OPINION

A unifying motor control framework for task-specific dystonia

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Abstract | Task-specific dystonia is a movement disorder characterized by a painless loss of dexterity specific to a particular motor skill. This disorder is prevalent among writers, musicians, dancers and athletes. No current treatment is predictably effective, and the disorder generally ends the careers of affected individuals. Traditional disease models of dystonia have a number of limitations with regard to task-specific dystonia. We therefore discuss emerging evidence that the disorder has its origins within normal compensatory mechanisms of a healthy motor system in which the representation and reproduction of motor skill are disrupted. We describe how risk factors for task-specific dystonia can be stratified and translated into mechanisms of dysfunctional motor control. The proposed model aims to define new directions for experimental research and stimulate therapeutic advances for this highly disabling disorder.

We enjoy marvelling at a musician in full flow during a performance or at the grace of a tennis player during a game. Although such activities appear effortless to the casual observer, these individuals have honed their motor ability through years of rigorous practice. We reward their expertise by filling concert halls and sports venues, and consider such exquisite movement control one of the pinnacles of human development. However, in a proportion of individuals, this repetitive practice of motor skill comes at a price — the development of a painless loss of coordination specific to their skill, termed task-specific dystonia. The motor impairment only manifests during a single task, yet its wider impact is considerable, as some of the individuals affected define our arts and sports communities.

Task-specific dystonia is currently considered a subtype of dystonia[1]. However, a long-standing debate continues as to whether the different types of dystonia represent a single disease entity with shared pathophysiology or whether each dystonia subtype is distinct[2]. Task-specific dystonia is unique in a number of ways[1]. Primarily, the isolated and highly task-specific nature of this condition ties the problem directly to the control of a specific motor task. In the affected body region, other fine motor tasks are initially unaffected[3]. Another distinctive feature is the range of environmental risk factors highlighted in this framework, which are repeatedly linked to symptomatology in epidemiological studies and clinical practice[4,5]. In many published studies, traditional neurophysiological markers of dystonia, such as abnormal sensorimotor plasticity and impaired inhibition, are also implicated in task-specific dystonia[6]. However, general changes in plasticity, inhibition or somatosensory representation are unable to explain why only an individual task is affected (as such abnormalities have also been documented in circuits subserving unaffected body regions[7–9]). Such neurophysiological markers are also highly variable in health, and abnormalities do not reliably or specifically identify patients with task-specific dystonia[10]. These observations point to the need to search for additional ways to understand the disorder.

In this Opinion article, we review emerging evidence that task-specific dystonia has its origins within normal compensatory mechanisms of a healthy motor system in which the representation and reproduction of motor skill are disrupted in response to a varied set of risk factors. We identify mechanisms by which sensorimotor control can be overwhelmed and discuss the translational implications this motor control framework yields for the prevention and treatment of task-specific dystonia.

Motor skill learning in health

A broad definition of motor skill learning is any neuronal change that enables an organism to accomplish a motor task faster and more precisely than before[11]. Increasing expertise is characterized by optimization of speed and accuracy[12], high consistency in achieving the movement goal (effectiveness), fluent and economical movement execution (efficiency) and automaticity[13].

Studies of motor skill learning suggest that a hierarchical organization of neuronal networks encodes the different skill components required for expert performance[11] (FIG. 1). The main broad division proposed within the motor hierarchy is between action selection and execution[11,14]. At the top is the selection level, which links the task goals to motor control circuits. This level takes into account the whole repertoire of movements and weighs up their motor costs and rewards before making the most appropriate selection. At the bottom of the hierarchy is the execution level, which involves neuronal populations that coordinate contraction of the muscles.

At the execution level, experimental data suggest that the primary motor cortex encodes small movement fragments or 'motor synergies' within stable neuronal networks[15,16]. Gestures as complex as grasping or licking can be reproducibly evoked via simple electrical stimulation of the primary motor cortex[17] (FIG. 2a). Furthermore, the range of motor synergies seems to depend on experience; skilled musicians have a motor synergy repertoire tailored to the instrument they have trained on[18]. As such, representations at the execution level can be considered building blocks for the motor system.

In the early stages of skill learning, most task requirements are thought to be explicitly processed at the action selection level and then directly mapped to the most appropriate execution elements[11]

