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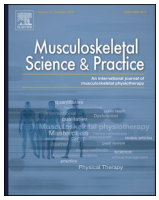

Sensorimotor control of functional joint stability: Scientific concepts, clinical considerations, and the articuloneuromuscular cascade paradigm in peripheral joint injury

Nicholas C. Clark

School of Sport, Rehabilitation, and Exercise Sciences, University of Essex, Wivenhoe Park, Colchester, Essex, CO4 3SQ, UK, United Kingdom

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ABSTRACT

Human movement depends on sensorimotor control. Sensorimotor control refers to central nervous system (CNS) control of joint stability, posture, and movement, all of which are effected via the sensorimotor system. Given the nervous, muscular, and skeletal systems function as an integrated "neuromusculoskeletal system" for the purpose of executing movement, musculoskeletal conditions can result in a cascade of impairments that affect negatively all three systems. The purpose of this article is to revisit concepts in joint stability, sensorimotor control of functional joint stability (FJS), joint instability, and sensorimotor impairments contributing to functional joint instability. This article differs from historical work because it updates previous models of joint injury and joint instability by incorporating more recent research on CNS factors, skeletal muscle factors, and tendon factors. The new 'articuloneuromuscular cascade paradigm' presented here offers a framework for facilitating further investigation into physiological and biomechanical consequences of joint injury and, in turn, how these follow on to affect physical activity (functional) capability. Here, the term 'injury' represents traumatic joint injury with a focus is on peripheral joint injury. Understanding the configuration of the sensorimotor system and the cascade of post-injury sensorimotor impairments is particularly important for clinicians reasoning rational interventions for patients with mechanical instability and functional instability. Concurrently, neurocognitive processing and neurocognitive performance are also addressed relative to feedforward neuromuscular control of FJS. This article offers itself as an educational resource and scientific asset to contribute to the ongoing research and applied practice journey for developing optimal peripheral joint injury rehabilitation strategies.

1. Introduction

Human movement depends on sensorimotor control (Clark et al., 2015a). Sensorimotor control refers to central nervous system (CNS) control of joint stability, posture, and movement, all of which are effected via the sensorimotor system (Clark et al., 2023; Riemann and Lephart, 2002a). Musculoskeletal conditions include diseases (e.g., osteoarthritis) and injuries (e.g., ligament sprain) of the musculoskeletal system, (World Health Organization, 2022) with peripheral joint (e.g., shoulder, knee) conditions being common (Govaerts et al., 2021; Prieto-Gonzá et al., 2021). Given the nervous, muscular, and skeletal systems function as an integrated "neuromusculoskeletal system" for the purpose of executing movement, (Lloyd et al., 2023) musculoskeletal conditions can result in a cascade of impairments that affect all three systems negatively (e.g., Fig. 1; skeletal, 'Articular Soft Tissue Damage'; nervous, 'Altered CNS Processing'; muscular, 'Impaired Muscle Physiology and Mechanics').

Injury control refers to preventing or reducing the severity of injury and includes prevention, acute care, and rehabilitation (Johnston and Rivara, 2003). To develop optimal injury control strategies for a peripheral joint (hereafter, 'joint'), it is necessary to understand how the neuromusculoskeletal system can be affected by joint injury. To this end, the purpose of this article is to revisit the concepts of functional joint stability (FJS) and functional joint instability (FJI) in the context of the sensorimotor system. This article differs from historical work because it updates previous models of joint injury and joint instability (Hurley, 1997; Lephart and Henry, 1996; Lephart et al., 1994; Needle et al., 2017; Stokes and Young, 1984) by incorporating more recent research on CNS factors (e.g., cerebral neuroplasticity), skeletal muscle factors (e.g., contractility), and tendon factors (e.g., material stiffness). Specifically, this article presents a new paradigm (Fig. 1) which offers a framework for facilitating further investigation into physiological and

E-mail address: n.clark@essex.ac.uk.

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biomechanical consequences of joint injury and, in turn, how these follow on to affect physical activity (functional) capability. The term 'injury' is used relative to traumatic versus gradual-onset injury (Olsen et al., 2005; Soligard et al., 2009) (Table 1). It is clinically important to distinguish between the two because the pathoaetiology/pathophysiology of the former differs to the latter; therefore, clinical reasoning for the former will also differ to that for the latter.

Although the term "neuromusculoskeletal system" has been used by others, (Lloyd et al., 2023) the articuloneuromuscular cascade paradigm is structured specifically to recognise the functional relationship between the body systems as being both 'in-series' (skeletal → nervous → muscular; Fig. 1) and 'in-parallel' (e.g., impaired feedforward neuromuscular control, impaired feedback neuromuscular control; Fig. 1). The paradigm is also structured to consider how 'downstream' consequences (e.g., impaired feedforward neuromuscular control) follow 'upstream' factors (e.g., impaired proprioception) (Fig. 1). Because no research study can answer all research questions, (Mabry et al., 2010) and because technology limitations are barriers to answering some research questions in humans, (Clark et al., 2021) this article uses a "systems science" approach to translate multiple sources and levels of evidence into a rational paradigm (Mabry et al., 2010; Clark et al., 2021) (Fig. 1). It is anticipated this article will serve as an educational resource and scientific asset which contributes to the research and applied practice journey to develop optimal joint injury rehabilitation strategies.

Table 1
Injury terminology and definitions.

Injury Term	Definition
Injury	An onset of symptoms (e.g., pain) that restricts physical activity (functional) capability for a period-of-time and/or requires an individual to seek out treatment
Traumatic injury	A sudden onset of symptoms that follows a single known event and where it is logical that tissue damage has more likely than not occurred (e.g., ligament sprain)
Gradual-onset injury	A gradual-onset of symptoms that does not follow a single known event and where it is logical that tissue damage may or may not have occurred (e.g., stress fracture or patellofemoral pain, respectively)

2. Joint stability

2.1. Joint stability

To understand sensorimotor control of joint stability, it is necessary to delineate the concept of joint stability. Joint stability has been defined as the ability of a joint to remain in or return to neutral alignment through the equalization of forces (Riemann and Lephart, 2002a) or the ability of a joint to resist abnormal displacement of the articulating bones (Hall, 1991). The critical concepts are "alignment" and "displacement"; alignment being a straight-line arrangement of objects; (Waite, 2012) displacement being a change in position (Hall, 1991). During human movement, joints move away from neutral alignment cyclically in different planes-of-motion and in different directions (Neumann, 2002; Perry, 1992). The clinically-relevant issue is whether an individual can control the *amount* of joint displacement *away from* neutral alignment without tissue damage occurring (e.g., ligament sprain) and then return the joint *back towards* neutral alignment in preparation for the next movement cycle. Therefore, joint stability is defined here as *the ability of a joint to resist excessive displacement and return towards neutral alignment using internal moments that limit and counter the effects of opposing internal or external moments*. Terms used in this definition are explained in Table 2. Limiting and countering internal moments are also explained in Table 2. Overall, joint stability is a concept which includes both mechanical joint stability and FJS (Riemann and Lephart, 2002a).

