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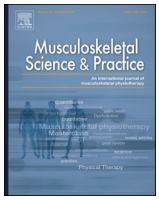

Technical and measurement report

Measuring complexity of muscle force control: Theoretical principles and clinical relevance in musculoskeletal research and practice

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ABSTRACT

Musculoskeletal conditions affect bones, joints, and muscles of the locomotor system and are a leading cause of disability worldwide. This suggests that current musculoskeletal rehabilitation techniques fail to target the characteristics (e.g., physiological/physical/psychological) most influential for long-term musculoskeletal health. To identify whether a physiological characteristic is impaired, it must be measured. In neuromuscular control, traditional research approaches use magnitude-based measurements (e.g., peak force/standard deviation of force/coefficient of variation of force). However, magnitude-based measurements miss 'hidden information' regarding a physiological system's status across time. To better identify physiological characteristics that are clinically-important for long-term musculoskeletal health, other measurement approaches currently less applied in musculoskeletal research may be helpful. The purpose of this article is to present an introduction to technical and measurement principles for quantifying the 'complexity' of muscle force control as one representation of peripheral joint neuromuscular control. Complexity measurements are time-based and consider the irregular temporal structure of physiological signals. We review theoretical principles underlying measuring complexity of muscle force control and explain its clinical relevance for musculoskeletal scientists and clinicians. The principles include sensorimotor control of peripheral joints, muscle force signal construction and features, muscle force control measurement procedures, and variability and complexity variables. We propose the potential utility of measuring the complexity of muscle force control for diagnosing sensorimotor system impairment and prognosis following musculoskeletal disease or injury. This article will serve as an educational asset and a scientific resource that will inform future research directions to optimise rehabilitation for people with peripheral joint disease and injury.

1. Introduction

Musculoskeletal conditions affect the bones, joints, and muscles of the locomotor system and are a leading cause of disability worldwide (World Health Organization, 2022; Vos et al., 2017). Musculoskeletal conditions include disease (e.g., osteoarthritis) and injury (e.g., ligament sprain) of the musculoskeletal system (World Health Organization, 2022). Peripheral joint conditions are common (Govaerts et al., 2021; Hootman et al., 2007). Because musculoskeletal conditions are a leading cause of disability worldwide (World Health Organization, 2022; Vos et al., 2017), this suggests, in our opinion, that current musculoskeletal rehabilitation techniques may be sub-optimal and fail to target the characteristics (e.g., physiological/physical/psychological) most influential for long-term peripheral joint health. However, musculoskeletal rehabilitation techniques cannot target clinically-important characteristics unless such characteristics have first been identified. To identify physiological characteristics that are clinically-important for long-term peripheral joint health, other measurement approaches currently less applied in musculoskeletal research may be helpful.

The term 'neuromuscular control' is used in many disciplines to describe some form of interaction between the nervous and muscular systems for the purpose of controlling movement (Williams et al., 2001; Riemann and Lephart, 2002). Thus, the measurement of neuromuscular control can be represented by any measurement procedure that characterises some aspect of skeletal muscle activation or force generation via the efferent components of the nervous system (Riemann et al., 2002) (Table 1). The purpose of this article is to present an introduction to technical and measurement principles for quantifying 'complexity' of

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Table 1
Terminology used in the sensorimotor control of peripheral joints[a].