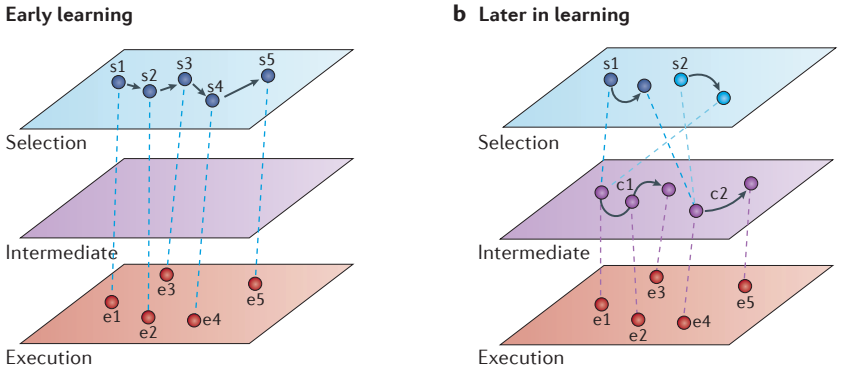

Figure 1 | **Motor hierarchy in skill learning. a** | In early learning, explicit or cognitive processing of task instructions occurs at the selection level (s1–s5). At the execution level, the most appropriate set of motor elements (e1–e5) is mapped to task requirements[11]. **b** | Later in learning, task performance is largely automatic. Skill elements become encoded at an intermediate level within the dynamic neural network, and the flow of motor elements requires little explicit or cognitive control. Motor chunking refers to the linking of elemental execution representations within the intermediate level. For example, a specific motor goal at the selection level (s1) might initiate two motor chunks at the intermediate level (c1, c2) that lead to distinct motor sequences (e1–e3 in the case of c1 and e4–e5 in the case of c2). A second motor goal (s2) has a different order requirement but is built from the same components; initiating the two chunks in reverse order (c2 then c1) still produces the required motor behaviour (e4–e5 from c2 and e1–e3 from c1). These intermediate-level representations link elementary units, enabling them to be activated in a fluent manner without the need for direct mapping from the selection level. Figure adapted with permission from REF. 11, Elsevier.

(FIG. 1a). However, this is an effortful and time-consuming process, as multiple alternatives within the movement repertoire need to be considered. Therefore, later in learning, intermediate-level skill representations are thought to be formed, binding together elementary execution components (such as motor synergies) within a dynamic control network (FIG. 1b). Subsequently, the selection level might only need to trigger a corresponding intermediate network, which reduces load at the selection level and lessens the explicit processing of task requirements.

One experimental line of evidence that supports the existence of intermediate representations is the concept of 'motor chunking' — the grouping of elementary components of a sequential action into one representational unit[19]. With learning, as the completion of motor sequences becomes faster and more accurate, sequence execution starts to show idiosyncratic temporal groupings or chunks[20]. Novel sequences are performed faster if previously established chunks of trained motor sequences are preserved than if the chunks are regrouped[21]. As such, these chunks are thought to be linked at the intermediate level, and the transfer or flexibility of motor skill learning across different actions can be explained by the reuse of existing chunks in new motor sequences[22] (FIG. 2b). Chunk-specific neuronal activity has been shown experimentally in the (pre-) supplementary motor area, lateral premotor cortical areas and the striatum[22–26].

Besides the sequential order of individual movements, complex skill reproduction requires the integration of other features such as the temporal profile or rhythm of the sequence. Interestingly, such characteristics might be encoded separately from the motor sequence. Advantages emerge when previously trained spatial or temporal features of movement sequences are transferred to new spatiotemporal combinations, suggesting that these movement signatures are represented independently within the brain[27–29] (FIG. 2c). Accordingly, chunk-specific activity in striatal medium spiny neurons in rodents is not modulated by changes in the speed or timing of the trained motor action, suggesting that these neurons specify the order of movements in a chunk but not its full spatiotemporal implementation[30]. At the same time, motor sequence learning often leads to a separable representation of temporal features, which can be understood as a more abstract form of chunking — a temporal grouping of sequence elements that is transferable across different movements and effectors[29,31,32].

Thus, within this hierarchical model of motor skill learning, intermediate-level representations are thought to provide an architecture that enables the flexible modification and recombination of acquired movement chunks and timing, maximizing both the plasticity and efficiency of the motor skill network involving primarily cortical and striatal areas. This function contrasts with spatiotemporally intricate but stereotyped reflex movements (such as swallowing), which are controlled at the brainstem level and have a limited capacity for modification. The cortico-spinal and cortico-striatal pathways might, therefore, be biologically predisposed to the flexible control of skilled movements[33]. This informative literature exploring motor skill learning in health can offer important insights into disorders of skill reproduction such as task-specific dystonia.

Risk factors

Both genetic and environmental factors are thought to be important in the aetiology of task-specific dystonia. Genetic influences are suggested by the male preponderance and positive family history of movement disorders in a proportion of patients, and the *ARSG* gene (encoding arylsulfatase G) has been identified as a possible susceptibility locus[34–36]. However, how genetic and epigenetic factors contribute to the risk

Glossary

Automaticity
A mode of motor control in which movements operate with very little conscious knowledge of the actions required to perform them.

Chunking
Collection of elementary units that are inter-associated, stored in memory as one unit, and act as a coherent, integrated group when retrieved.

Dystonia
A movement disorder characterized by sustained or intermittent muscle contractions causing abnormal movements, abnormal postures or both.

Individuation
The degree to which a single finger can move without unintended movements of the other fingers of the same hand.

