VIEWPOINTS

Rethinking Movement Disorders

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ABSTRACT: At present, clinical practice and research in movement disorders (MDs) focus on the "normalization" of altered movements. In this review, rather than concentrating on problems and burdens people with MDs undoubtedly have, we highlight their hidden potentials. Starting with current definitions of Parkinson's disease (PD), dystonia, chorea, and tics, we outline that solely conceiving these phenomena as signs of dysfunction falls short of their complex nature comprising both problems and potentials. Such potentials can be traced and understood in light of well-established cognitive neuroscience frameworks, particularly ideomotor principles, and their influential modern derivatives. Using these frameworks, the wealth of data on altered perception-action integration in the different MDs can be explained and systematized using the mechanism-oriented concept of perception-action binding. According to this concept, MDs can be understood as phenomena requiring and fostering flexible modifications of perception-action associations. Consequently, although conceived as being caught in a (trough) state of deficits, given their high flexibility, people with MDs also have high potential to switch to (adaptive) peak activity that can be conceptualized as hidden potentials. Currently, clinical practice and research in MDs are concerned with deficits and thus the "deep and wide troughs," whereas "scattered narrow peaks" reflecting hidden potentials are neglected. To better delineate and utilize the latter to alleviate the burden of affected people, and destigmatize their conditions, we suggest some measures, including computational modeling combined with neurophysiological methods and tailored treatment. © 2024 International Parkinson and Movement Disorder Society.

Key Words: movement disorders; ideomotor theory; metacontrol; hidden potential

In clinical practice and research, actions and behaviors are currently characterized as "normal," that is, within a range of optimal performance versus "disordered"/dysfunctional, that is, deviating from "normality" in certain ways. Medical advice and treatment goals for movement disorders (MDs) are predominantly focused on means to "normalize" movements. In the current article, we argue that this dominating view is partly misleading because it systematically hinders the development of a more holistic perspective on MD, according to which motor problems in these patients may lead to or are associated with hidden potentials (Table 1) that are currently often not considered in clinical research and practice.

Arguments to complement research and practice of MD could be derived not exclusively from motor features and motor physiology per se but rather from well-supported cognitive science concepts linking motor processes, perception, and cognition. This may lead to a more balanced view on MD and deficit-oriented views dichotomizing between normal and abnormal or health and disease by emphasizing continuous dimensions of MD, where individuals with MD have currently unrecognized potentials (Table 1). Of late, this is, for instance, reflected by suggestions to refer to those affected by Parkinson's disease (PD) as persons with PD rather than PD patients.[28] However, it must be emphasized that such a concept, as well as the present review,

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TABLE 1 *Examples of hidden potentials of movement disorders*

Condition	Example	Explanation/interpretation	Practical implication
Parkinson's disease	Amelioration of freezing by external cues or altering proprioceptive information.[1]	Flexible use of perceptual input to generate and maintain coordinated movements.	Using transversely placed lines on the walking lane, geometrical visual patterns, or laser beams to overcome freezing.[1]
	Ability to run swiftly when chasing a ball, eg, during basketball.[2]	Flexible and rapid switch to activation of fast locomotor circuits when those primarily mediating walking are deficient.[2]	Physiotherapy focusing on external cueing.
	Straightening of stooped posture when closing eyes.[3]	Usage of visual feedback to actively assume a stooped posture as protection from falling backwards.[3]	
	"Kinesia paradoxa," ie sudden transient ability to perform a task a person was previously unable to perform,[4] eg, ability to run when threatened by fire or an imminent accident.	Flexible handling and rapidly switching of perception-action associations.	Using kinesia paradoxa as a rehabilitation tool[5]; consider virtual reality tools to prevent habituation.[6]
	Surprising resilience, eg, increased creativity or major physical accomplishment, eg, long-distance cycling.		Promoting contests
Dystonia	Taking the trunk rather than the head midsagittal line as a reference when judging the visual vertical and visual straight ahead (cervical dystonia).[7]	Adjustment of postural control by shifting postural references from the head to the trunk when abnormally held, involuntarily moving neck has become an unreliable reference.[7]	
	Sensory "tricks" leading to normalization of body postures, eg, touching specific parts of the face, cheek, chin, or other parts of the face or neck, raising the arm and holding the finger near the target region without touching the face, tightly strapping a backpack in cervical dystonia (Fig. 1).[9]	Flexible, dynamical use of perceptual information and re-contextualization of perception-action couplings.[9]	Developing tools/applications on the basis of reported sensory "tricks"[9]; blocking muscle afferents or cooling affected body part.[12-14]
Chorea	Increased variability and excessive grasping forces during precision grip experiments (Fig. 1) (Huntington's disease).[10,15]	Flexible increase of force compensates for variations in grip force creating a larger safety margin to ensure contact with the object to be lifted or pulled and attenuates the consequences of choreic movements to prevent slips during task execution.[10,15]	Physiotherapy or occupational therapy to further foster adaptive potentials.[16]
	Voluntary suppression of chorea on demand in some people with chorea.[17,18]	Flexible use of proactive and reactive inhibition.	Developing strategies of flexible inhibition as part of cognitive-behavioral training.

