J Med Sci Sports* 2016;26:1072–9.
9 Buhmann R, Trajano GS, Kerr GK, *et al*. Increased short interval Intracortical inhibition in participants with previous hamstring strain injury. *Eur J Appl Physiol* 2022;122:357–69.
10 Gómez-Feria J, Martín-Rodríguez JF, Mir P. Corticospinal adaptations following resistance training and its relationship with strength: a systematic review and multivariate meta-analysis. *Neurosci Biobehav Rev* 2023;152:105289.
11 Orssatto LBR, Blazevich AJ, Trajano GS. Ageing reduces persistent inward current contribution to motor Neurone firing: potential mechanisms and the role of exercise. *The Journal of Physiology* 2023;601:3705–16.
12 Plotkin DL, Roberts MD, Haun CT, *et al*. Muscle fiber type transitions with exercise training: shifting perspectives. *Sports (Basel)* 2021;9:127.
13 Wang Y, Pessin JE. Mechanisms for fiber-type specificity of skeletal muscle atrophy. *Curr Opin Clin Nutr Metab Care* 2013;16:243–50.
14 Sterling M, Jull G, Wright A. The effect of musculoskeletal pain on motor activity and control. *The Journal of Pain* 2001;2:135–45.
15 Taylor JL, Amann M, Duchateau J, *et al*. Neural contributions to muscle fatigue: from the brain to the muscle and back again. *Med Sci Sports Exerc* 2016;48:2294–306.
16 Martinez-Valdes E, Negro F, Arvanitidis M, *et al*. Pain-induced changes in motor unit discharge depend on recruitment threshold and contraction speed. *Journal of Applied Physiology* 2021;131:1260–71.
17 Latella C, Kidgell DJ, Pearce AJ. Reduction in Corticospinal inhibition in the trained and untrained limb following unilateral leg strength training. *Eur J Appl Physiol* 2012;112:3097–107.
18 Haff G, Triplett NT. Essentials of Strength Training and Conditioning4th edn. Champaign, IL: Human Kinetics, 2016.
19 Leung M, Rantalainen T, Teo W-P, *et al*. Motor cortex excitability is not Differentially modulated following skill and strength training. *Neuroscience* 2015;305:99–108.
20 Rio E, Kidgell D, Moseley GL, *et al*. Tendon Neuroplastic training: changing the way we think about tendon rehabilitation: a narrative review. *Br J Sports Med* 2016;50:209–15.
21 Kim K-M, Croy T, Hertel J, *et al*. Effects of neuromuscular electrical stimulation after anterior Cruciate ligament reconstruction on quadriceps strength, function, and patient-oriented outcomes: a systematic review. *J Orthop Sports Phys Ther* 2010;40:383–91.
22 Bornheim S, Thibaut A, Beaudart C, *et al*. Evaluating the effects of tDCS in stroke patients using functional outcomes: a systematic review. *Disabil Rehabil* 2022;44:13–23.
23 Machado S, Jansen P, Almeida V, *et al*. Is tDCS an adjunct Ergogenic resource for improving muscular strength and endurance performance. *Front Psychol* 2019;10:1127.
24 Hendy AM, Kidgell DJ. Anodal-tDCS applied during unilateral strength training increases strength and Corticospinal excitability in the untrained Homologous muscle. *Exp Brain Res* 2014;232:3243–52.
25 Hendy AM, Teo WP, Kidgell DJ. Anodal transcranial direct current stimulation prolongs the cross-education of strength and Corticomotor plasticity. *Med Sci Sports Exerc* 2015;47:1788–97.
26 Chang W-J, Bennell KL, Hodges PW, *et al*. Addition of transcranial direct current stimulation to quadriceps strengthening exercise in knee osteoarthritis: a pilot randomised controlled trial. *PLoS One* 2017;12:e0180328.
27 Tavares DRB, Okazaki JEF, Santana MV de A, *et al*. Motor cortex transcranial direct current stimulation effects on knee osteoarthritis pain in elderly subjects with dysfunctional descending pain inhibitory system: a randomized controlled trial. *Brain Stimul* 2021;14:477–87.