Motor hierarchy
A functional hierarchy of the motor system, with each level having specific roles in motor encoding and control of movement.

Motor synergies
Elemental action units, characterized by groups of weighted muscle activations that are coordinated in space and time.

Representation
Activity in neural substrates containing information about the external or internal state of the system, including motor output.

profile of an individual remains to be determined. Such factors could influence a wide range of processes, from the gating of synaptic plasticity through to the determination of personality traits or musical ability.

Environmental risk factors associated with task-specific dystonia are diverse and reveal great heterogeneity between patients[37,38]. Importantly, many of these risk factors suggest potential mechanisms by which motor control might malfunction. A pragmatic method to assess risk factors, which can also be used to guide rehabilitation strategies, is to identify factors associated with each of the essential components required for the performance of a given skill: task, tool, periphery and theCNS (FIG. 3). The periphery describes the characteristics of the body region that performs the task with the tool. The CNS includes the network that encodes skill performance, modulated by the individual's psychological state. As all components are required to work in concert to maintain task performance, a change in one component prompts a change or shift in other components (FIG. 3). A further dynamic element is that the risk factor profile of an individual can also change over time. Qualifying specific risk factors as predisposing, triggering and/or maintaining influences is often useful.

Task. The highest relative prevalence of task-specific dystonia is found in musicians (1 in 100 for musicians' dystonia versus 1 in 15,000 for writers' dystonia[39,40]), and the specific influences of task can be readily exemplified in this group. For example, dystonia in musicians preferentially involves the hand demanding the highest spatiotemporal acuity (right hand in keyboard players, left hand in players of bowed instruments)[4,37,40]. In bow-arm dystonia, although the effectors involved in arm movements are much larger than those involved in hand movements, the spatiotemporal demands involved in producing a pure note are similarly high. Greater neuronal organization is likely to be needed to achieve a desired skill when the task requirements are a large departure from the evolutionarily designed role of the relevant muscle groups. Task-specific dystonia is frequently observed in classical musicians, in part owing to the requirement for classical musicians to execute performances according to the invariant temporal and spatial parameters defined by their sheet music[41]. Motor impairments are less frequently seen in jazz musicians, as a certain flexibility of note and tempo is intrinsic to this music form[42]. Task-specific dystonia also typically affects the performance of highly rehearsed skills, tasks that have been performed repetitively for many hours[37]. Professional musicians typically accumulate 10,000 h of practice before symptom onset[43]. Thus, high-risk groups are characterized by exceptionally high task-accuracy requirements, a high cost associated with any deviation from predefined parameters, and highly rehearsed motor skills.

Tool. Changes in the presentation of patients with task-specific dystonia over the centuries highlight the importance of specific tools in the pathogenesis of task-specific dystonia[5,44]. In the 19th century, the change from feather quills to steel nibs in scriveners and clerks was the cause of a dramatic increase in the prevalence of occupational motor problems in the British Civil Service[45]. The new nibs altered the dynamics of the tool, as well as the corresponding task kinematics. Steel nibs also did not require writers to stop periodically to sharpen them, which offered a brief rest from what would otherwise be a continuous task, predisposing writers to muscle fatigue[45,46]. More than 10% of telegraph operators communicating in Morse code developed motor problems[47]. Here, the requirement for individuation and