2.2. Mechanical joint stability and functional joint stability

Mechanical joint stability results from the non-contractile tissues giving a joint its unique shape and structure (e.g., bone-ends, capsule, ligaments), being termed "static restraints" (Riemann and Lephart, 2002a). The static restraints function to maintain optimal mechanical joint stability (Riemann and Lephart, 2002a). Functional joint stability is

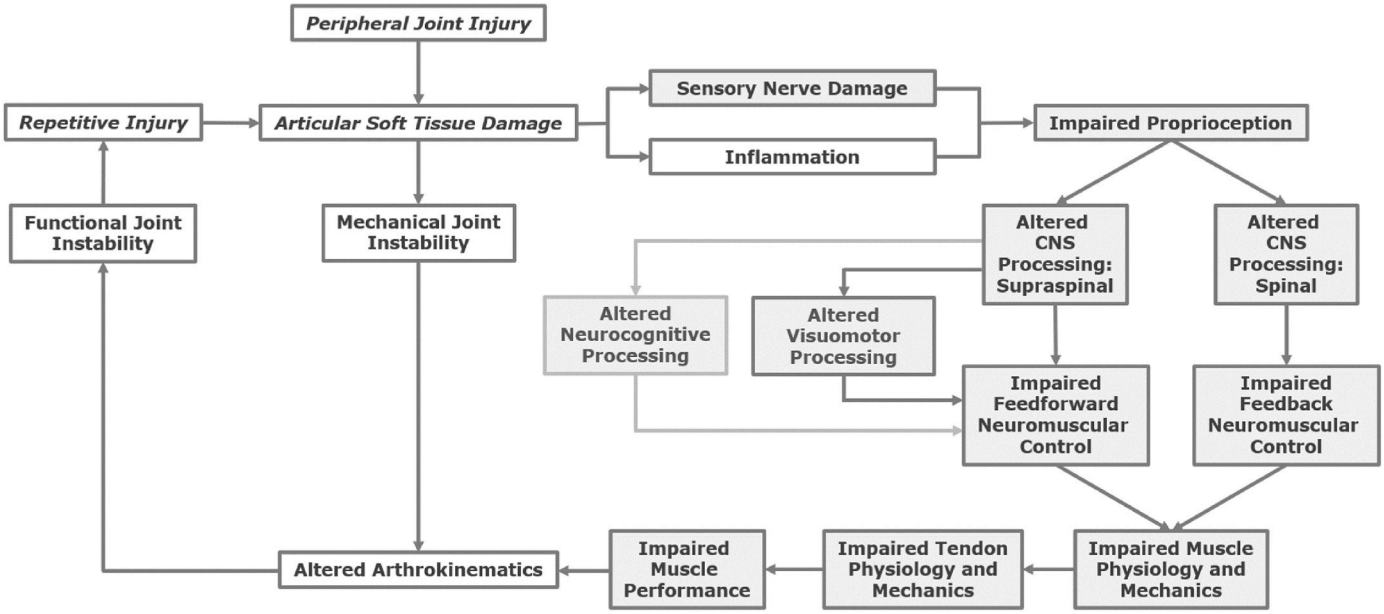

Fig. 1. Articuloneuromuscular cascade paradigm of functional joint instability
Adapted and updated from Lephart and Henry (1996). CNS = central nervous system.
The term 'articuloneuromuscular' represents the functional relationship between the body systems as being in an 'in-series' configuration; skeletal articulations (peripheral joints) → nervous system → muscular system.
First-time peripheral joint injury triggers a cascade of immune system (e.g., inflammation) and sensorimotor system (e.g., impaired proprioception) consequences that can contribute to cycles of functional joint instability and repetitive injury.

Table 2
Joint stability definition terms and explanations.

Term	Explanation
Excessive displacement	The amount of displacement causing tissue damage
Neutral alignment	The typical anatomical reference position for a joint
Internal moments	Forces generated inside the body (e.g., muscle forces) that tend to cause joint rotation
External moments	Forces generated outside the body (e.g., ground reaction forces) that tend to cause joint rotation
Limiting internal moment	Internal moment involving isometric or eccentric muscle actions; these restrain potentially excessive displacement of a joint *away from* its neutral alignment
Countering internal moment	Internal moment involving concentric muscle actions; these overcome potentially excessive displacement of a joint in one direction by moving it in the opposite direction *back towards* a more neutral alignment

the ability to maintain joint stability during functional tasks (e.g., walking, carrying) (Riemann and Lephart, 2002a; Johansson et al., 1991) being the product of mechanical joint stability combined with the action of skeletal muscles that influence joint alignment (Riemann and Lephart, 2002a; Clark, 2001). Muscles that influence joint alignment are termed "dynamic restraints" (Riemann and Lephart, 2002a). When considering FJS, the principal role of dynamic restraints is to "stress--shield" non-contractile tissues from excessive forces that cause injury (Clark et al., 2015a, 2023). In other words, the dynamic restraints operate 'on top of' the static restraints to limit excessive joint displacement and protect non-contractile tissues from loads/deformations (stresses/strains) that elicit tissue damage. Thus, FJS results from a co-ordinated interaction between static and dynamic restraints which is effected via sensorimotor control.

3. Sensorimotor control of functional joint stability

3.1. Sensorimotor control

Sensorimotor control operates on a 'sensory-motor' basis, meaning sensory input to the CNS is required before effective motor output from the CNS can be generated (Clark et al., 2023; Riemann and Lephart, 2002a; Wolpert et al., 2013). Sensorimotor control is achieved via the sensorimotor system, which includes sensory (afferent), processing (CNS), and motor (efferent) components (Clark et al., 2023; Riemann and Lephart, 2002a; Lephart et al., 2000). Sensory components refer to afferent pathways that include the somatosensory pathways (e.g., proprioception, nociception) and vestibular and visual pathways (Clark et al., 2023; Gardner et al., 2013) (Fig. 2). Processing components refer to all levels within the CNS with a role in *sensorimotor transformations*; the transformation of sensory inputs to motor outputs (Clark et al., 2023; Wolpert et al., 2013). The three levels of the CNS involved in sensorimotor transformations (i.e., three levels of sensorimotor control) are the spinal cord, brainstem (medulla/pons/midbrain), and cerebral cortex (Lephart and Henry, 1996; Wolpert et al., 2013) (Fig. 2). Also involved in sensorimotor transformations, and adjacent to the brainstem, is the cerebellum (Prati et al., 2024; Lisberger et al., 2013). Motor components refer to efferent pathways that include the descending tracts which innervate skeletal muscle (Clark et al., 2023; Riemann and Lephart, 2002a; Lephart et al., 2000). To appreciate how sensorimotor control maintains FJS, it is necessary to understand the role fulfilled by each component of the sensorimotor system, beginning with the afferent component of sensorimotor control.