(Continues)

TABLE 1 *Continued*

Condition	Example	Explanation/interpretation	Practical implication
Tics/Tourette syndrome	Propensity toward artistic domains and professional fields, in which creativity is required,[19] reflected by higher scores in some dimensions assessed by creativity tests[20]; tics/Tourette might be more common in some groups of performing artists, eg, singers.[21]	Increased cognitive flexibility, particularly when being examined under time pressure[20,22]; increased habit learning tendencies[23]; and experimental evidence for increased perception-action binding.[24–26]	Counseling with a view to professional training; using adapted behavioral treatments aiming at focusing on external stimuli, eg, attention training.[27]

is in no way intended to downplay the burden of persons affected by MD that often have signs of progressive neurodegenerative diseases or to romanticize their potential. On the contrary, the motivation to broaden the view on the manifold consequences of MD, including possible potentials, is to reduce stigmatization and improve the quality of life of affected persons.

In addition to PD, typical examples of MD are dystonia, chorea, for example, in Huntington's disease (HD), and tics, for example, in Tourette syndrome. PD is predominantly characterized by bradykinesia, that is, slowness or paucity of spontaneous movements, in combination with either rest tremor or rigidity, that is, velocity-independent increased muscle tone, or both.[28] In addition, body posture is typically stooped, and gait is characterized by small steps (Fig. 1). People with dystonia feature sustained or intermittent muscle contractions causing abnormal, often repetitive, movements, postures, or both, with dystonic movements typically being patterned and twisting (Fig. 1).[29] Chorea, a cardinal sign in HD, is characterized as a hyperkinetic continuous non-patterned MD typically appearing flowing and jerky with speed being variable and timing and direction unpredictable,[30] sometimes giving the impression that the affected person is dancing (Fig. 1). Tics are repetitive, often patterned action fragments resembling spontaneous movements but appearing exaggerated (Fig. 1).[31–34] In the present article, taking signs of

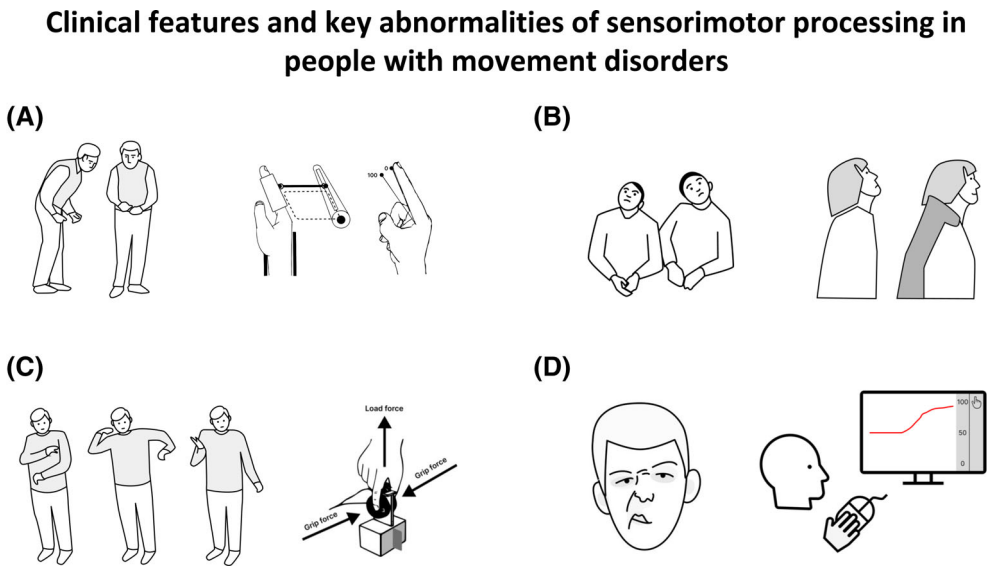

FIG. 1. Characteristic clinical features and key abnormalities of sensorimotor processing in people with movement disorders are illustrated. (**A**) In *Parkinson's disease* (PD), in addition to bradykinesia, rigidity, and tremor, a stooped posture is typical. Using an apparatus, where a finger holder is moved by a motor handle, so that the finger follows the angle of the motor handle, the perception of finger displacements matched to visually presented finger displacements, that is, kinesthesia, was reduced in people with PD (adapted from[8]). (**B**) In people with *dystonia*, agonist–antagonist co-contractions cause abnormal posture, for example, torti-, latero-, or retrocollis. Sensory "tricks," for example, strapping a backpack to the trunk, altering sensory input, can lead to considerable improvement or even normalization of posture (adapted from[9]). (**C**) Continuous flowing movements in *chorea* give the impression that the affected person is dancing. The intricate interplay of tactile afferent input and motor output as a prerequisite of holding and manipulating objects during precision grip has been shown to be disturbed in people with chorea (adapted from[10]). (**D**) *Tics*, that is, repetitive, often patterned movements resembling spontaneous movements but appearing exaggerated often affect the face, for example, causing facial grimacing. As a sign of altered perceptual-motor interaction, tics are typically preceded by an urge to move, which can be captured using psychophysical measurements, where the intensity of urges is recorded by operating a scroll bar using a mouse pad to indicate the intensity of the current urge (adapted from[11]). [Color figure can be viewed at wileyonlinelibrary.com]