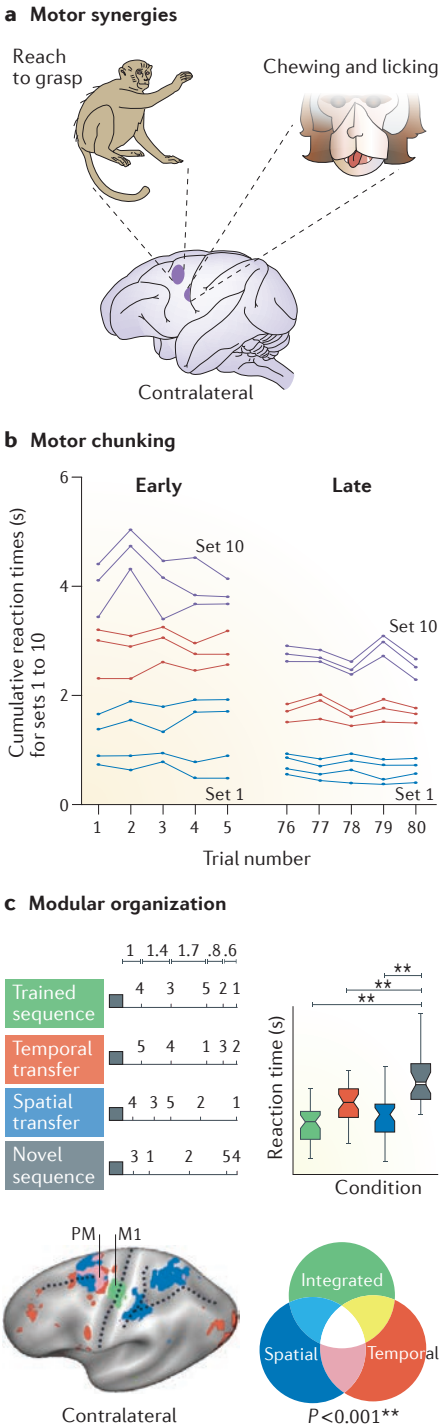

Figure 2 | **Evidence for a motor hierarchy. a** | Motor synergies are fragments of movement sequences encoded within the motor cortex. Complex gestures such as grasping or licking can be reproducibly evoked by electrical stimulation of the shaded regions of primary motor and premotor cortices in monkeys. **b** | The results of an experiment in which human volunteers learned 10 sets of two button presses. Each point on the graph represents one set, and the spacing between adjacent lines corresponds to the time intervals between initiation of each set. During early learning (trials 1–5), the total time taken for the sequence to be executed was longer, and dispersion of the 10 sets through time was approximately even. During late learning (trials 76–80), the total time taken for the entire sequence had decreased, and motor chunking is evident — the different sets are grouped into three chunks (sets 1–4, sets 5–7, sets 8–10). **c** | Evidence for a modular representation of rhythm or 'temporal chunking'. The timing and finger order of four different sequences are shown: trained (green); temporal transfer (red); spatial transfer (blue); and novel (grey). Behavioural benefits (reaction time decreases) of new sequences retaining either the trained temporal or spatial features are seen in comparison to entirely novel sequences. Multivariate analysis of functional MRI data reveals independent representations (red, blue) of these spatial and temporal features, some of which occur in overlapping (pink) regions of the premotor cortex (PM). The primary motor cortex (M1), by contrast, contains integrated (that is, non-separable) representations of the two sequence features (green). Part **a** adapted with permission from REF. 17, Elsevier. Part **b** adapted with permission from REF. 21, Springer. Part **c** adapted with permission from REF. 11, Elsevier.

stereotyped finger movements seemed to be particularly problematic[47]. By contrast, computer-related dystonia is infrequently described in the literature, perhaps owing to the use of ergonomic keyboard designs[48]. In musicians, the incidence of task-specific dystonia increases as the size of the string instrument decreases, suggesting that tools requiring a higher spatial resolution confer an increased risk of task-specific dystonia[49]. Clearly, tools largely confer risk as a result of the specific task requirements. However, occasionally, the risks conferred by task and tool are independent (for example, the higher incidence of task-specific dystonia in classical pianists than in jazz pianists), and a tool is not an essential component for the development of task-specific dystonia, as exemplified by the occurrence of task-specific singing impairment[50].

Periphery. Fatigue, overuse and injury are important risk factors for task-specific dystonia[37,38,45]. For example, facial injuries can precipitate motor impairments affecting the embouchure in wind and brass instrument players[42,51]. In some individuals, injury might be caused by excessive practice or performance. However, injury of the body part in a context removed from the task also increases the risk of task-specific dystonia[4]. Anatomical limitations of the body region required for the task are another important consideration in the assessment of task-specific dystonia. Some individuals are born with a musculoskeletal system that favours skilled performance (such as an optimal range of motion), whereas others have biomechanical constraints that predispose them to develop motor dysfunction[38].

CNS. All individuals are likely to have a ceiling capacity of their nervous system for encoding the different elements of a movement. Determinants of this capacity are likely to include a combination of nature (inherent talent or capacity for neural plasticity and processing) and nurture (exposure and training). Furthermore, exposure might have to occur within a window during which the conditions most favour skill learning. Musicians who start practising after the age of 10 years have an increased risk of developing task-specific dystonia[41]. This age is after the most sensitive periods of neural development have occurred, during which training is thought to have its greatest effects on brain structure and behaviour[52].

Finally, multiple cognitive and emotional processes can influence motor control. Compared with unaffected musicians, those with task-specific dystonia are six times as likely to exhibit elevated anxiety, perfectionism and evidence of stress, and such characteristics seem to predate the onset of dystonia[53–55]. In some patients, performance-related stress is clearly present in the run up to development of task-specific dystonia. In one case series, in which dystonia was evident only when writing a single letter or number, all patients were linked by the requirement to write the letter or number under stressful situations[56].

Implications for motor control

Interestingly, these risk factors for task-specific dystonia imply specific mechanisms by which the motor control system can become vulnerable to malfunction. Here, we discuss a number of unifying themes that describe how dysfunctional neural representations of motor skill might arise.