Term	Definition and Explanation
Sensorimotor control	The control of joint stability, posture, and whole-body movement via the sensorimotor system
Sensorimotor system	All nervous system components involved in the acquisition of a sensory stimulus and its transmission to the CNS, the processing of that sensory stimulus within the CNS, and the resultant motor output from the CNS
Proprioceptor	A mechanoreceptor that transduces mechanical stimuli in joint soft tissues and skeletal muscles to electrical signals for transmission to the CNS
Proprioception	The sensory component of sensorimotor control, including [1] the sense of joint position (joint position sense), [2] the sense of joint movement (kinaesthesia), and [3] the sense of force (force sense)
Sensory (afferent) pathway	A pathway formed by all nervous system components involved in the transmission of afferent information, including [1] the mechanoreceptor nerve ending, [2] its afferent nerve fibre, [3] the synapses and interneurons within the afferent pathway, and [4] the ascending tracts to sensory nuclei in the brainstem, cerebellum, and cerebral cortex
Central nervous system processing	All spinal cord, brainstem, cerebellar, and cerebral physiological processes involved in the [1] transmission and [2] perception of afferent information and [3] the transformation of that afferent information into efferent commands
Motor (efferent) pathway	A pathway formed by all nervous system components involved in the transmission of efferent information, including [1] upper motor neurons, [2] the descending tracts from motor nuclei in the cerebral cortex and brainstem, [3] the synapses and interneurons within the efferent pathway, and [4] the lower motor neurons terminating at the motor endplate
Neuromuscular	A general term that describes any interaction between [1] the nervous system and [2] the muscular system; together termed the neuromuscular system
Neuromuscular control	The motor component of sensorimotor control, defined as activation of the dynamic restraints (skeletal muscles) [1] in preparation for and [2] in reaction to joint loading and movement Neuromuscular control involves both muscle activation and force generation elements Measurement of neuromuscular control involves any measurement that results in the creation of a variable to represent some aspect of skeletal muscle activation and/or force generation in the context of joint stability and joint health; example measurement procedures include electromyography, maximum muscle strength assessment, rate of force development assessment, and muscle force control assessment
Muscle force control	One representation of neuromuscular control, involving the ability to control (i.e., determine, scale, regulate) muscle force generation via pre-synaptic inputs to the lower motor neurons of a skeletal muscle The assessment of muscle force control typically involves sustained isometric muscle actions at submaximal efforts against a dynamometer The characteristics and behaviour (e.g., complexity) of the muscle force signal are used to make inferences about the status and ability of the sensorimotor system to control the generation of muscle force

CNS = central nervous system

[a] Definitions and explanations paraphrased from: Clark and Lephart (2015) Kandel (2013), Lephart et al. (2000), Nowak et al. (2013), Ostry and Feldman (2003), Pethick et al. (2022a), Riemann and Lephart (2002), Williams et al. (2001)

muscle force control as one representation of neuromuscular control (Clark and Pethick, 2022) (Table 1). Specifically, we review theoretical principles underlying the measurement of complexity of muscle force control and explain its clinical relevance for musculoskeletal scientists and clinicians. We anticipate this article will serve as an educational asset and a scientific resource that will inform future research directions to optimise rehabilitation for people with peripheral joint disease and injury.

2. Theoretical principles

2.1. Sensorimotor control of peripheral joints

To understand how neuromuscular control influences peripheral joint health, it is necessary to understand sensorimotor control of peripheral joints and the terminology used therein (Riemann and Lephart, 2002; Clark and Herrington, 2010; Lephart et al., 2000; Kandel et al., 2013) (Table 1). The sensorimotor system includes sensory (afferent), processing (central nervous system [CNS]), and motor (efferent) components (Riemann and Lephart, 2002; Wolpert et al., 2013) (Fig. 1). The sensorimotor system operates on a 'sensory-motor' basis, meaning sensory input to the CNS is necessary before motor output can be generated (Riemann and Lephart, 2002; Wolpert et al., 2013) (Fig. 1).

Skeletal muscle fibres are innervated by lower motor neurons (LMN) stimulated by spinal inputs from articular and muscle mechanoreceptors (proprioceptors) (Johansson et al., 1986; Pearson et al., 2013), along with supraspinal inputs via the descending tracts (Nathan et al., 1990, 1996). These inputs are modulated by pre-synaptic excitatory and inhibitory interneurons (Pearson et al., 2013; Brownstone and Bui, 2010). Motor units are, subsequently, the "final common pathway" by which multiple excitatory and inhibitory inputs are filtered to culminate in the activation of skeletal muscle and the generation of muscle force (Enoka and Farina, 2021; Sherrington, 1906) (Fig. 2).

Skeletal muscle protects bone and cartilage by absorbing vibration and compression forces (Rudenko et al., 2016; Pain and Challis, 2006). Skeletal muscle also protects joint capsules and ligaments by resisting excessive tension forces (Olmstead et al., 1986; McQuade and Murthi, 2004). Because skeletal muscle "stress-shields" articular tissues from potentially damaging forces (Clark and Lephart, 2015), peripheral joint health relies on optimal neuromuscular control and muscle force generation appropriate for a given physical task.

