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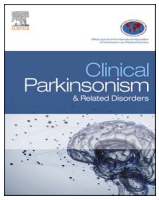

Pathophysiology of dystonia: Through the lens of Mark Hallett☆

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ABSTRACT

Dystonia was originally framed as a basal ganglia disorder, yet the mechanisms linking abnormal basal ganglia output to its defining clinical features have remained uncertain. Over four decades, Mark Hallett approached this problem by posing deceptively simple mechanistic questions and addressing them through careful physiological investigation, progressively reshaping the conceptual understanding of dystonia. Across these investigations, the concept of dystonia evolved from abnormalities of muscle activation toward broader concepts of motor control and network dysfunction. Emerging evidence suggested that dystonia reflects abnormal regulation within distributed sensorimotor networks, where impaired inhibition, abnormal premotor influences, altered sensory modulation, and dysregulated plasticity interact across cortical and subcortical circuits. In this framework, dystonia is not simply excessive movement, but a failure of the mechanisms that normally refine and shape motor behaviour. At the same time, Hallett consistently emphasized the heterogeneity of dystonia and cautioned against overgeneralization. By considering individual dystonic entities on their own terms, his work provides more than a catalogue of physiological findings: it offers a coherent framework for understanding how abnormal physiological processes may become acquired and stabilized within motor networks. The persistence of dystonia despite diverse treatments suggests that the decisive factor may lie not in outward phenomenology, but in the durability of network modification itself. Understanding how such pathological motor memories are maintained—and how they might be safely reversed—remains central to future therapeutic progress.

1. Introduction

Dystonia has long remained what Mark Hallett himself described as a mysterious disease: a disorder capable of producing striking and often bizarre involuntary movements, yet leaving the underlying neural mechanisms obscure [1]. Although traditionally framed as a basal ganglia disorder, the pathophysiological link between abnormal basal ganglia output and the defining clinical features of dystonia—co-contraction, motor overflow, loss of selectivity, and task specificity—was never self-evident. Over more than four decades, Hallett did not simply refine the description of dystonia [2,3]; he fundamentally contributed to the evolution of its conceptual framework by interrogating its physiology.

In the 1980s, his work began to redefine dystonia as a disorder of motor regulation rather than posture alone. Electromyographic (EMG) analyses of focal dystonia revealed patterns of co-contraction and impaired selectivity that pointed toward abnormal motor control [4]. Reflex studies demonstrated reduced reciprocal inhibition and altered brainstem inhibitory mechanisms across phenotypes [5,6]. These findings, present at multiple levels of the neuraxis and sometimes beyond clinically affected regions, suggested that dystonia reflected abnormal regulation of motor circuits. Even then, Hallett emphasized that abnormalities observed at spinal or brainstem levels were unlikely to represent primary pathology, but rather the downstream expression of dysfunction arising within higher-order motor networks [7].

During the 1990s, his attention increasingly turned to cortical and sensorimotor physiology. The distinction between sensory and motor systems, he argued, was in many respects more conceptual than biological [1]. Clinical phenomena such as the sensory trick, the induction of dystonic posturing by vibration, and the modulation of symptoms by peripheral afferent input challenged a purely motor interpretation of dystonia. Primary sensory modalities remained intact, yet their integration into motor output appeared unstable. Dystonia emerged not simply as excessive movement, but as a disorder of how movement is shaped by sensory context.

In subsequent decades, this physiological inquiry expanded to

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encompass motor preparation, cortical inhibition, maladaptive plasticity, and distributed network interactions [8,9]. At the same time, Hallett consistently cautioned against overgeneralization [10]. Dystonia comprises clinically diverse syndromes, ranging from task-specific focal forms such as writer's cramp (WC) to early-onset genetic dystonia such as *TOR1A*-related dystonia, which differ in age at onset, anatomical distribution, and task dependence. These differences suggest that dystonia cannot be attributed to dysfunction within a single structure but instead reflects a distributed network involving basal ganglia, cerebellum, and sensorimotor cortex [9]. As a result, physiological findings may represent predisposing traits, correlates, compensations, or epiphenomena rather than a single unifying defect. Even as recurring principles—impaired inhibition, altered sensorimotor integration, and abnormal plasticity—became apparent, dystonia resisted reduction to a single structure or mechanism.

Given the scale of his contributions, we do not attempt a comprehensive review. Instead, we focus on selected conceptual milestones representing major paradigm shifts that best illustrate the chronological evolution of Hallett's physiological understanding of dystonia and their influence on current concepts of the disorder. We adopt a question–answer structure, reflecting an approach that was characteristically his: posing deceptively simple mechanistic questions and addressing them through careful physiological investigation. Revisiting this conceptual evolution remains relevant not only from a historical perspective but also because it provides a framework for interpreting current questions in dystonia research and understanding how diverse physiological observations may converge within broader network models of the disorder.

2. Where in the act of movement does dystonia truly begin?

The earliest physiological investigations did not portray dystonia as a problem of strength or tone, but as a failure of motor selectivity. In patients with focal hand dystonia (FHD)—particularly task-specific dystonia, such as WC and musician's dystonia—EMG recordings revealed that antagonist muscles, which normally alternate cleanly during rapid movements, frequently co-contracted. EMG bursts were often prolonged beyond the brief durations typical of ballistic activation, and attempts at individuated finger movements were accompanied by inappropriate recruitment of adjacent muscles. At times, the opposite problem was seen: failure of an intended movement to occur at all [4,7]. These abnormalities indicated that the difficulty lay not in generating force, but in organizing and selecting the appropriate pattern of muscle activation.