PD, dystonia, chorea, and tics as starting points, we will outline that solely referring to these phenomena as signs of disease or dysfunction that "simply" need to be eliminated falls short of their complex nature comprising both problems and potentials and will do so by taking a cognitive science stance incorporating motor and perceptual processes and their interrelatedness.

Ideomotor Concepts—A Basis to Change Views on MDs

Based on cardinal clinical features and definitions, MDs are conceived as prime examples foremost of motor dysfunction. Importantly, though, motor processes are closely linked to and cannot be understood in isolation from perceptual, particularly sensory processes, which are often considered only to represent the input to motor processes.[35] However, sensory and motor processes are interdependent.[35,36] For instance, if a person grasps a cup of coffee, the features of the movement, the stimulus (the cup), and the effect become associated (eg, the tactile feedback touching the cup, the visual feedback that the hand actually holds the cup, and finally the smell and taste of coffee). A common theme underlying such seemingly self-evident natural scenarios, often strongly impaired in MD, is that there are continuous contingencies between activities and their impact on the environment.

Although MDs have traditionally been conceived and categorized as motor disorders, it has, in fact, become clear that perceptual aspects, particularly somatosensory processing and sensorimotor integration, are affected in many ways in nearly all MDs.[24,37-40] However, despite the wealth of evidence for the role of both motor and perceptual processes in MDs, their intricate interplay has rarely been conceptualized. Here, we propose that ideomotor principles[35] (see Box 1) and influential derivatives such as the "Theory of event coding (TEC)"[41,42] (see Box 2) are very useful cognitive frameworks to better understand MD and to fathom their (hidden) potentials (Table 1).

Ideomotor concepts date back to philosophers from the 19th century arguing that humans and animals establish bi-directional links between perception and action (Box 1; Fig. 2).[35,42] Ideomotor theory is more than what is currently considered in clinical practice. An example is ideomotor apraxia, defined as an inability to transfer an idea of how to act into a movement. Even though the original term, stemming from the Greek word "*idea*" (= *form*) and the Latin word "*motare*" (= *to move about*), implies some willfully prepared aspects of motor outputs that are captured by ideomotor principles, ideomotor principles are, in fact, not restricted to willfully prepared movements. This is important concerning MD, where symptoms cannot be regarded as an instance of willfully controlled behavior. Ideomotor principles even apply to simple reflexes.[41] When motor neuron activity (M) leads to muscular activity and movements, this provides sensory (kinesthetic) feedback by activating afferent neurons (K). If this happens only once, not much will follow.

Box 1. Ideomotor principles

Continuously picking up contingencies between own activities and their impact on the environment (ie, to integrate perceptual and motor processes) is the core of the so-called ideomotor theory and central to the ability to act on purpose.[35,41,42] The central assumption of the ideomotor theory is that for an action to be accomplished adequately, one first has to anticipate the perceptual effect the action will produce.[35] Action-learning enables one to understand the relation between movements and their perceptual effects, which leads to the ability to act on purpose, that is, to anticipate particular (perceptual) effects a future action will produce and hence to select and initiate actions according to their outcomes in relation to current goals.[43] Put differently, humans learn to act intentionally; they are able to "imagine" (ie, internally activate the representation of) a wanted outcome and can act on purpose to achieve goals.

Box 2. Theory of event coding (TEC)

The principle idea of the theory of event coding is that perceived and produced events (stimuli and responses) are cognitively represented in so-called event files, that is, episodic representations of events, and that these representations interact to generate all kinds of perceptions and actions.[42] TEC is a general framework that explains the modulation of action due to retrieval of previously established event files. In addition, the planning and selection of actions due to anticipated action effects trigger the motor programs that were formerly integrated with these effects in event files. The latter can thus be defined as internal representations of stimuli, responses, and effect features. The concept of an event file follows the tradition of Kahneman's and Treisman's object files (that consisted only of stimulus features).[44] In an event file, all features describing a stimulus (S), the associated motor response (R), and the likely effect (E) are stored in a way that each stimulus feature (S) becomes associated (bound) with each feature defining the response (R), and its effect (E).