2.2. Muscle force signal construction

Muscle force generation is examined commonly using dynamometry (Riemann et al., 2002) (Fig. 3). The process involves a physiological characteristic (muscle force) being detected by a sensing device (load cell), quantified using a scale (e.g., Newton-metres), and recorded by another device (computer) for *one moment-in-time* as a single data-point (Bruce, 2000). When measurements continue *across a period-of-time*, thousands of data-points can be recorded (Bruce, 2000). Thousands of data-points collected at specific intervals over a period-of-time (e.g., one per millisecond [ms] for 5 s [s] = 5000 data-points) are termed a 'time-series' (Bruce, 2000). When time-series are plotted in graphical form, a visual representation of a physiological 'signal' is constructed (Bruce, 2000) (Fig. 4).

Physiological signals represent the interaction of multiple physiological components and feedback loops operating over different spatial and temporal scales (Lipsitz and Goldberger, 1992). For example, motor pathways include upper motor neurons descending from supraspinal nuclei to the anterior horn of the spinal cord, various spinal interneurons, and LMNs to peripheral joint muscles (Nathan et al., 1996; Macpherson et al., 2013). Therefore, for skeletal muscle force generation to occur, action potentials generated in supraspinal nuclei travel substantial distances (spatial scale) through multiple nervous system components to the motor endplate; this requires 50–120 ms (temporal scale) for reflex and voluntary muscle activation conditions (Crevecoeur and Kurtzer, 2018). Because physiological systems contain multiple anatomical components of different sizes, with each anatomical component containing physiological processes requiring different timeframes, a system's output (i.e., signal) is characterised by constant changes (i.e., *fluctuations*) across periods-of-time (Lipsitz and Goldberger, 1992). Therefore, quantifying muscle force signal fluctuations is

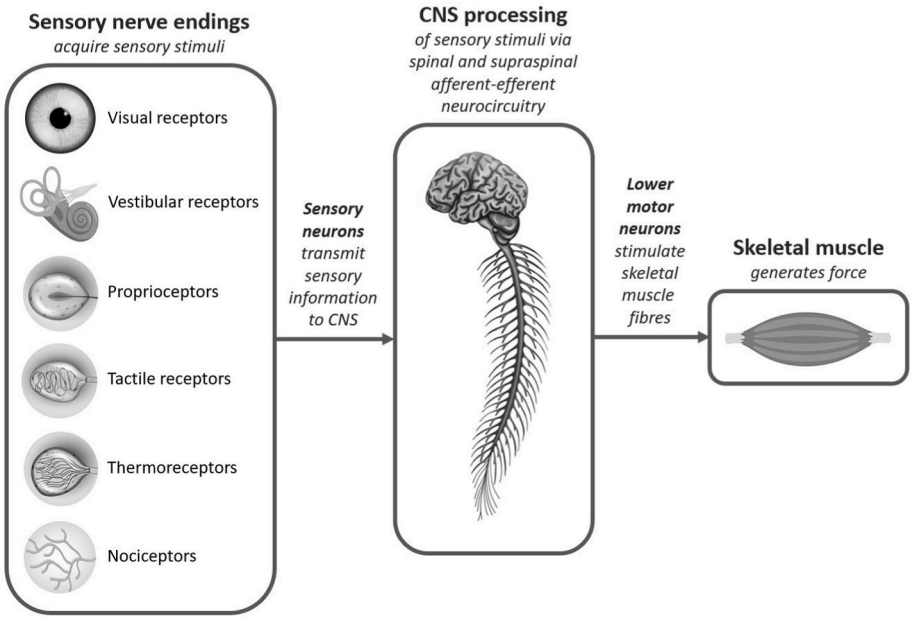

Fig. 1. Sensorimotor system configuration. CNS = central nervous system.

needed to understand a person's neuromuscular control characteristics better (Clark and Pethick, 2022).

2.3. Muscle force signal features

Muscle force signal features can be observed in the amplitude dimension (Fig. 4, *y*-axis) and time dimension (Fig. 4, *x*-axis). In musculoskeletal science, researchers commonly focus on the amplitude dimension with interest in a single data-point representing peak force (i.e., maximum strength) (Shaffer et al., 2000; Saccol et al., 2014). Beyond peak force, the features of a muscle force signal can be observed further using approaches that examine variability or complexity (Slifkin and Newell, 1999; Stergiou and Decker, 2011).