Neural correlates of skill expertise. Particularly in professional musicians and athletes, the limitations of neural networks supporting their skill expertise are likely to be an important contributory factor in the development of task-specific dystonia[57] (FIG. 4). Experimental data suggest that the repetitive practice of long sequences of movements can lead to the formation of progressively longer motor chunks over time[24,58,59], leading to performance gains that are increasingly contextual and tied to the individual task or body region[24,60–64]. Poor transfer of these performance gains to other tasks seems to be accentuated if a narrow training repertoire is applied, in contrast to more varied training approaches[65]. Practice predating the development of task-specific dystonia is often particularly

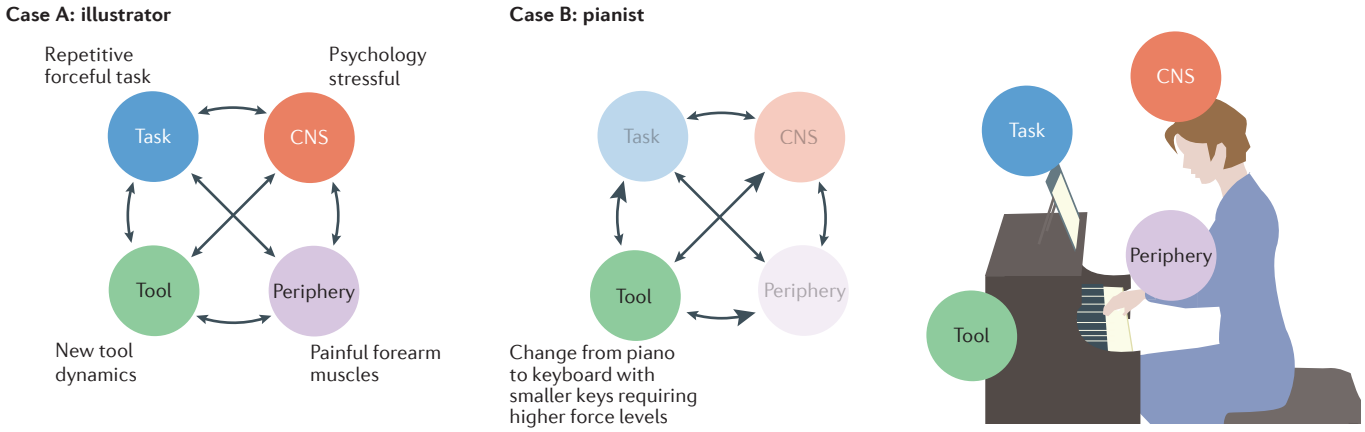

Figure 3 | **Components required for skill performance and the dynamic interactions between risk factors.** Risk factors can be comprehensively identified by considering all the components that interact in the performance of a given skill (CNS, periphery, task and tool). Case A exemplifies an illustrator who had taken a prestigious but demanding new job in animation. The patient was required to use a tablet and stylus rather than the usual paintbrush (tool), and the work required thousands of dots, demanding forceful demarcation with the stylus (task). The patient worked for many hours until a painful forearm overuse injury occurred (periphery) and was highly stressed by the attempt to meet her imposed deadline (psychology). Here, multiple risk factors probably interacted in the development of task-specific dystonia. Other patients present with fewer risk factors. Case B exemplifies a classical pianist who took a job playing in a musical. This required a subtle change in instrument; the pianist was using a smaller keyboard, with keys that were less responsive than those of a piano. In this case, the change in tool was the dominant risk factor, but this change had repercussions for task parameters and sensorimotor control (indicated by the arrows linking skill components).

extensive and stereotyped (for example, some musicians frequently practise each section of music in the same manner, with the same rhythm and same fingering, over and over again). In such highly rehearsed tasks, intermediate-level representations that previously conferred flexibility for related tasks (such as those involving the same chord transitions or rhythms) would become redundant as highly stereotyped sequences begin to dominate the movement repertoire. The original transferrable chunk structure could disappear, as concatenation into long, execution-bound synergies effectively replaces such intermediate elements (FIG. 4a). Such an architecture within the motor hierarchy could reliably encode an extreme optimization of performance parameters that pushes variability towards zero (FIG. 4b) but retains little capacity for flexibility and generalization to other contexts.

Capacity versus requirements. Many triggers for task-specific dystonia can be helpfully conceptualized as an unresolvable mismatch between the capacity of the motor system and the task requirements[38]. Capacity in this sense is defined by the limits of the neural control network and the periphery (for example, the range of feasible movements at a particular joint)[38]. Requirements are the exact movement trajectory, timing, force and accuracy required of the body in order to achieve the desired movement goals, as largely defined by the task and tool. Some mismatches between capacity and requirements are biomechanical in nature. For example, a task with a high force requirement will limit the capacity to make individuated finger movements, resulting in greater unintentional and undesired movements of neighbouring fingers[66–68]. Changes in capacity due to fatigue or injury of the body can also result in an effector system that responds more variably to a given motor command. Alternatively, a change in task requirements might result from external factors, such as changes in the size of a tool or an attempt by the performer to change their instrumental technique. If the neural representation of a skill can accommodate changes in the task requirements by adjusting and scaling its motor commands to maintain performance, an effective neural compensation has been found[38]. However, if the new task requirement cannot be accommodated by the existing representation, the performer is pushed outside the overlearned boundaries of the skill. An inability to transfer the highly optimized skill to a novel parameter will cause performance to break down because no effective motor compensation is available[38]. As we have already described in relation to professional performers affected by task-specific dystonia, skill representations that are highly optimized are likely to be particularly narrow in their ability to cope with a change in task requirements. This factor could help to explain the high prevalence of task-specific dystonia in these individuals, as well as why the triggering factors are often subtle[69–71] (FIG. 4c).