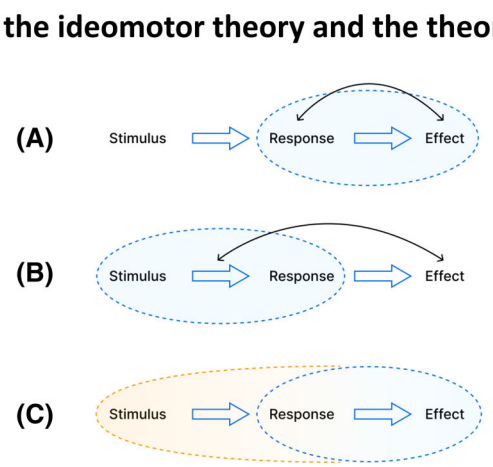

FIG. 2. Principles of the *ideomotor theory* and the *theory of event coding* are shown. (**A**) Any movement, for example, a response to a given stimulus, has effects, including proprioceptive feedback and changes in the environment, which are considered during motor planning continuously creating connections between own activities and their impact on the environment. If certain movements are repeated, motor activations and their effects such as perceptual information become associated. Importantly these associations are bi-directional. (**B**) Responses, including their consequences, are automatically re-activated when stimuli of previously established response-effect associations are re-encountered. (**C**) The core idea of the theory of event coding is that following their association stimuli, responses and effects become coupled, or bound, and are integrated into integrated sensorimotor bindings, which in the theory of event coding are conceptualized as event files. [Color figure can be viewed at wileyonlinelibrary.com]

Importantly, however, in MD, the presence of motor symptoms over a long time period provides a suitable basis for learning and neural plasticity principles to become effective between motor and sensory processes. Ideomotor concepts are based on Hebbian learning principles. If the activation of M regularly leads to the activation of K, trace conditioning creates an association between M and K, following the Hebbian principle that what "fires together wires together."[41] Consequently, M and K become closely associated (bound) with each other (Fig. 2). Obviously, this logic is not restricted to kinesthetic feedback but can involve any sensory system feedback. Therefore, aspects of any perceptual consequence or effect of movements/MD can become integrated with the motor activity producing this movement. Influential conceptualizations of these processes—rooted in cognitive science—have delineated that such associative processes establish a pattern of interconnected perceptual and motor elements[41,42,45] that is essential for coherent perception, action, and the integration of perceptual and motor processes. This is also often referred to as the "neural binding problem"[46,47] describing how processes that are encoded by distinct brain circuits can be combined for perception and action (including basic motor processes).

In this respect, particularly basal ganglia-thalamo-cortical loops are of relevance. They constitute key relays of motor and perceptual information processing and are altered in MD.[48] Several lines of evidence suggest that basic visual inputs enter the basal ganglia networks[49,50] via dorsal midbrain structures (ie, the superior colliculus).[50] Similarly, there is input from somatosensory and auditory cortices.[50] Convergent motor and perceptual input to the basal ganglia provides a necessary prerequisite to couple motor and sensory processes and may therefore set the ground for ideomotor principles to emerge.

Crucially, associations built between motor and perceptual processes as the central mechanism behind ideomotor principles are bi-directional. Such a bi-directional relationship between perception and action is the central functional principle of TEC and its "event file" concept (Box 2; Fig. 2). Event files describe the close association between perceptual and motor processes.[45] As outlined later, functional principles of "event files" also underlie MD. Before we detail the consequences and repercussions this may have, we review that there is ample evidence supporting an ideomotor principles perspective on MD.

Sensory and Sensorimotor Puzzles in Different MDs

Somatosensory processing and sensorimotor integration are increasingly recognized as important components in MD.[32,38-40] For instance, in people with PD, the ability to distinguish the shape, orientation, and texture of an object by active touch of a surface and manipulation of an object in space is impaired, providing evidence for defective proprioceptive processing in them.[51] Also, people with PD have elevated thresholds for detecting limb position[52] and also passive motion independent of dopaminergic medication.[53] Importantly, in PD, kinesthesia, that is, the sense of motion, is also reduced (Fig. 1).[8] At first sight, this is puzzling

because the underestimation of limb movements is expected to lead to an overproduction of spontaneous or voluntary movements and not hypokinesia as observed in PD. However, the findings can be interpreted such that in PD, both motor command/corollary discharge during performance and kinesthetic feedback are proportionately reduced, implying that the sensorimotor apparatus is "tuned down" in them.[8] Impairments in gait and balance in PD[54] are likely multifactorial, arising from altered multimodal integration of perceptual information, including vestibular, visual, and proprioceptive input.[3,55] A typical example is freezing of gait, that is, an inability to move, which may suddenly occur, particularly in narrow spaces both in real situations and in virtual reality environments.[56]

In dystonia, there is ample evidence for altered central perceptual processing[57] and altered cortical representation of perceptual, particularly sensory information,[58] which is illustrated, for example, by the clinical phenomenon that applying vibration to the hands of people with focal limb dystonia or graphospasm can induce or worsen dystonic movements.[12,59] Dystonic arm tremor is often position-specific, becoming worse in certain positions,[60] underscoring the role of proprioceptive input.