3.2. Afferent pathways

The somatosensory pathways are most important for FJS (Riemann and Lephart, 2002a; Gardner et al., 2013). A somatosensory pathway includes the sensory nerve ending (e.g., mechanoreceptor; Fig. 2), sensory nerve fibre, afferent synapses and interneurons, and ascending tracts (Clark et al., 2015a; Riemann and Lephart, 2002b). Of the somatosensory pathways, the proprioception pathway is critical for sensorimotor control of FJS (Riemann and Lephart, 2002a; Gardner et al., 2013). Proprioception involves the subconscious or conscious sense of joint position (joint position sense), joint movement (kinaesthesia), and force (force sense) (Clark et al., 2023; Riemann and Lephart, 2002a; Kandel et al., 2013). Proprioception results from mechanoreceptor stimulation in skeletal (e.g., ligament) and muscle tissues (Gardner et al., 2013). A mechanoreceptor is a specialised sensory nerve ending sensitive to tissue mechanical stimuli (e.g., tension, compression). (Gardner et al., 2013; Lephart et al., 1997).

In static restraints (e.g., ligaments), mechanoreceptors include Ruffini, Pacinian, and Golgi endings; (Kennedy et al., 1982; Michelson and Hutchins, 1995; Moraes et al., 2011) these function as transducers that convert a non-contractile tissue's mechanical stimuli into electrical signals for transmission to the CNS (Gardner et al., 2013). The static restraints, therefore, fulfil neurological as well as mechanical functions (Clark et al., 2015a; Kennedy et al., 1982). In dynamic restraints (i.e., muscle-tendon units), mechanoreceptors include muscle spindles and Golgi tendon organs; (Gardner et al., 2013) these also function as transducers and transmit electrical signals to the CNS regarding muscle-tendon unit length, velocity of lengthening, and tension (Gardner et al., 2013). All joint and muscle-tendon unit

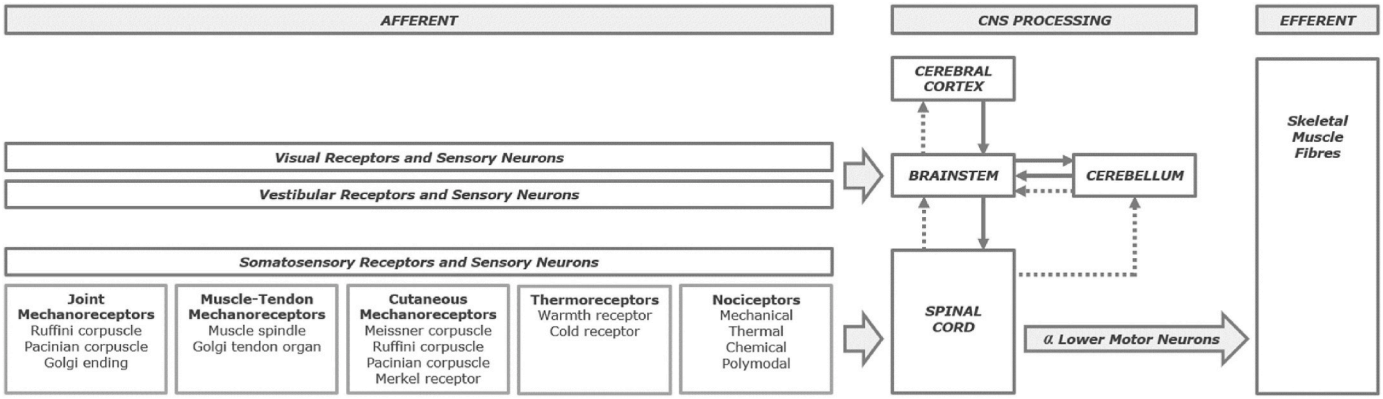

Fig. 2. Simplified configuration of the sensorimotor system and three levels of sensorimotor control
CNS = central nervous system, α = alpha
The three levels of sensorimotor control and CNS processing are the spinal cord, brainstem, and cerebral cortex.
Thin dotted arrows represent the ascending tracts.
Thin solid arrows represent the descending tracts.

mechanoreceptor information is transmitted to the CNS via first-order afferent neurons which collectively form different elements of the ascending tracts (Gardner et al., 2013; Prati et al., 2024; Lisberger et al., 2013; Tan et al., 2023) (Table 3).

3.3. Central nervous system processing

Central nervous system processing refers to sensorimotor transformations occurring at different levels of the CNS. At the spinal cord level, joint mechanoreceptor neurons make monosynaptic and polysynaptic connections with cell bodies of alpha (α) and gamma (γ) lower motor-neurons (LMN) innervating extrafusal (skeletal muscle) and intrafusal (muscle spindle) muscle fibres, respectively (Gardner et al., 2013; Johansson et al., 1990; Sjölander et al., 1994). Of clinical interest is that mechanical stimulation of capsuloligamentous structures elicits subconscious (reflex) electrical activity in periarticular skeletal muscle fibres, (Mobargha et al., 2019; Wallace et al., 1997; Seitz et al., 2021) which enhances stability of the underlying joint. Also of clinical interest is that ligament mechanoreceptor neurons input to γ-LMNs, thereby influencing muscle spindle sensitivity and the stiffness of skeletal muscle fibres, (Johansson et al., 1990; Sjölander et al., 1994) which again enhances the stability of the underlying joint. Muscle mechanoreceptors make monosynaptic and polysynaptic connections with α-LMNs of homonymous and antagonist muscles (Pearson et al., 2013). Such neurocircuitry illustrates how proprioception is employed at the spinal cord level in both joint-muscle and muscle-muscle reflexes.

At brainstem and cerebellum level, proprioceptive information transmitted by ascending tracts is received predominantly by the ipsilateral spinocerebellum (Table 3), from where proprioceptive information is transmitted to the contralateral premotor and primary motor cortices, midbrain, and thalamus (Lisberger et al., 2013; Tan et al., 2023; Haines and Dietrichs, 2012). Proprioception is employed at brainstem and cerebellum level for regulating postural stability, gait, and motor learning (Lisberger et al., 2013; Haines and Dietrichs, 2012).

At cerebral cortex level, proprioceptive information transmitted by ascending tracts is received by the contralateral primary somatosensory cortex (Table 3), from where proprioceptive information is then transmitted via transcortical axons to the ipsilateral somatosensory association area and supplementary motor area, along with ipsilateral premotor and primary motor cortices (Wolpert et al., 2013; Gardner et al., 2013; Tan et al., 2023). Proprioception is employed at cerebral cortex level for voluntary movement and motor learning (Wolpert et al., 2013). Electroencephalography (Roland, 1978; Tamá et al., 2018; Wheaton et al., 2007) and functional magnetic resonance imaging (Hollnagel et al., 2011; Grooms et al., 2017; Khorrami et al., 2011; Shitara et al., 2022) studies have demonstrated that cerebral cortex somatosensory areas are highly active simultaneously with other brain regions during voluntary movement.