Ill-equipped corrective mechanisms. Once a critical mismatch between capacity and requirement has occurred, novel motor control strategies alien to the existing neural representation of skill must be employed to maintain task performance. However, *de novo* motor control solutions are unlikely to be able to match or maintain the level of skill performance that was formerly encoded by a hierarchy of neuronal elements optimized over many years of practice. The skills that are usually affected in task-specific dystonia are characterized by automaticity, with little conscious control of movement[72]. By contrast, during *de novo* learning, task requirements are explicitly mapped to basic execution elements[21], a time-consuming process that conflicts with the demand for rapid task reproduction within a millisecond timescale. Access to subcomponents of more-abstract movement elements, which previously underpinned some features of expert task performance, is limited. Thus, once task performance has broken down, alternative motor control options are ill-equipped to immediately reinstate motor performance using new elements. Inappropriate and dysfunctional movements are likely to be produced, which are unable to match required task performance levels, marking the onset of task-specific dystonia (FIG. 5a).

Encoding of dystonic movement. If stereotyped dystonic movements are repeatedly practised, they will become encoded in a manner similar to that for any other sequence of movements. Conscious control of the dystonic movement elements declines, causing frustration for individuals with undiagnosed task-specific dystonia as they attempt to implement strategies to address their movement difficulties (FIG. 5b). This formulation might partially explain one of the most puzzling features of task-specific dystonia: why normal

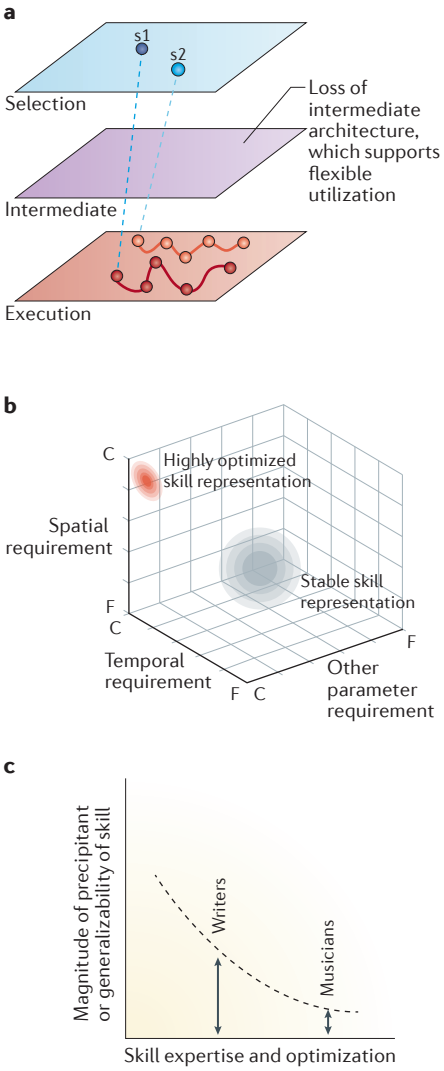

Figure 4 | **Vulnerabilities of highly skilled representations. a** | The neural architecture of overlearned skills is not well defined. Highly trained skills are represented here as a rigid, synergy-like execution pattern with limited use of an intermediate architecture that would permit the flexible utilization of motor features across other tasks. **b** | Performers have exceptionally high motor encoding requirements, and training is likely to lead to a highly optimized skill representation (red region) close to the ceiling (C) of attainable values for both spatial and temporal encoding. By contrast, stable, everyday tasks (grey region) are illustrated here at a midpoint between floor (F) and C values of temporal and spatial ranges. The cost of such optimization in performers is largely unknown. For example, a high degree of optimization might limit the degree of flexibility available to respond to new task requirements (narrow diameter of skill representation) and/or other parameters might not be optimized (shown close to F). **c** | With increasing skill expertise, the magnitude of the precipitant of task-specific dystonia decreases. In part, this association might be due to the reduced generalizability of highly optimized skill representations.

task performance, which was previously achievable, can no longer be easily reinstated. Skill representations that are activated for a particular context or performance goal could become corrupted, with dystonic movements incorporated into their architecture[57].