Variability refers to the normal fluctuation in sensorimotor control characteristics across consecutive repetitions of a movement task (Stergiou et al., 2006). Variability measures are *magnitude-based* (Enoka et al., 2003; Pethick et al., 2015); they consider the nature of fluctuation in the amplitude dimension (*y*-axis) (Slifkin and Newell, 1999; Stergiou et al., 2006) (Fig. 4, solid arrow). Complexity refers to the irregular structure of physiological signals across time (Slifkin and Newell, 1999). Complexity measures are *time-based* (Goldberger et al., 2002; Pincus, 1991); they consider the nature of fluctuation in the time dimension (*x*-axis) by examining how a signal's structure (i.e., waveform slope/amplitude/period) is irregular and fluctuates over time (Slifkin and Newell, 1999; Pethick et al., 2015) (Fig. 4, dotted arrow). For example, Fig. 4 shows the fluctuating structure of a muscle force signal across several seconds in the form of rapidly changing waveform periods within each second. Variables commonly used in quantifying variability and complexity are described below.

Historically, signal structure fluctuations were viewed as unwanted 'noise' relative to the perspective that healthy physiological processes (e.g., heart rate) operate to reduce variability and maintain constant (non-random/periodic) function (Lipsitz and Goldberger, 1992; Sejdić and Lipsitz, 2013). Subsequently, it was common to undertake signal noise reduction using mathematical filters (Bruce, 2000). More recently, *irregular (random/chaotic) behaviour of physiological signals have been observed consistently in healthy people*; for example, heart rate, respiratory rhythm, and brain activity (Goldberger et al., 2002). In contrast, it has been demonstrated repeatedly that *regular (non-random/periodic) behaviour of physiological signals characterises disease and injury* (Fino et al., 2016; Vaillancourt et al., 2001; Pethick et al., 2022b).

Because irregular signal fluctuations are evident in the physiological outputs of healthy people, signal filtering risks removing valuable information about a physiological system's status (Lipsitz and Goldberger, 1992; Sejdić and Lipsitz, 2013). Further, magnitude-based analyses of signal behaviour alone can miss 'hidden information' regarding time-based fluctuations (Slifkin and Newell, 1999) (Fig. 5). In other words, focusing on information from the amplitude dimension of a signal alone (Fig. 4, *y*-axis, solid arrow) fails to see information about fluctuations that would otherwise remain 'hidden' in the time dimension of a signal (Slifkin and Newell, 1999) (Fig. 4, *x*-axis, dotted arrow; Fig. 5). For example, in people with anterior cruciate ligament (ACL) injury, complexity measures of knee extensor muscle force control have uncovered injured versus uninjured limb differences whereas variability measures of knee extensor muscle force control have not (Hollman et al., 2021; Skurvydas et al., 2011); specifically, measures revealed lower complexity in the ACL-injured limb versus the uninjured contralateral limb (Hollman et al., 2021; Skurvydas et al., 2011). Therefore, time-based fluctuations ('complex fluctuations') in muscle force signals are now of special interest to researchers because they act as a biomechanical (kinetic) surrogate measurement that gives novel insight into peripheral joint sensorimotor control (neurophysiological) strategies (Nowak et al., 2013; Nagamori et al., 2021; Tracy, 2007).

2.4. Muscle force control: measurement procedures and variables

Muscle force control is one representation of neuromuscular control (Clark and Pethick, 2022) (Table 1). The hardware and software used for measuring peripheral joint muscle force control have been described elsewhere by researchers working with healthy people (Pethick et al., 2015; Vaillancourt and Newell, 2003) and those with disease (Vaillancourt et al., 2001; Chow and Stokic, 2014) and injury (Skurvydas et al., 2011; Hollman et al., 2021). Muscle force control is examined using computerised dynamometry (Fig. 3). Typically, isometric submaximal efforts at set percentages (e.g., 20–70%) of previously measured maximal efforts are held for sustained periods-of-time (e.g., 5–25s) (Pethick et al., 2015; Vaillancourt and Newell, 2003; Stanislovaitienė et al., 2009). Isometric (static) versus anisometric (dynamic) efforts are preferred in order to eliminate extraneous sources of force fluctuation that occur with joint movement (e.g., stretch reflex servo-loop,