The sensory-motor configuration of the CNS illustrates how proprioception derived from joint and muscle-tendon mechanoreceptors is transformed to modify motor output at all three levels of the CNS; in other words, sensory information from afferent elements of the CNS consistently 'feeds' into efferent elements of the CNS to generate motor commands. Although three levels of the CNS are described separately/hierarchically in-series (Fig. 2), all operate simultaneously in-parallel to transform sensory information into motor commands transmitted to skeletal muscle via the efferent pathways (Riemann and Lephart, 2002a; Wolpert et al., 2013).

Table 3
Principal ascending tracts in sensorimotor control of functional joint stability.

Tract	Origin[a]	Decussation Site	Termination[b]
Cerebellar tracts			
Posterior/dorsal spinocerebellar tract	• Thoracic lamina V, VI, VII (Clarke's nucleus)	• None	• Ipsilateral spinocerebellum
	• Lumbar and sacral lamina V, VI, VII (Clarke's nucleus)	• None	• Ipsilateral spinocerebellum
Anterior/ventral spinocerebellar tract	• Lumbosacral lamina V, VII (Clarke's nucleus)	• Primary: anterior/ventral white commissure • Secondary: pons	• Primary: contralateral spinocerebellum • Secondary: ipsilateral spinocerebellum
Rostral spinocerebellar tract	• Cervical and thoracic lamina VI, VII (Clarke's nucleus)	• None	• Ipsilateral spinocerebellum
Cuneocerebellar tract	• Cervical lamina I, V, VI, VII	• None	• Ipsilateral spinocerebellum
Dorsal column-medial lemniscal system			
Gracile fasciculus tract	• Dorsal root ganglion (thoracic, lumbar, and sacral first-order afferent neurons)	• Medulla: gracile nucleus	• Contralateral primary somatosensory cortex
Cuneate fasciculus tract	• Dorsal root ganglion (cervical and thoracic first-order afferent neurons)	• Medulla: cuneate nucleus	• Contralateral primary somatosensory cortex

[a] Location of tract cell body.
[b] Location of tract axon terminals which synapse with cell bodies of other cerebellar or cerebral neurons.

3.4. Efferent pathways

An efferent pathway includes upper motor-neurons, efferent synapses and interneurons, descending tracts, α or γ LMNs, and motor end plates (Clark et al., 2015a, 2023; Riemann and Lephart, 2002a, 2002b; Lephart et al., 2000). Efferent pathways are the effectors for neuromuscular control (NMC) of FJS. Neuromuscular control involves activation of the dynamic restraints in preparation for or reaction to joint loading and motion, and includes both activation and force generation elements (Clark et al., 2023; Riemann and Lephart, 2002a, 2002b). Neuromuscular control manifests as the coordinated dampening of joint loads, restraint of excessive joint motion, and facilitation of FJS (Riemann and Lephart, 2002a; Lephart et al., 1997).

At cerebral cortex level, the principal efferent pathway includes upper motor-neurons forming different tracts contributing to pyramidal tracts (Table 4); these receive input from the ipsilateral supplementary motor area, ipsilateral premotor cortex, and contralateral spinocerebellar cortex (Amaral et al., 2013; Nathan et al., 1990;Nyberg- et al., 1963). At brainstem level, the principal efferent pathways include motor-neurons forming different tracts contributing to extrapyramidal tracts (de Oliveira-Souza, 2012; Fisher et al., 2021; Yang et al., 2011) (Table 4). The reticulospinal tracts receive vast input from the primary somatosensory cortex, supplementary motor area, and primary motor cortex, along with vestibular, auditory, and visual nuclei (Fisher et al., 2021; Baker, 2011; Peterson, 1979). The rubrospinal tract receives its input from the ipsilateral premotor cortex, primary motor cortex, and thalamus, along with ipsilateral and contralateral spinocerebellar cortices (Lisberger et al., 2013; Yang et al., 2011; Milardi et al., 2016). At spinal cord level, the efferent pathways include LMNs innervating skeletal muscle directly; specifically, α-LMNs and γ-LMNs (Clark et al., 2023; Johansson et al., 1991; Pearson et al., 2013). Because α-LMNs are stimulated by multiple inputs from afferent pathways (e.g., joint mechanoreceptors) and efferent pathways (e.g., reticulospinal tract) that include both excitatory and inhibitory pre-synaptic interneurons

Table 4
Principal descending tracts in sensorimotor control of functional joint stability.

Tract	Origin[a]	Decussation Site	Termination[b]
Pyramidal tracts			
Crossed lateral corticospinal tract	• Primary motor cortex, premotor cortex, supplementary motor area	• 75–90% of all corticospinal tract neurons: medulla • Pons	• Contralateral cervical, thoracic and lumbar lamina VIII (interneurons), IX (α motor-neurons) • Contralateral cerebrocerebellum
Uncrossed anterior corticospinal tract	• Primary motor cortex, premotor cortex, supplementary motor area	• 10–25% of all corticospinal tract neurons: none	• Ipsilateral cervical and thoracic lamina VIII (interneurons), IX (α motor-neurons)
Crossed anterior corticospinal tract	• Primary motor cortex, premotor cortex, supplementary motor area	• 10–25% of all corticospinal tract neurons: anterior/ventral white commissure	• Contralateral cervical and thoracic lamina VIII (interneurons), IX (α motor-neurons)
Extrapyramidal tracts			
Lateral reticulospinal tract	• Reticular formation (medullary area)	• None	• Ipsilateral cervical, thoracic, and lumbar lamina VIII (interneurons), IX (α motor-neurons, γ motor-neurons)
Medial reticulospinal tract	• Reticular formation (pontine area)	• None	• Ipsilateral cervical, thoracic, and lumbar lamina VIII (interneurons), IX (α motor-neurons, γ motor-neurons)
Rubrospinal tract	• Red nucleus (midbrain area)	• Anterior/ventral tegmental decussation	• Contralateral cervical and thoracic lamina V (interneurons), VI (interneurons), VIII (interneurons), IX (α motor-neurons, γ motor-neurons)

[a] Location of tract cell body.
[b] Location of tract axon terminals which synapse with cell bodies of lower motor-neuron pre-synaptic interneurons, cell bodies of alpha (α) motor-neurons, or cell bodies of gamma (γ) motor-neurons.

(Clark et al., 2023; Johansson et al., 1991; Pearson et al., 2013; Nathan et al., 1990), α-LMNs have been described as the "final common pathway" by which multiple converging inputs are filtered to culminate in skeletal muscle activation and force generation (Clark et al., 2023; Sherrington, 1906; Enoka and Farina, 2021).