Psychology of motor control. Finally, the influence that misdirected cognitive influences can have on skill performance is worth emphasizing[73]. An attentional focus on the mechanics of movement rather than on the external consequences or goals of movement has consistently been shown to worsen skill performance[13]. Deterioration in function can be shown experimentally for writing (when attention is focused on hand movements rather than on output of the tool)[49] and for musical performance (when attention is focused on finger movements rather than on the sound)[74]. Personality traits seen in musicians with task-specific dystonia, such as anxiety and perfectionism, are linked to a highly attentive manner of motor control, a situation that has repercussions for both the development of the disorder and how we treat it[13]. The negative effects of self-focus are commonly discussed within the sports science literature (for example in relation to 'the yips' in golfers)[73], but these effects might be equally relevant in forms of motor impairment that share phenomenology in musicians and writers, such as motor block or 'choking' under pressure[56,73] (BOX 1). Aside from personality traits, other triggering factors such as injury, pain and explicit attempts to alter technique or performance will also naturally focus attention on the body region, to the detriment of motor control[13]. Misplaced attention can impede the normally automatic reproduction of highly skilled tasks. For a subset of patients with task-specific dystonia, this is an important mechanism through which performance can deteriorate.

Clinical implications

Prevention. Defining task-specific dystonia as a modifiable disorder of motor control has the important implication that a proportion of cases of task-specific dystonia might be preventable. Many occupational forms of task-specific dystonia are characterized by mismatches between natural capability and the tool or task requirements. Improving the ergonomics of tools and limiting task parameters that stress the motor system might be beneficial[40]. However, professional musicians and athletes cannot modify their tool or task requirements to any great extent. As such, prevention strategies that focus on the control system, maximizing the 'resilience' of relevant representations in the brain and nurturing a healthy psychological profile, could reduce the risk of task-specific dystonia. Practice plans that emphasize flexibility of motor performance should be encouraged; 'healthy' practice routines defined by musicians have a reassuring resonance within the framework we outline[41]. For example, pianists at the Moscow Conservatory are encouraged to practise on pianos with different weights, and the famous cellist Rostropovich recommended practising different versions of difficult sections and experimenting with rubato and altered emphasis (which subtly changes movement parameters) so that "the brain is relieved of the pressure of performing an action in a single rigid way" (REF. 75). Such practice techniques could feasibly consolidate intermediate-level connections and reduce the development of rigid effector representation, facilitating flexibility and resilience when any changes in task parameters are required.

Retraining dysfunctional movements. Once a motor problem has developed, a careful assessment of risk factors reveals a mechanism profile specific to that individual. This process is important owing to the great heterogeneity within this group of patients. For example, although psychological factors are likely to be influential in a subset of patients with task-specific dystonia, a considerable proportion do not exhibit any signs of anxiety, perfectionism or stress[55]. The identification of mismatches between task requirement and capacity is already a widely established tool used to pragmatically guide selection of treatment strategies within rehabilitation disciplines (the person–environment–occupation model)[76]. Techniques from the sports science literature can also be integrated into treatment plans as the role of attention in task-specific dystonia becomes increasingly appreciated. For example, focusing attention away from the mechanics of movement and onto the goals of movement can help to prevent anxiety-related blocks in performance[42,77].

Box 1 | **Features of task-specific dystonia**

Subtypes of motor impairment

- Motor fatigue
- Overuse injury
- 'Choking' under pressure
- Dynamic stereotype
- Focal dystonia

This classification (developed in musicians) emphasizes that motor failure can develop along a continuum[42]. Subtle movement degradation can occur owing to fatigue or overuse. Dynamic stereotypes are an early and modifiable movement abnormality that might develop into dystonia if risk factors persist[42].

Task specificity

At its most specific, deficits in writing particular letters or isolated musical phrases must be encoded within a representation used only for that particular context[37,56]. If the motor impairment starts to involve other tasks, the dystonic representation could also become incorporated into additional skill networks (for example, motor synergies used in playing a piano sequence might be reused when typing)[92,93]. Occasionally, the contralateral hand is affected, reflecting either modification of a new representation by persistent risk factors or recruitment of dysfunctional representations that are hand-independent (such as representations that control sequence learning[94]). An apparently task-selective deficit can be the presenting feature of other movement disorders, such as DYT1 dystonia or neurodegenerative conditions[41,95,96]. We believe this situation to reflect a threshold effect, as a lesser pathological insult is required to reveal a deficit in a skilled rather than unskilled action, and a generalized motor impairment eventually prevails[97].

Sensory trick manoeuvres

In rare cases, distorting sensory feedback (for example, by wearing a latex glove) can induce short-term performance improvements[98,99]. From a motor control perspective, the model of the skilled movement needs updating to produce an altered prediction of the sensory feedback[100–102]. Shortly after putting on the glove, the increased mismatch between predicted and actual sensory feedback might be helpful in disrupting dystonic movements through error-driven plasticity[103]. However, on continued wearing of the glove, this positive effect is likely to be lost as the corrupted motor model remains structurally unchanged despite adaptation. The period of heightened plasticity at the beginning might present a unique window for retraining in patients who respond to sensory tricks[98].