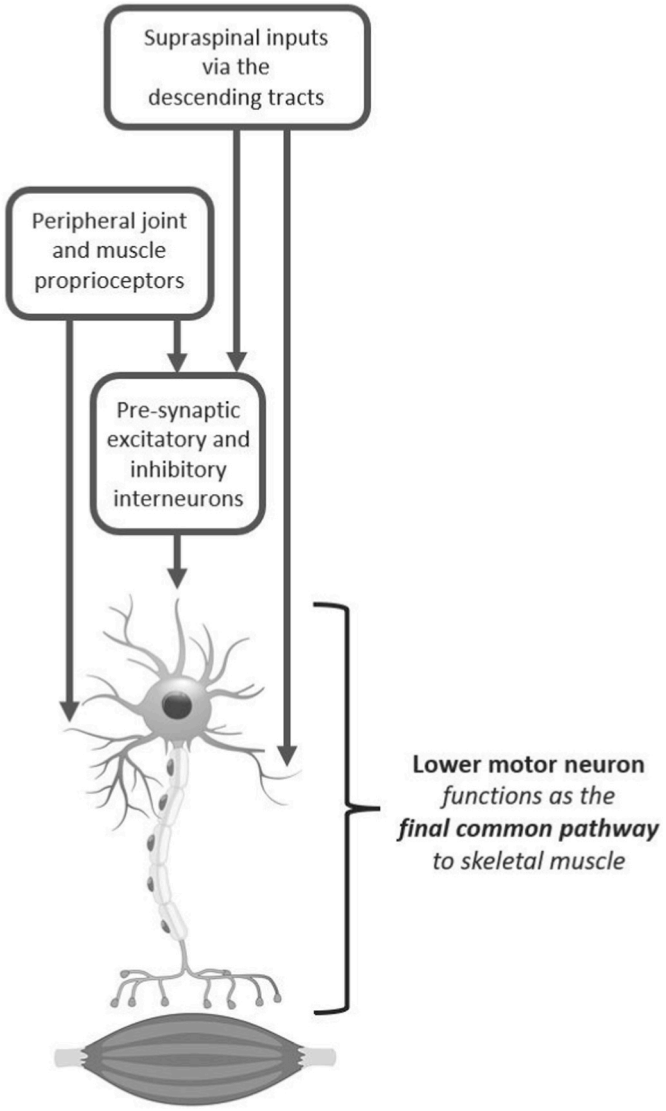

Fig. 2. Final common pathway.

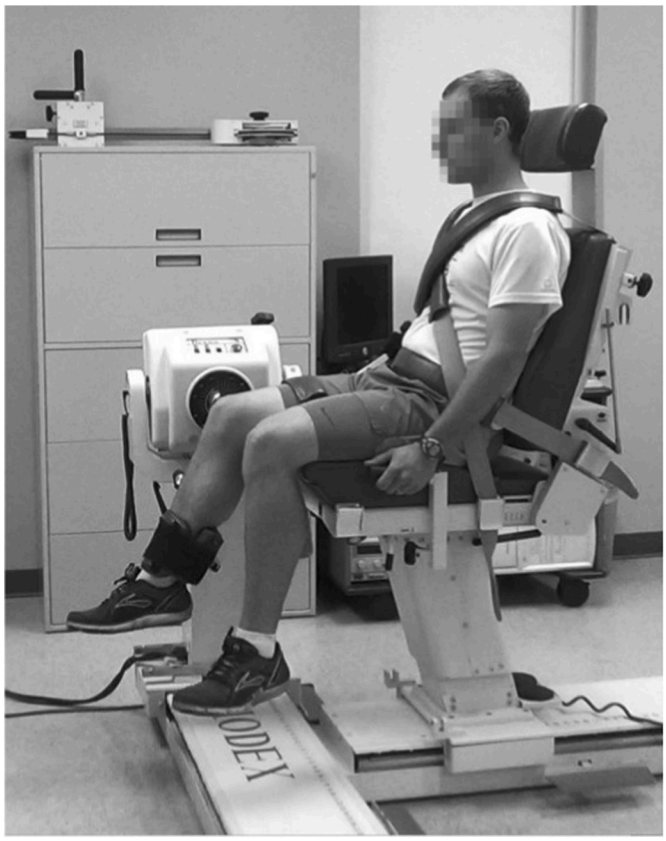

Fig. 3. Isometric knee extension dynamometry.