Skeletal muscle activation and force generation occurs in two contexts; feedforward and feedback (Riemann and Lephart, 2002a; Wolpert et al., 2013; Pearson et al., 2013; Saper et al., 2000) (Table 5). Feedforward NMC increases muscle stiffness resulting in greater sensitivity for and reaction to unanticipated joint loading/motion (Wolpert et al., 2013; Lephart et al., 2000). Muscle stiffness refers to the extent to which a muscle *resists* elongation and is represented by a ratio of a change in tensile load to a change in muscle length (Δtensile load ÷ Δmuscle length); (Blackburn et al., 2009) the greater the tensile load required to elongate a muscle, the stiffer the muscle. Thus, feedforward NMC involves preparatory stimulation of upper motor-neurons and LMNs by supraspinal centres to increase skeletal muscle stiffness and, in turn, increase stability of the underlying joint (Wolpert et al., 2013; Lephart et al., 1997; Kalaska et al., 2013). Feedback NMC involves reactive stimulation of α-LMNs by mechanoreceptor neurons or interneurons in the spinal cord and reflex activation of skeletal muscle for prompt recovery of FJS following unanticipated joint perturbation (Riemann and Lephart, 2002a; Johansson et al., 1991; Pearson et al., 2013). Although feedforward and feedback NMC have been described separately, they maintain FJS by operating concurrently in-parallel for the co-ordinated activation of a skeletal muscle-tendon unit (Wolpert et al., 2013; Kalaska et al., 2013).

Table 5
Feedforward and feedback neuromuscular control definitions and context.

Type	Definition	Context	Relationship to Sensory Information
Feedforward neuromuscular control	Preparatory activation (preactivity) of and force generation by the dynamic restraints	Before the onset of afferent stimuli signalling joint loading or motion (e.g., *before* foot contact with the ground during running, landing, etc.)	Proprioceptive information from previous experience is used to modify feedforward motor programmes stored in cerebellar and cerebral cortices
Feedback neuromuscular control	Reactive activation (reactivity) of and force generation by the dynamic restraints	After the onset of afferent stimuli signalling joint loading or motion (e.g., *after* foot contact with the ground during running, landing, etc.)	Proprioceptive information enters the central nervous system immediately before α and γ lower motor-neurons are stimulated

α = alpha; γ = gamma.

3.5. Skeletal muscle-tendon unit

Skeletal muscle is the penultimate element in the sensorimotor system. Following activation (electrical stimulation) by α-LMNs, skeletal muscle is a molecular motor that converts chemical energy to mechanical energy (Mukund and Subramaniam, 2020). After skeletal muscle has been stimulated by α-LMNs, the electromechanical delay (EMD) and rate-of-force development (RFD) are important mechanisms for timely force generation. The EMD represents the timeframe between the onset of muscle activity and the generation of force, being the product of complex physiological events (e.g., intramuscular action potential propagation, series-elastic-component elongation) (Winter and Brookes, 1991; Yavuz et al., 2010). The RFD represents the rate-of-rise in (i.e., speed of) muscle force development, also being the product of complex physiological events (e.g., rate coding, muscle-fibre type) (Aagaard et al., 2002; Maffiuletti et al., 2016). Together, EMD and RFD combine to influence muscle stiffness. Because muscle stiffness increases proportionally with muscle activation, the stiffer a muscle, the stiffer and more stable the underlying joint (McQuade and Murthi, 2004; Mirbagheri et al., 2000; Olmstead et al., 1986; Vigotsky et al., 2020). The EMD and RFD are essential in contexts involving feedback NMC where rapid force generation and development of muscle stiffness is required to restrain potentially excessive joint displacement, thereby maintaining FJS and preventing/limiting non-contractile tissue damage. Further, frequent and sufficient activation of skeletal muscle is essential to maintain optimal muscle tissue composition and quantity (Eberstein and Ebertstein, 1996; Rochester et al., 1995a, 1995b). For skeletal muscle to operate effectively as a dynamic restraint for FJS, frequent activation by α-LMNs and more upstream elements of the efferent pathways is critical.

Tendon is the last element in the sensorimotor system. Tendon is a multi-unit hierarchical collagen structure that transmits forces generated by skeletal muscle to bone (Nakamichi and Asahara, 2024). Frequent and sufficient loading of tendon tissue is necessary to maintain optimal tissue composition and mechanical properties (e.g., structural stiffness, material stiffness) (Lavagnino et al., 2015). Structural stiffness

refers to the relationship between load and deformation in a structure *without* consideration for its dimensions (e.g., cross-sectional area) (Whiting and Zernicke, 2008). Material stiffness refers to the relationship between load and deformation in a structure *with* consideration for its dimensions; here, load and deformation then become stress and strain, respectively (Whiting and Zernicke, 2008). Material stiffness *is measured from the elastic region of a stress-strain curve* and, subsequently, is commonly referred to as the 'modulus of elasticity' or the 'elastic modulus' (i.e., Young's modulus) (Neumann, 2002; Whiting and Zernicke, 2008; Norkin and Levangie, 1992). Although the terms 'elasticity' and 'elastic' are used, the ability of a material to return/recoil to its original dimensions after a stress is removed is *not* what is measured; rather, the modulus of elasticity and elastic modulus are still representations of material stiffness and the extent to which a material *resists* strain (e.g., elongation) (Neumann, 2002; Whiting and Zernicke, 2008; Norkin and Levangie, 1992). Material stiffness gives greater insight into the mechanical properties of a tissue than does structural stiffness (Whiting and Zernicke, 2008). Optimal tendon material stiffness is critical for transmitting as much force as possible from skeletal muscle to bone, minimising the amount of force lost to elongation by taking up tendon tissue crimp/slack/strain prior to sufficient force propagating to the bone to influence joint motion (Bojsen-Mø et al., 2005; Quinlan et al., 2018). For tendon to operate effectively *with* skeletal muscle as a dynamic restraint for FJS, frequent tensile loading *secondary to* skeletal muscle activation is important. Frequent and sufficient activation and tensile loading of a muscle-tendon unit contributes to maintaining skeletal muscle performance.

3.6. Muscle performance

Muscle strength is defined as the ability of a muscle to produce force (Clark, 2001). There are three types of muscle action (Cavanagh, 1988) and five types of muscle strength (Dick, 2014; Kent, 2006) (Table 6) yielding multiple combinations by which a muscle can produce force (e.g., isometric absolute strength, eccentric relative strength). As such, "muscle performance" is preferred as a term for describing skeletal muscle function broadly in injury control and sensorimotor control (Mayhew et al., 1985; Sapega, 1990; Riemann et al., 2002). When describing muscle performance, different constructs can be used (e.g., manual muscle test grade, one repetition maximum, peak torque, time-to-peak-torque) (Clark, 2001; Clark et al., 2019, 2022; Lee et al., 2023). To this end, both EMD and RFD can also be used as descriptors of muscle performance. Because skeletal muscle is a dynamic restraint that stress-shields joints from excessive forces that cause injury, (Clark et al., 2015a, 2023) and muscle-tendon units are the ultimate effectors of FJS, measurement of muscle performance is fundamental when considering joint stability and instability in joint injury control (Clark, 2001; Mayhew et al., 1985; Riemann et al., 2002).