In HD, typically manifesting with chorea in adults, sensory symptoms and signs are usually not part of the clinical presentation, or may be overlooked. Also, pain is probably underreported.[61] A classical finding in people with HD is reduced amplitude of the early components of sensory-evoked potentials,[62,63] probably reflecting the abnormal transmission of perceptual information from the basal ganglia–thalamic complex to the sensory cortex. They also have abnormal electromyography responses to muscle stretch, with long-latency reflex components being absent or reduced in the hand and forearm muscles.[62,63] In addition, suppression of the startle reflex is reduced in them[64] suggesting a disruption of subcortical sensorimotor gating that is also shown in the auditory modality.[65,66] Also, people with HD have specific deficits in grasping and transporting objects, particularly as regards the timing and magnitude of precision grip, indicating deficits to process-relevant tactile afferent input (Fig. 1).[10,16,67]

In Tourette syndrome as a prototypical tic disorder, sensory phenomena are a core feature.[68] First and foremost, abnormalities on a perceptual level are evidenced by urges associated with tics (Fig. 1)[11,68] and hypersensitivity to external stimuli,[69] for example, washing instructions or stitching in clothing, which altered sensory perception thresholds cannot explain because quantitative sensory testing[70] is normal in them.[71] Hypersensitivity to external stimuli, therefore, likely reflects alterations in central processing of perceptual information. This is corroborated by experimental data. Thus, afferent inhibition of a transcranial magnetic stimulation pulse administered to the motor cortex by peripheral electrical stimulation is reduced in Tourette syndrome[72,73], and "pre-pulse inhibition" of the blink reflex is attenuated,[74] suggesting deficient sensorimotor gating.[75,76] Another striking feature in people with Tourette syndrome is echo phenomena, that is, automatic repetitions of other people's movements (echopraxia) or sounds/words (echolalia).[77,78] They tend to imitate the movements or gestures of people surrounding them and imitate others' tics.[78] Also, tic frequency is higher when these people watched themselves ticcing in a mirror[79] or when paying attention to their tics as compared to paying attention to non-tic finger movements.[80] This illustrates that in Tourette syndrome, the activation of action effects, that is, seeing oneself or someone else performing an already-learned behavior, readily activates motor tendencies to perform the act that produces this effect.[81]

These examples suggest that perceptual signals modulate MD and that abnormal sensorimotor integration contributes to altered motor control. The numerous puzzle pieces call for an explanatory concept encompassing motor phenomena and underlying processes, and perceptual processing.

An Integrative Ideomotor Principle View on MD—Unraveling Hidden Potentials

Surprisingly, the wealth of clinical and neurophysiological data on people with MD documenting that the relation between motor output and perceptual processing is altered in many ways in them has so far not been used to conceptualize MD using ideomotor principles. Using the ideomotor framework (Box 1), specific characteristics and problems of different MDs could be described in a common language enabling to formulate specific hypotheses as to the underlying mechanisms and problems.

According to the ideomotor theory, actions are partly represented by their perceivable effects (eg, sensory or visual effects). Activating these action effects, for instance, by imagining or anticipating an action or seeing someone else performing an already-learned behavior[82] fosters motor tendencies to perform the act that produces this effect.[35] The TEC as a more modern descendant of the ideomotor theory proposes that perception and action are not distinctly represented but are stored in common "event files,"[42] which, as outlined earlier, specify how perceptual and motor processes are interrelated (Box 2; Fig. 2). In the following, we outline how perception-action abnormalities in people with MD could be integrated into ideomotor thinking.

Box 3. Metacontrol concept

People can vary in their cognitive processing style on a dimension characterized by its two poles: "persistence" and "flexibility."[83-86] Whereas a high degree of persistence corresponds to the original idea of cognitive control as willpower with a strong focus on one goal and the information related to it, a high degree of flexibility is characterized by a more integrative, less selective, and exclusive processing style, which facilitates switching between tasks, ideas, and actions, and taking into consideration a broader range of possibilities.[84,87] The metacontrol model follows a bipolar logic: cognitive control arises from an effective, situationally adequate equilibrium between the opposing poles of the persistence-flexibility dimension. This equilibrium resembles a trade-off between these opposing poles in a way that a more stable cognitive persistence comes at the cost of cognitive flexibility, and the opposite holds for enhanced cognitive flexibility, which comes at the price of cognitive persistence.