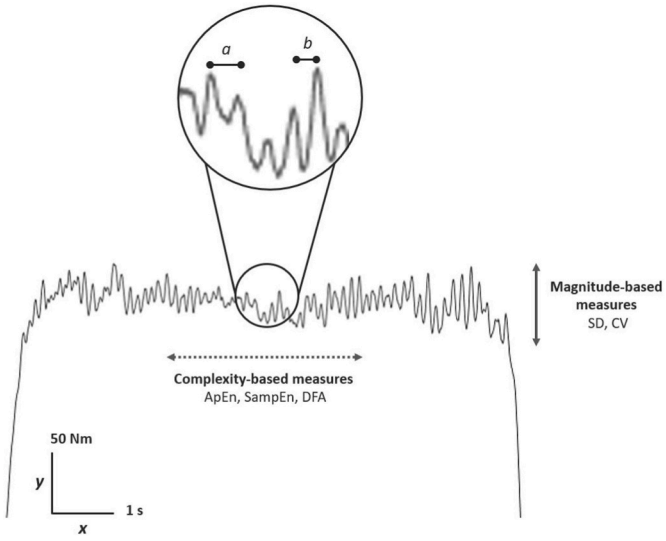

Fig. 4. Visual representation of an isometric force signal during a knee extension task.
This figure illustrates how variability-based (magnitude-based, amplitude dimension, *y*-axis) and complexity-based (time-based, time dimension, *x*-axis) variables relate to the graphical plot of the biomechanical (kinetic) signal. SD = standard deviation; CV = coefficient of variation; ApEn = approximate entropy; SampEn = sample entropy; DFA = detrended fluctuation analysis α; Nm = Newton-metres; s = seconds. See main text and Table 2 for explanation of variables. Note the example fluctuation of waveform periods 'a' and 'b', where period 'a' is of greater duration than period 'b'.

dynamometer mechanical vibration) (Furness et al., 1977). Submaximal efforts are preferred because low-magnitude muscle forces are adequate to enhance peripheral joint stability (Olmstead et al., 1986; McQuade and Murthi, 2004) and are more representative of muscle force magnitudes during day-to-day activities (Kern et al., 2001). After raw data has been recorded, it is processed in software using elaborate equations to create variables that quantify muscle force fluctuations. Example sources which explain in detail the equations and variables used by researchers are listed in Table 2.

2.4.1. Variables commonly used to quantify variability of muscle force control

Variability analyses draw from linear dynamics and employ descriptive statistical procedures (Slifkin and Newell, 1999; Stergiou et al., 2006). These analyses represent the nature of signal fluctuation in the amplitude dimension (*y*-axis, Fig. 4, solid arrow) around the mean of a time-series (Slifkin and Newell, 1999; Stergiou et al., 2006). The standard deviation (SD) and coefficient of variation (CV) are used to quantify absolute variability and relative variability normalised to the mean, respectively (Pethick et al., 2015; Galganski et al., 1993) (Table 2). The SD and CV yield values of different information for time-series of different levels of isometrics efforts (Enoka and Farina, 2021). Therefore, both should be used when examining the variability of

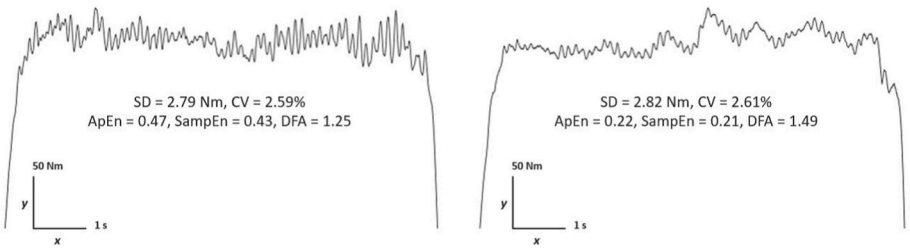

Fig. 5. Comparison of variability and complexity measures across two isometric force signals during a knee extension task.
This figure illustrates how the variability variables (SD = standard deviation; CV = coefficient of variation) are of almost identical values for both signals but the complexity variables (ApEn = approximate entropy; SampEn = sample entropy; DFA = detrended fluctuation analysis α) are of very different values for both signals. *Focusing on the variability (magnitude-based) variables alone would miss the information quantified by the complexity (time-based) variables*. Nm = Newton-metres; s = seconds. See main text and Table 2 for explanation of variables.

muscle force control (Pethick et al., 2022b).