Table 6
Muscle performance, muscle action, and muscle strength terms and definitions.

Term	Definition
Muscle performance	The ability of a muscle or muscle group to perform a function using an isometric, concentric, or eccentric muscle action, at a specific joint angle or in a specific range-of-motion, at a specific velocity-of-motion, while expressing a specific type of muscle strength
Isometric muscle action	A muscle action where there is no or negligible change in muscle length (synonymous with static muscle action)
Anisometric muscle action	A muscle action where there is a change in muscle length (synonymous with dynamic/isotonic muscle action)
Concentric muscle action	A muscle action where the muscle shortens
Eccentric muscle action	A muscle action where the muscle lengthens
Maximum muscle strength	Force generated in a single maximum voluntary muscle action (MVMA)
Absolute muscle strength	Maximum force generated without consideration for bodymass or muscle size (synonymous with maximum muscle strength)
Relative muscle strength	Maximum force generated with consideration for (relative to) bodymass or muscle size
Elastic muscle strength	Force generated at high velocity of anisometric muscle action
Muscle strength endurance	Force generated for sustained (isometric) or repeated (anisometric) muscle actions against a sub-maximal resistance while resisting fatigue

4. Joint instability

Joint instability includes mechanical joint instability (MJI) and FJI. Mechanical joint instability is evident when joint physiological passive range-of-motion (e.g., extension, external rotation) and/or joint laxity special tests (e.g., talar tilt, elbow valgus) are performed and post-injury "pathological laxity" (i.e., abnormal looseness) (Hertel, 2002; Magee, 2014) or "hyperlaxity" (Rupp et al., 2023) is observed relative to the contralateral side and/or population reference values. Such laxity indicates substantial tissue damage to the static restraints. Mechanical joint instability can feed into FJI due to grossly/subtly altered arthrokinematics (Fig. 1). (Lephart and Henry, 1996; Lephart et al., 1994; Swanik et al., 1997)

Functional joint instability refers to physical activity (functional) limitations due to post-injury symptoms including pain and/or a sensation of joint 'weakness', and signs including sudden episodes of a joint 'giving way' (i.e., collapsing) during movement (Magee, 2014; Freeman, 1965). Repeated episodes of FJI can feed into repetitive injury and articular soft tissue damage (Lephart and Henry, 1996; Lephart et al., 1994; Basciani et al., 2024) (Fig. 1). It is possible to reacquire FJS even though MJI persists (Thoma et al., 2019; Rosen et al., 2014; Woodmass et al., 2019). Conversely, it is also possible for FJI to persist even after MJI has been resolved via surgery (Thoma et al., 2019; Bashaireh et al., 2024; Xu et al., 2022). Therefore, to develop optimal joint injury control strategies for both the former and latter scenarios, it is necessary to understand sensorimotor impairments that can contribute to FJI.

5. Sensorimotor impairments contributing to functional joint instability

An "impairment" is an abnormality in or loss of body structure or function that contributes to reduced physical activity capability (World Health Organization, 2002). Skeletal and sensorimotor impairments manifest consistently after joint injury (Clark et al., 2015a, 2015b, 2021; Roijezon et al., 2015). Given the in-series relationships between the skeletal, nervous, and muscular systems, such impairments can cascade from one to another (Fig. 1).

5.1. Sensory nerve damage, inflammation, and impaired proprioception

Joint injury results in tissue damage; this results in sensory nerve damage due to destruction of non-contractile tissue mechanoreceptor nerve endings and their associated afferent nerve fibres (Dhillon et al., 2010; Bali et al., 2012). Destruction of non-contractile tissue is followed frequently by impaired proprioception. Joint position sense, (Boyle and Negus, 1998; Kim et al., 2014; Zuckerman et al., 2003) kinaesthesia, (Zuckerman et al., 2003; Borsa et al., 1997; Safran et al., 1999) and force sense (Docherty and Arnold, 2008; Xiao et al., 2024; Héroux and Tremblay, 2005) can all be reduced after joint injury. Inflammation is a non-specific defensive response of tissues to injury (Kent, 2006). Inflammation after joint injury involves increases in intra-articular and peri-articular pro-inflammatory cytokines (e.g., Tumor Necrosis Factor-α, Interleukin-1β) (Adams et al., 2015; Irie et al., 2003). Such cytokines alter neuronal excitability and synaptic transmission culminating in nerve cell dysfunction and abnormal neural network excitability (Vezzani and Viviani, 2015). Inflammation also includes pain and

swelling (Kent, 2006). Both joint pain and swelling (effusion) can impair proprioception. Studies report that pain can reduce kinaesthesia (Matre et al., 2002; Sole et al., 2015) and effusion can reduce joint position sense (Baxendale and Ferrell, 1987; Cho et al., 2011; Guido et al., 1997). Loss of afferent signals to the CNS is termed "deafferentation" (Latash, 2012). Following joint injury, this is termed "articular deafferentation" (Freeman et al., 1965). Deafferentation and loss of proprioception drive downstream CNS alterations (Casseb et al., 2019; Courtney et al., 2005; Lund et al., 1994; Pons et al., 1991).

5.2. Altered CNS processing: supraspinal and impaired feedforward neuromuscular control

Neuroplasticity is the ability of the nervous system to respond to increases/decreases in external/internal stimuli by reorganising its structure, function, and synaptic connections (Casseb et al., 2019). Following joint injury, electroencephalography (Baumeister et al., 2007, 2011). Zhang et al., 2022) and functional magnetic resonance imaging (Grooms et al., 2017; Shitara et al., 2022; Xie et al., 2022; Kapreli et al., 2009) demonstrate reorganised activation patterns and structure of cerebral sensorimotor areas. Following joint injury, functional magnetic resonance imaging has also demonstrated reorganised activation patterns in cerebellar sensorimotor areas (Grooms et al., 2017; Shitara et al., 2022; Xue et al., 2021). Across these studies, findings consistently point to compensations involving increased activation of brain areas dedicated to visuomotor and neurocognitive (attention, visual working-memory) processing. Others report that sensorimotor performance (e.g., single-leg balance) degrades substantially more for those with joint injury compared to those without joint injury when vision is removed, (Mattacola et al., 2002; Okuda et al., 2005; Kim, 2020) indicating 'sensory reweighting' (Friedrich et al., 2008) from proprioception-motor (down-weighting) to visual-motor (up-weighting) movement control. For neurocognitive processing (i.e., neurocognitive performance), others also report that an eyes-open single-leg balance task degrades substantially more for those with joint injury compared to those without joint injury when a visual working-memory challenge is superimposed on the sensorimotor challenge of the single-leg balance task itself (Monfort et al., 2022). These findings suggest that for those with joint injury and upstream impairments of proprioception, there follows a downstream greater reliance on vision versus proprioception for the control of FJS where such control also requires higher levels of concentration and neurocognitive processing (Fig. 1). Using transcranial magnetic stimulation, researchers report altered excitability of the primary motor cortex after joint injury (Héroux and Tremblay, 2006; McLeod et al., 2015). With electromyography, researchers also report altered feedforward muscle activation (preactivity) patterns after joint injury (Caulfield et al., 2004; De Mont et al., 1999; Feger et al., 2015). Using peripheral electrical stimulation, others report an impaired ability to voluntarily fully activate an injured joint's prime movers (Courtney et al., 2005; Williams et al., 2005; Chmielewski et al., 2004). Together, studies consistently observe a variety of alterations representing impaired feedforward NMC and a decreased ability to protect an already injured joint *before* recurrent and anticipated (planned) loading/motion occurs.