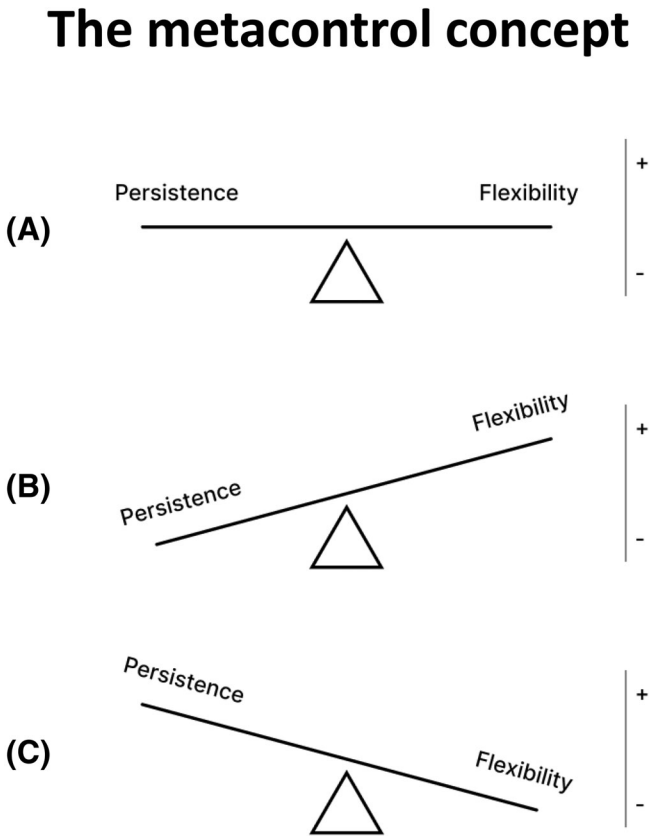

FIG. 3. Illustration of the *metacontrol concept*. (**A**) Cognitive processes subserving goal-directed behavior, including perception-action integration, need to be balanced between persistence and flexibility. (**B**) A deflection toward persistence subserves the attainment of long-term goals through shielding intentions from competing goals, thus avoiding premature goal shifts rendering behavior disorganized. (**C**) A flexibility-leaning metacontrol state fosters rapid adaptation in a changing and unpredictable environment.

Before detailing this, it is important to consider that there are different "styles," in which cognitive operations linking perception and action, as well as adaptive behavior, are carried out.[83] These can be detailed in a "metacontrol" concept (Box 3),[83,86,88,89] describing that all aspects of perception-action integration processes subserving goal-directed behavior require a dynamic, context-sensitive balancing of different subprocesses (Fig. 3). On the one hand, to attain long-term goals, it is necessary to shield intentions from competing goals to prevent premature goal shifts, rendering behavior disorganized. In other words, at least some *persistence* is required. On the other hand, in a changing and partly unpredictable environment, also *flexibility* is necessary to adapt behavior. Thus, *persistence*- and *flexibility*-leaning metacontrol states influencing perception-action integration processes have to be balanced (Box 3; Fig. 3).[83,86,89] This is modulated by dopamine and (mesofrontal and nigrostriatal) dopaminergic pathways.[90] For instance, there is evidence that dopamine levels and also COMT- or dopamine transporter-gene polymorphisms modulate cognitive flexibility[91,92]; that is, the dopaminergic system relevant for the emergence of MDs is also important for metacontrol processes.

In people with PD, defective proprioceptive processing has emerged as a common theme.[51-53] According to ideomotor principles, there is an intricate relationship between proprioceptive information and motor planning and control.[93] Inaccurate information about current limb positions and motion in PD likely affects motor performance and vice versa. Thus, on the one hand, distorted/unreliable proprioceptive information may systematically cause movement errors in the spatial and time domain, ultimately leading to hypometric and slow movements. On the other hand, in a bradykinetic state, expectations regarding sensory consequences of an action are violated; that is, the degree of change of afferent input is reduced compared to the physiological state. In other words, the association between the planned action, along with its anticipated afferent consequences, and the actual afferent feedback, becomes more variable increasing motor demands.

Interestingly, there is evidence for increased sensitivity to the energetic demands of movements in PD.[94] This rather than an inability to move fast has been suggested as an explanation for reduced movement vigor, that is, the likelihood of moving at a certain speed.[94] In the context of TEC, it is to be expected that the binding between perceptual elements and actions will become less reliable and will therefore be reduced in PD. In a metacontrol context, it is likely that the handling of perception-action integration processes needs to be and,

in fact is, more *flexible* in PD. Examples are given in Table 1.

In dystonia and chorea, chronically abnormal postures (dystonia) and aberrant and variable repetitive movements (dystonia and chorea) lead to distorted relations between motor plans, including expected afferent feedback and actual perceptual information, particularly proprioception. Finely tuned perception-action relations in previously established event files will, therefore likely, become unreliable and disrupted. This may lead to novel but dysfunctional perception-action couplings as exemplified by usually neutral vibratory stimuli applied to a limb inducing or worsening abnormal movements in dystonia.[12,59] Probably as compensation, perceptual information that has become unreliable is not physiologically "used" as in pre-established event files but instead ignored or blocked. For instance, it was shown experimentally that the gain, or central weighting, of vestibular input to higher-order systems controlling neck position is reduced in cervical dystonia[95-98] as if vestibular information was ignored or not fully integrated during motor programming, probably because constant involuntary head movements render vestibular signals unreliable so that these are attenuated centrally. In patients with chorea as a manifestation of HD, vibration-induced activation of the globus pallidus and parietal as well as frontal areas has been shown to be reduced in a PET study[99] and processing of perceptual information to be attenuated.[99-101]

These examples document the weakening of perception-action binding in people with dystonia and chorea. In a metacontrol context, such weakened binding will likely also increase the flexibility of perception-action processing (see Table 1; Fig. 1).