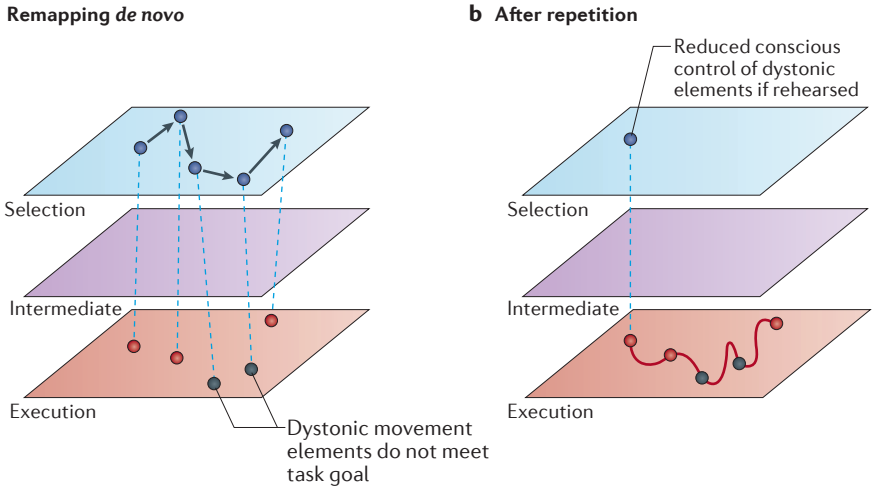

Figure 5 | **The development of task-specific dystonia. a** | If the existing representation at the execution level can no longer accommodate task requirements, novel motor control options must be sought. Solutions are likely to require mechanisms comparable to early learning states, in which task requirements are explicitly mapped to basic execution elements. Such mechanisms are ill-equipped to immediately reinstate previous levels of skill performance, which were encoded by neuronal elements optimized over many years of practice. Movements that are either inappropriate or non-physiological might start to be produced, and they can be classed as dystonic as they no longer attain task goals. **b** | If dystonic movements are rehearsed, they are likely to become encoded in a manner similar to any other learned sequence of movements, with a shift of motor control towards increased automaticity and reduced explicit cognitive monitoring of movement sequences. In this situation, dystonic movement sequences become increasingly difficult to correct.

Retraining therapies for patients with task-specific dystonia have shown encouraging results despite often including techniques based on traditional models of dystonic pathophysiology, which might not be optimal[78]. Overall, our framework predicts multifaceted interventions tailored to specific individuals' risk profiles to be the best treatment approach for task-specific dystonia. Centres offering combined therapeutic approaches report better outcomes for patients with task-specific dystonia than do centres lacking such resources[79].

Traditional dystonia treatments. Conventional dystonia treatments rarely offer adequate relief of task-specific dystonia. Oral medications (such as trihexyphenidyl) have been tried, but responses are inconsistent and their use is often limited by adverse effects[79–81]. An initial benefit from botulinum toxin injections in specialist settings is often seen, but marked variability in responses to this treatment and difficulty in avoiding disabling weakness mean that only a subset of patients continue this therapy long term[79,82,83]. It is likely that although botulinum toxin injections can treat the end point of task-specific dystonia — the inappropriate muscle contractions associated with performing a given task — they cannot in isolation address the underlying mechanisms. Similarly, deep brain stimulation and thalamotomy (which have been trialled in case series) are nonspecific in their action, and randomized controlled trials are needed before such invasive approaches can be validated[84–89]. Noninvasive brain stimulation techniques (such as transcranial magnetic stimulation), which aim to either disrupt or augment the defining physiology in a favourable manner, are increasingly being studied in patients with task-specific dystonia[9,90]. Theoretically, the most attractive study designs pair brain stimulation with concurrent task-relevant behavioural training, thereby activating the corresponding neuronal network subserving that skill[91].

Conclusions

In this Opinion article, we have presented task-specific dystonia in the context of motor skill learning in health, suggesting that they are two sides of the same coin. Our framework integrates established risk factors for task-specific dystonia with known mechanisms of motor skill learning, and describes how they might interact to disrupt the neural representation of motor skills. We hope that this perspective will help to define new directions for research and to promote much-needed therapeutic advances.

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A.S. and K.K. collated data and papers relevant to the article, developed its content, and wrote the manuscript. J.C.R. and M.J.E. contributed substantially to discussions of the article content and to review of the manuscript.

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A.S. and K.K. declare that they have no competing interests. J.C.R. declares that he has received speaker's travel costs from the Movement Disorders Society. M.J.E. declares that he receives royalties for the *Oxford Specialist Handbook of Parkinson's Disease and Other Movement Disorders* (Oxford University Press, 2008) and that he has received speaker's honoraria from UCB pharmaceuticals.

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Subject categories

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ToC blurb

000 **A unifying motor control framework for task-specific dysfunction**

Anna Sadnicka, Katja Kornysheva, John C. Rothwell and Mark J. Edwards

Sadnicka and colleagues present task-specific dystonia in the context of motor skill learning in health. Their framework integrates established risk factors for task-specific dystonia with mechanisms of motor skill learning, to indicate how disrupted neural representations of motor skills might arise.