5.3. Altered CNS processing: spinal and impaired feedback neuromuscular control

The term "arthrogenic muscle weakness" has existed for >40 years, (Stokes and Young, 1984) more recently being termed "arthrogenic muscle inhibition" (Hurley et al., 1994; Hopkins and Ingersoll, 2000). Arthrogenic muscle inhibition refers to spinal reflex inhibition (i.e., decreased excitability) of α-LMNs for a joint due to aberrant sensory traffic from that joint to the spinal cord (Hurley, 1997; Stokes and Young, 1984; Hurley et al., 1994). Aberrant sensory traffic from an injured joint occurs due to non-contractile tissue damage/pain/effusion (Hart et al., 2014; Needle et al., 2013; Grigg et al., 1978). In such instances, aberrant sensory traffic induces pre-synaptic and post-synaptic spinal reflex inhibition of α-LMNs (Hopkins and Ingersoll, 2000; McVey et al., 2005; Palmieri et al., 2005). Joint effusion is particularly potent at inducing aberrant sensory traffic from capsular afferents (Spencer et al., 1984; Iles et al., 1990) and reducing the excitability of that joint's α-LMNs (Kennedy et al., 1982; Spencer et al., 1984; Hopkins et al., 2001). Joint-muscle reflexes are also consistently altered after joint injury. With electromyography, researchers report slower feedback muscle activation (reactivity) patterns after joint injury (Beard et al., 2000; Myers et al., 2004; Rein et al., 2021; Wojtys and Huston, 1994). Also with electromyography, others report a lower magnitude of feedback muscle activation patterns after joint injury (Palmieri-Smith et al., 2009; Rudolph and Snyder-Mackler, 2004). Collectively, studies consistently observe a variety of alterations representing impaired feedback NMC and a decreased ability to protect an already injured joint *after* recurrent and unanticipated (unplanned) loading/motion has occurred.

5.4. Impaired muscle physiology and mechanics

Muscle physiology and mechanics alter after joint injury, disuse, and immobilisation (Clark et al., 2021). Following joint injury, muscle biopsy and histology studies report intramuscular physiological and structural changes, including decreased muscle-fibre type-IIA cross-sectional area, decreased muscle-fibre pennation angle, and a proliferation of collagen and expansion of the extracellular matrix (Noehren et al., 2016). Concurrent are type-I and type-II muscle-fibre denervation, a decrease in numbers of satellite cells, and increase in numbers of fibroblasts (Fry et al., 2017). Muscle contractility is also decreased, (Gumucio et al., 2018) as represented by force generating and velocity-of-shortening capacity for biopsied muscle fibres (Gumucio et al., 2018; Toth et al., 2020). Following unloading of muscle due to disuse/immobilisation, as is the case following joint injury, myofibril protein synthesis reduces (de Boer et al., 2007a; Nunes et al., 2022). For passive and active muscle stiffness, this can be reduced across a range of different joint injuries (Olds et al., 2011; Swanik et al., 2004). Together, these works observe an intramuscular progression away from contractile proteins toward more abundant non-contractile connective tissue, a corresponding decrease in rapid force generating capabilities of muscle fibres, and potential for a decrease in muscle stiffness. Such alterations represent muscle-level impairments that can contribute to impaired feedforward and feedback NMC of an injured joint.

5.5. Impaired tendon physiology and mechanics

Tendon physiology and mechanical properties alter secondary to disuse/immobilisation of a joint. Following unloading of tendons due to disuse/immobilisation, as is the case following joint injury, tendon collagen degrades and synthesis reduces (de Boer et al., 2007a; Christensen et al., 2008; Dideriksen et al., 2017). Also following short-term disuse/immobilisation, both tendon structural stiffness and material stiffness (Young's modulus) decrease (de Boer et al., 2007b; Couppé et al., 2012; Loitz et al., 1989). Collectively, studies indicate that tendons become less stiff due to increased elongation for a given stress, making tendons less effective for the timely propagation of muscle force to bone. Therefore, this in turn can also contribute to impaired feedforward and feedback NMC and the timely acquisition or re-acquisition of FJS, respectively.

5.6. Impaired muscle performance

Impaired muscle performance after joint injury is evident using a variety of muscle action-muscle strength configurations, demonstrating consistently decreases in isometric maximum strength, (Gumucio et al., 2018; Perry et al., 2015) isometric relative strength, (Noehren et al.,

2016; Perry et al., 2015) isokinetic-concentric maximum strength, (Gumucio et al., 2018; Chang et al., 2024) isokinetic-concentric relative strength, (Wenning et al., 2023; Lee et al., 2020) isokinetic-eccentric maximum strength, (MacLean et al., 1999) and isokinetic-eccentric relative strength (David et al., 2013). After joint injury, the EMD can be increased (Flevas et al., 2017) and RFD can be decreased (Lee et al., 2023; Qiu et al., 2024) both of which are negative adaptations if the rapid development of muscle force and stiffness is desired to limit potentially excessive sudden joint displacements. The RFD is characterised under isometric/static conditions (Aagaard et al., 2002; Maffiuletti et al., 2016; Lee et al., 2023; Qiu et al., 2024). The time-to-peak-torque is characterised under isokinetic/dynamic conditions (Clark et al., 2022). Because both RFD and time-to-peak-torque are both representations of how rapidly muscle can generate force, time-to-peak-torque can be viewed as a dynamic analogue of the static RFD. After joint injury, isokinetic-concentric time-to-peak-torque has been reported as becoming prolonged, (Lee et al., 2018; Wojtys and Huston, 2000) indicating that concentric muscle force generation takes longer periods-of-time. Together, studies indicate that muscle performance is impaired in a way that negatively affects peak force and reactive force generating capabilities, both of which can also contribute to impaired feedforward and feedback NMC of an injured joint, respectively.