In contrast to PD, dystonia, or chorea, relations between perceptual information and motor output have not become distorted in people with tics. Instead, they are tighter and more reliable because the same tic is carried out repeatedly and can thus be considered as a novel event with very constant and presumably increased perception-action couplings resembling habits. There is indeed evidence that habit formation tendencies are increased in people with Tourette syndrome.[23] Also, there is experimental evidence that perception-action binding is increased in them[24-26] with the strength of perception-action binding positively correlating with tic frequency, suggesting that increased binding strength represents a core feature related to the pathophysiology and the dominating motor phenotype of Tourette syndrome.[24] The finding that people with Tourette syndrome show event file hyperbinding[24] implies that they are also in a metacontrol state leaning toward cognitive flexibility.

Such hyperbinding is reflected by increased reaction times and higher error rates during stimulus–response reconfiguration processes,[24] probably as a consequence of higher interference by task-irrelevant information, which is a characteristic of a metacontrol state toward flexibility rather than persistence.[102] This is the case because more top-down control sustaining an effective focus on one goal and relevant information leads to irrelevant (ie, not goal-compatible) information being discarded, reducing time-consuming re-configuration processes during event file binding. Thus, less top-down control or a metacontrol state toward the more flexible end is then accompanied by more interference and increased time-consuming event file re-configuration. Indeed, people with Tourette syndrome show increased cognitive flexibility, particularly when being examined under time pressure, compared to typically developing controls (see also Table 1).[20,22,103]

Taken together, using ideomotor principles and a "TEC-language," it can be concluded that event file binding is likely reduced in people with PD, dystonia, and chorea but rather increased in people with tics. Importantly, as regards *persistence-* versus *flexibility*-leaning metacontrol states, all MDs considered here appear to be in states of increased flexibility fostering adaptive potentials.

Deficits and Potentials Viewed in the Net Zero-Sum Framework

When assessing and weighing deficits and hidden or overt potentials in MD, it is important to consider one of the most central mechanisms in the (human) brain, that is, "competition,"[104,105] according to which circuits compete and give rise to a net zero-sum framework of brain functions.[106-108] This framework draws on a fundamental physical principle of any closed system referring to the first law of thermodynamics, where losses in some functions are balanced by gains of others, and vice versa. Because the brain operates under the constraint of a finite amount of energy and processing capacities, any loss of function will lead to at least some gain of function so that fundamental physical principles of closed systems are not violated.[109,110] This fundamental physical principle of a closed system gives rise to the assumption of disease-associated gains of neural function.[106,107] As outlined earlier, a potential gain of function in people with MD may be increased flexibility (see also Table 1).

Altered activity in frontostriatal loops is a core abnormality in MDs. Parallel dopamine-modulated frontostriatal loops involving *direct* and *indirect pathways* project from specific cortical regions to specific sections of the striatum and thalamus with loop projections back to the cortex.[111-113] Dysfunctions in balancing the direct and indirect pathways, well-established in MD, are examples of the net zero-sum principles. Moreover, several lines of evidence show

that processes at the level of the basal ganglia can be described by a "winner-take-all" mechanism.[114-116] In biologically constrained computational models of basal ganglia information processing, these mechanisms are modeled as the action of striatal medium spiny neurons and thus fundamental microstructural units underlying the pathophysiology of many MDs.[117] Consider the observation that deep-brain stimulation in PD alleviating motor symptoms can be associated with some decline in cognitive functions.[118-120] Such findings underscore the relevance of competition between circuits and ultimately between brain functions. Such net zero-sum framework principles of brain function are directly reflected by metacontrol[107] and related accounts of cognitive functions routed in biophysical considerations,[121-123] according to which representations of goals compete with each other.

The working principle can be envisaged as valleys and peaks in a landscape or islands and trenches in the sea reflecting patterns of neural activity. When there is a dominant representation (eg, behavioral tendency or cognitive mode), this corresponds to a fixed state with low energy at the bottom of the valley,[121] akin to a ball that rolls into the nearest valley (Fig. 4). It is difficult to move the neural systems' state out of that attractor to get the ball off the valley bottom. Likewise, people with MDs are perceived as being caught in a state of deficits. Crucially, however, any trough in the "neural activity landscape" must, according to the net zero-sum principles of brain function, be accompanied by a "peak." Such peaks can be conceptualized as hidden potentials. Critically, though, peak states are fragile—consider balancing a ball on your fingertip—and even the slightest perturbations of the system can cause the system to return to a prevailing state of deficits, that is, the attractor in the landscape. This notwithstanding, peaks exist but may be elusive or ill-defined, or both, in current clinical practice/research of MD, that is, scattered narrow peaks in a landscape of deep and wide attractors. As outlined earlier, there are several instances of increased flexibility representing functional peaks, that is, superior performance in MD, which are not only conceptually appealing but also practically relevant in terms of tailored treatment (Table 1).