2.4.2. *Variables commonly used to quantify complexity of muscle force control*

Complexity analyses draw from nonlinear dynamics and include approaches from information theory (e.g., entropy statistics) and fractal geometry (Lipsitz and Goldberger, 1992; Peng et al., 2009). These analyses examine the nature of signal fluctuation in the time dimension (*x*-axis, Fig. 4, dotted arrow) by characterising the moment-to-moment relationships between successive data-points and how a signal's structure is irregular and changes over time (Slifkin and Newell, 1999; Pethick et al., 2015). Approximate entropy (ApEn) and sample entropy (SampEn) quantify the apparent regularity or randomness of a signal (Lipsitz and Goldberger, 1992; Richman and Moorman, 2000; Peng et al., 2009) (Table 2). The term 'entropy' is not used in the thermodynamics sense (Lipsitz and Goldberger, 1992). Rather, it relates to the amount of information derived from a signal (Pincus, 1991; Pethick et al., 2021). Both ApEn and SampEn, however, are limited because they only examine signal regularity/randomness over one timescale (Pethick et al., 2021, 2022a; Costa et al., 2002). Alternatively, multiscale entropy (MSE) can be used to examine signal regularity/randomness over multiple timescales (Costa et al., 2002; Seely and Macklem, 2004).

For the physical aspect of a system, the term 'fractal' refers to the geometric measurement of irregular shapes (spatial scale) with underlying structural patterns of units, sub-units, and sub-sub-units (Lipsitz and Goldberger, 1992) (e.g., neuronal dendrites). The phenomenon is that when 'zooming in' to inspect the physical structure of sub-sub-units and sub-units, both resemble the physical structure of the larger unit, a feature termed "self-similarity" (Lipsitz and Goldberger, 1992). In other words, regardless of the level of magnification and the length scale used (e.g., millimetre, micrometre), the physical structure of a sub-sub-unit will resemble that of a sub-unit and a unit (Lipsitz and Goldberger, 1992). For the physiological aspect of a system, signals are fractal if, as a function of time (temporal scale), they undergo self-similar fluctuations regardless of the timescale over which data-points are sampled (e.g., ms, microsecond) (Goldberger et al., 2002). Therefore, the fractal concept can be translated from contexts examining physical structures across multiple different length scales to those examining physiological functions (i.e., signal structures; e.g., Fig. 4) across multiple different timescales (Lipsitz and Goldberger, 1992). An example of a fractal variable is detrended fluctuation analysis (DFA) which quantifies a signal's structural pattern over several timescales (Pethick et al., 2022a; Goldberger et al., 2002; Peng et al., 1994) (Table 2). Because entropy and fractal variables represent different physiological characteristics, both should be used to examine muscle force control signal complexity thoroughly (Clark and Pethick, 2022; Pethick et al., 2022a; Lipsitz and Goldberger, 1992).

3. Clinical relevance

Motor units are the final common pathway by which multiple inputs from articular and muscular proprioceptors, descending tracts, and interneurons are filtered to activate skeletal muscle and generate muscle force (Enoka and Farina, 2021; Sherrington, 1906) (Fig. 2). Therefore, following disease or injury, it can be hypothesised that *changes in the complexity of muscle force control give insight into the status of the sensorimotor system anywhere 'upstream' to the cell body of a LMN*. In disease and injury, sensorimotor impairments include altered proprioceptor morphology (Ludwig et al., 2015), the destruction of proprioceptors (Bali et al., 2012), modified cortical processing (Valeriani et al., 1999; Parker et al., 2017), and altered descending tract function (Terada et al., 2016). Because skeletal muscle protects bone, cartilage, and capsuloligamentous structures from excessive forces (Clark and Lephart, 2015; Rudenko et al., 2016; Pain and Challis, 2006; Olmstead et al., 1986; McQuade and Murthi, 2004), sensorimotor impairments that result in altered inputs to LMNs and concurrently altered muscle activation patterns and force control could have clinically-important implications for peripheral joint tissue health and daily function (Pethick et al., 2022b). For example, the inability to "fine tune" muscle force control may contribute to excessive tissue loading (Pain and Challis, 2006; Wakeling et al., 2001) and impaired muscle force control may reduce the ability to resist unpredictable external mechanical perturbations (Peng et al., 2009).