6. Summary

This article revisited concepts in joint stability, sensorimotor control of FJS, joint instability, and sensorimotor impairments contributing to FJI. In doing so, this article presented a new paradigm which includes factors not previously combined in one model of joint injury and joint instability (Fig. 1). Although this paradigm is considered primarily from a sensorimotor control perspective, it is clinically-important to recognise that in real-world human function neurocognitive processing is also important in NMC of FJS. This paradigm is also considered primarily from a post-injury impairment perspective. Rational interventions cannot be planned unless the underlying pathoaetiology/pathophysiology of an injury and its subsequent impairments are understood. Understanding the configuration of the sensorimotor system and the cascade of post-injury sensorimotor impairments is particularly important for clinicians reasoning appropriate interventions for patients with MJI and/or FJI. As such, it is useful to mention that the downstream flow from 'articular soft tissue damage' to 'mechanical joint instability' (Fig. 1) can be modified by reparative/reconstructive surgery, and the downstream flow from 'sensory nerve damage' and 'inflammation' to 'impaired proprioception' (Fig. 1) can be modified with physical rehabilitation (Myers and Lephart, 2002). It is also important to mention that concurrent with the sensorimotor and neurocognitive impairments described here, post-injury disuse/immobilisation can compound clinical problems due to negative adaptations in the static and dynamic restraints following reduced relative loading of body tissues (Clark et al., 2021). Skeletal, (Clark et al., 2015a, 2015b; Clark, 2015) sensorimotor, (Clark et al., 2010, 2015a, 2015b; Swanik et al., 1997; Norte et al., 2021) and neurocognitive (Grooms et al., 2015) rehabilitation interventions for people with joint injury and with or without surgery have been discussed elsewhere in detail. Selected potential interventions are presented in Table 7 (Clark et al., 2010, 2015a, 2015b; Swanik et al., 1997; Clark, 2015; Norte et al., 2021; Grooms et al., 2015) relative to the elements presented in Fig. 1. Because musculoskeletal conditions continue to be a major burden worldwide, (World Health Organization, 2022) and given physical rehabilitation for joint injury with or without surgery can still be unsuccessful for a substantial proportion of patients, (Thoma et al., 2019; Rosen et al., 2014; Woodmass et al., 2019; Bashaireh et al., 2024; Xu et al., 2022) this suggests that current physical rehabilitation techniques for joint injury are not yet as optimal as could be (Clark et al., 2023). This article offers itself as an educational resource and scientific asset to contribute to the ongoing research and applied practice journey

Table 7
Selected potential interventions and example therapeutic purposes for people with peripheral joint injury and the onset of an articuloneuromuscular cascade.

Articuloneuromuscular Cascade Paradigm Elements	Potential Intervention	Example Therapeutic Purpose
Inflammation	• Cryotherapy • TENS • Taping • Bracing[a]	• Decrease pain
	• Joint mobilisation • CPM machine • Passive ROM exercise • Active ROM exercise	• Decrease pain and disperse effusion
Impaired proprioception	• Joint mobilisation • Passive ROM exercise	• Stimulate cutaneous mechanoreceptors, joint mechanoreceptors, spinocerebellum, and primary somatosensory cortex
	• Taping • Bracing	• Stimulate cutaneous mechanoreceptors and primary somatosensory cortex
	• Massage	• Stimulate cutaneous mechanoreceptors, muscle mechanoreceptors, spinocerebellum, and primary somatosensory cortex
	• Active ROM exercise • OKC strength training • CKC strength training • Balance training • Plyometric training • Change-of-direction training • Agility training • Vibration device training	• Stimulate joint mechanoreceptors, muscle mechanoreceptors, tendon mechanoreceptors, spinocerebellum, and primary somatosensory cortex
Altered CNS processing (supraspinal) and impaired feedforward neuromuscular control	• Visual disruption/ occlusion[b]	• Down-weight visual-motor movement control and up-weight proprioception-motor movement control
	• Visual distraction[c] • Visual-motor reactive balance and agility training[d]	• Down-weight visual-motor movement control and up-weight proprioception-motor movement control • Enhance integration of neurocognitive (conscious) and sensorimotor (subconscious-level) systems
	• Dual-task visual memory-motor training[e]	• Down-weight visual-motor movement control and up-weight proprioception-motor movement control • Enhance integration of neurocognitive (conscious) and sensorimotor (subconscious-level) systems

(continued on next page)

Table 7 (*continued*)

Articuloneuromuscular Cascade Paradigm Elements	Potential Intervention	Example Therapeutic Purpose
		• Expand neurocognitive (conscious) processing capacity
	• Active ROM exercise • OKC strength training • CKC strength training	• Activate supplementary motor area, premotor cortex, primary motor cortex, and cerebrocerebellum
	• Balance training • Plyometric training	• Stimulate α and γ lower motor-neurons • Increase magnitude of feedforward muscle activation and stiffness
	• Change-of-direction training • Agility training • Vibration device training	
Altered CNS processing (spinal) and impaired feedback neuromuscular control	• Cryotherapy • TENS	• Reverse arthrogenic muscle inhibition and increase lower motor-neuron pool excitability
	• OKC strength training • CKC strength training • Balance training • Plyometric training	• Stimulate joint-muscle reflexes • Stimulate muscle-muscle reflexes • Decrease time for (i.e., increase speed of) joint-muscle reflexes
	• Change-of-direction training • Agility training • Vibration device training	• Increase magnitude of feedback muscle activation and stiffness
Impaired muscle physiology and mechanics	• NMES • Blood flow restriction training	• Deter muscle fibre atrophy • Deter collagen proliferation and expansion of extracellular matrix • Deter decrease in muscle contractility
	• OKC strength training • CKC strength training	• Stimulate muscle hypertrophy • Increase muscle stiffness • Increase muscle contractility
Impaired tendon physiology and mechanics	• OKC strength training • CKC strength training • Plyometric training	• Deter tendon collagen degradation • Deter decrease in tendon collagen synthesis • Deter decrease in tendon structural and material stiffness • Increase tendon collagen synthesis • Increase tendon structural and material stiffness
Impaired muscle performance	• OKC strength training • CKC strength training • Balance training • Plyometric training	• Deter muscle weakness • Increase muscle strength • Increase rate-of-force development • Decrease time-to-peak-torque

TENS = transcutaneous electrical nerve stimulation; CPM = continuous passive motion; ROM = range-of-motion.
OKC = open kinetic chain; CKC = closed kinetic chain; CNS = central nervous system.
α = alpha; γ = gamma; NMES = neuromuscular electrical stimulation.
[a] Here, bracing refers to non-rigid elastic bandage tubing, neoprene sleeves, etc.
[b] Visual disruption/occlusion can be achieved using stroboscopic glasses *during* rehabilitation exercises.
[c] Visual distraction can be achieved using object tracking tasks (e.g., ball tracking) *during* rehabilitation exercises.
[d] Visual-motor reactive training can be achieved using movement tasks that require rapid responses to visual stimuli (e.g., random light cues).
[e] Dual-task visual memory-motor training can be achieved using short-term memory tasks (e.g., memorising a random number sequence) superimposed on a single-leg unstable surface balance task.

for developing optimal joint injury rehabilitation strategies.

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