Designing studies to further uncover, quantify, and utilize hidden potentials is not an easy task. It is important to precisely define functions that reflect such peaks/hidden potential. In this respect, computational modeling combined with neurophysiological recordings in tasks examining cognitive functions could be useful. There are a number of neurobiologically realistic computational models of frontostriatal loops.[124] They can be used not only to systematize data but also to generate new hypotheses as to where to look for possible hidden potentials.[125] Such models are particularly relevant for a better understanding of MDs.[126,127] Neurobiologically realistic modeling of frontostriatal mechanisms can be combined with neurophysiological investigations to detect and to explain paradoxical increases in cognitive performance, for example, in patients with chorea,[65,128-130] but also those with dystonia.[131] For instance, fine-grained cognitive neurophysiological investigation of specific cognitive functions thought to be globally compromised in later stages of HD has documented cognitive peaks, for example, superior performance in an auditory signal detection task.[65] The hypotheses-generating potential of computational models could guide the search for pathophysiologically relevant hidden potentials in MDs. Ideomotor theory and metacontrol concepts can

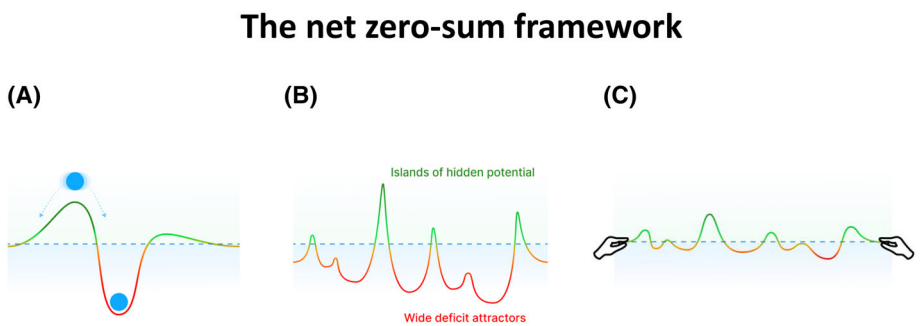

FIG. 4. Principles of the *net zero-sum framework* are shown. The principle can be illustrated as islands and trenches in the sea reflecting patterns of neural activity. (**A**) According to fundamental physical principle of any closed system referring to the first law of thermodynamics, states of low energy/activity depicted as trenches in the sea are accompanied by states of high energy/activity shown as islands, and vice versa, with the balls symbolizing a system's predominant state at a given time point and the dashed line, the "sea level," the average or "normal" activity state. (**B**) People with movement disorders are commonly perceived as being more or less continuously caught in abnormal deficit states, wide attractors, corresponding to trenches under the sea level. However, trenches in the "neural activity" sea must, according to the net zero-sum principles of brain function, be accompanied by "islands," that can be conceptualized as hidden potentials. Peak states, though, are fragile; that is, islands may be small and may be overlooked. (**C**) Management of people with movement disorders aims at normalizing functions/activity, that is, getting them out of the trenches or lifting the floor. However, according to the net zero-sum framework, effective treatment will not only affect low activity states but instead inevitably also high activity states, symbolized by the hands stretching the activity curve, which leads to lifting of the trenches, that is, improvement of deficits, *and* flattening of the islands, that is, attenuation of hidden potentials. [Color figure can be viewed at wileyonlinelibrary.com]

provide clues to these functions and inform about the starting point in computational modeling and neurophysiology. Importantly, re-thinking of disorders in terms of their hidden potential has already commenced for some psychiatric disorders.[132]

In the clinical course of MDs, particularly those associated with progressive disease, chances to identify and utilize hidden potentials, that is, functional peaks, are probably particularly high in early disease stages. Monogenic forms of MDs offer a unique opportunity to study hidden potentials in the earliest disease phases prior to clinical manifestation in individuals at high risk of developing the condition in question based on a causative pathogenic variant. These include premanifest monogenic PD[133] or HD,[134] where compensatory mechanisms can be identified that decrease across time when brain networks become increasingly dysfunctional. In other words, when the "engine is falling apart," peak activity may also expire. However, realizing the limited life span of peaks should not discourage but rather intensify the search for their discovery.

Conclusions

People with MD along with being impaired in different ways also have potentials that can be conceptualized using ideomotor principles and formulated in a "TEC-language" with perception-action binding likely being reduced in people with PD, dystonia, and chorea but increased in people with tics. In a metacontrol framework with *persistence*- versus *flexibility*-leaning states, all MDs represent states of increased flexibility, which can be taken as starting points for fostering adaptive treatment strategies. According to fundamental physical principle, MDs are good examples of brain states where gains in others balance losses in some functions. This is an incentive to further search and delineate these (as yet) hidden potentials.

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