Magnitude-based measures examining muscle strength represent system *capacity* (Hamberg-van Reenen et al., 2009). Contrastingly, time-based measures examining complex fluctuations are interpreted as representing system *adaptability*; the range across which a biological system functions and its ability to adapt (i.e., adjust, respond) to changing task or environmental demands (Stergiou and Decker, 2011; Goldberger et al., 2002; Sejdić and Lipsitz, 2013). For example, in the respiratory system, lower complexity of resting airflow patterns in those with asthma is associated with a decreased ability to adjust (increase) airflow relative to greater respiratory task demands (Veiga et al., 2011). In the sensorimotor system, specifically, lower complexity of muscle force control is associated with a decreased ability to respond to (perform) clinical co-ordination tasks in people with (Lodha et al., 2010) and without (Mear et al., 2023) pathology. Lower complexity, subsequently, is associated with decreased adaptability across multiple biological systems (Lipsitz and Goldberger, 1992; Veiga et al., 2011; Lodha et al., 2010; Mear et al., 2023). Therefore, for the musculoskeletal system, decreased sensorimotor system adaptability may result in ongoing vulnerability to injury and a reduced range of situations across which an individual can function effectively.

Since the complexity of muscle force control is implicated in peripheral joint tissue health, its measurement may be helpful following a history of musculoskeletal disease or injury for identifying (diagnosing) persistent sensorimotor impairment upstream from the LMN. Since muscle force control complexity is also implicated in neuromuscular control adaptability and daily functional ability, there may also be a role for its measurement in prognosis following musculoskeletal disease or injury. Therefore, measuring the complexity of muscle force control may be a first step along a path to identifying physiological characteristics that are clinically-important for long-term peripheral joint health. Subsequent steps will then include the development of rehabilitation

Table 2
Example muscle force control variables, scales, and interpretations.

Example Variables	Abbreviation	Example Scales and Interpretations	Example Sources Explaining the Equations Used by Researchers
Variability Variables			
Standard deviation	SD	• 0.0 to *x*, the scale and units of measurement are the same as that used by the measuring device (e.g., Newton-metre) • 0.0 = no absolute variance from the central point (mean) of the time series • *x* = an absolute variance of *x* from the central point (mean) of the time series • Lower values represent more force control steadiness, higher values represent less force control steadiness	(Enoka and Farina, 2021) (Galganski et al., 1993) (Pethick et al., 2015)
Coefficient of variation	CV	• 0.0%–100.0%, a unitless scale • 0.0% = no relative variance from the central point (mean) of the time series • 100.0% = a relative variance of 100% from the central point (mean) of the time series • Lower values represent more force control steadiness, higher values represent less force control steadiness	(Enoka and Farina, 2021) (Galganski et al., 1993) (Pethick et al., 2015)
Complexity Variables			
Approximate entropy	ApEn	• 0.0 to 2.0, a unitless scale • 0.0 = high signal regularity, low signal complexity • 2.0 = low signal regularity, high signal complexity	(Pethick et al., 2015) (Pincus, 1991) (Richman and Moorman, 2000) (Slifkin and Newell, 1999)
Sample entropy	SampEn	• 0.0 to 2.0, a unitless scale • 0.0 = high signal regularity, low signal complexity • 2.0 = low signal regularity, high signal complexity	(Pethick et al., 2015) (Pincus, 1991) (Richman and Moorman, 2000) (Slifkin and Newell, 1999)
Multiscale entropy	MSE	• 0.0 to 2.0, a unitless scale • 0.0 = high signal regularity, low signal complexity • 2.0 = low signal regularity, high signal complexity	(Costa et al., 2002)
Detrended fluctuation analysis alpha (α) exponent	DFA α	• ≈0.5 to ≈1.5, a unitless scale • ≈0.5 = random signal, high complexity • ≈1.0 = signal with self-similar fluctuations, moderate complexity • ≈1.5 = smooth signal, low complexity	(Goldberger et al., 2002) (Peng et al., 1994) (Pethick et al., 2015)

techniques that can modify the complexity of muscle force control in a way that contributes to enhanced clinical outcomes and a more optimal therapeutic journey for people with musculoskeletal disease or injury.

4. Summary

This article has presented an introduction to technical and measurement principles for quantifying complexity of muscle force control as one representation of peripheral joint neuromuscular control. Theoretical principles underlying the sensorimotor control of peripheral joints, muscle force signal construction, muscle force signal features, muscle force control measurement procedures, variability variables, and complexity variables have been presented. Commentary on the clinical relevance of these theoretical principles has been offered, including the potential utility of measuring muscle force control complexity for the diagnosis of persistent sensorimotor system impairment and prognosis following musculoskeletal disease or injury.

Ethical statement

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Declaration of competing interest

The authors declare there are no competing interests.

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