WC provided a particularly revealing model that Hallett repeatedly used to probe the mechanisms of dystonia. Handwriting is a uniquely human, highly practiced skill requiring precise temporal sequencing and selective recruitment of intrinsic hand muscles. It is acquired over years of repetition until the motor program becomes automatic [11]. In WC, this well-learned action becomes distorted in a task-specific manner: patients may perform other manual tasks normally, yet develop abnormal posturing and loss of control specifically during writing [12]. The specificity of the deficit suggested interference with an established motor program rather than a nonspecific impairment of motor function. Even when overt spasms were reduced, deficits of fine motor precision persisted [4], reinforcing the idea that the disturbance affects the elaboration of motor commands upstream of muscle activation itself.

Physiological studies of inhibitory reflexes initially appeared to localize the abnormality at spinal or brainstem levels [5,13]. However, similar abnormalities could be demonstrated in clinically unaffected body regions, indicating that the disorder was not confined to the symptomatic limb. Hallett and other researchers argued that these findings were unlikely to represent primary spinal or brainstem pathology; rather, they reflected altered descending control from higher centers [7,14]. The explanatory focus therefore moved from peripheral circuitry to the higher-order processes involved in motor command generation.

If the fundamental disturbance lies in the processing of motor commands, at what point in the motor control sequence does dystonia emerge?

Movement-related cortical potentials provided temporal resolution to address this question. In WC, the negative slope component preceding voluntary finger movement was reduced despite preserved task performance and even increased EMG amplitude [15]. The abnormality was confined to the late preparatory window, approximately 300–200 ms before movement onset, implicating impaired cortical activation during motor preparation rather than during execution itself. Complementary PET studies showed reduced activation of premotor cortex during writing and altered functional correlations between premotor cortical regions and the putamen [16]. Together, these findings pointed toward dysfunction within basal ganglia–thalamo–premotor–motor circuits during the generation and refinement of motor commands.

The notion that dystonia reflects abnormal shaping of motor output before execution gained further support from studies of cortico-cortical interactions. In WC, dorsal premotor–motor inhibition was abnormally present at rest and failed to show appropriate task-related modulation [17,18]. Similarly, ventral premotor–motor interactions, normally dynamic across phases of movement preparation, were absent or altered in patients [19]. Together, these studies demonstrated that premotor influences on primary motor cortex were not simply globally reduced or increased; rather, their physiological modulation across behavioural context was abnormal. This pattern suggests that the disturbance lies less in baseline excitability than in the dynamic physiological shaping of motor cortex activity during movement preparation. At a broader network level, structural and functional imaging extended these observations. During dominant handwriting, healthy individuals recruit a segregated parieto–premotor network—including posterior parietal cortex and dorsal premotor cortex—that was absent in WC [20]. Structural alterations in premotor cortex predicted abnormal connectivity and symptom duration, suggesting that specialization of networks supporting a highly trained motor skill is disrupted.

Multimodal work further refined this model. Merchant et al. (2020) demonstrated abnormal interactions among inferior parietal lobule (dorsal and anterior subdivisions), ventral premotor cortex, and M1 [21]. In WC, the baseline influence of dorsal inferior parietal lobule onto M1 was inhibitory, whereas it was facilitatory in controls, effectively reversing the normal interaction gradient. Resting parietal–premotor connectivity was also increased. Modulating dorsal parietal output partially restored downstream premotor–motor interactions, indicating aberrant but labile network dynamics within the parietal–premotor–motor circuit. Conceptually, this was framed as disruption of a "motor vocabulary." In task-specific dystonia such as WC, the motor program remains conceptually intact, but its execution becomes unstable. The multimodal parietal integration region—positioned to shape motor output—may exert abnormal downstream influence, leading to distorted activation patterns during a learned skill. Associative plasticity studies provided convergent evidence. Using parieto–motor cortico-cortical paired associative stimulation Cho et al. (2024) showed that M1 excitability increased selectively in WC but not in cervical dystonia or healthy volunteers [22]. The exaggerated plastic response did not correlate with clinical severity, suggesting intrinsic circuit dysfunction within the parieto–motor network rather than a secondary effect of symptoms. The absence of this effect in cervical dystonia indicates that plastic mechanisms differ across focal dystonias.

Viewed together, the physiological evidence points to a common conclusion: dystonia emerges during the preparation, selection, and stabilization of motor commands within distributed sensorimotor networks. In FHD, and particularly in WC, this disturbance becomes evident when a finely tuned, extensively trained motor program is recruited, exposing instability within the circuits responsible for precision and selectivity. However, dystonia encompasses clinically diverse syndromes, ranging from task-specific focal forms such as WC to early-onset

genetic dystonia such as *TOR1A*-related dystonia. These conditions differ in age at onset, distribution, and task dependence, suggesting that they may not arise from identical mechanisms. Although the precise distinctions remain unclear, it is likely that different dystonic syndromes reflect variations in the relative contribution of the nodes within the distributed motor network—including basal ganglia, cortex, cerebellum, and sensorimotor integration processes—rather than a single uniform pathophysiological defect.

3. How does the nervous system constrain and select movement, and why does this fail in dystonia?

Motor behaviour depends not only on the generation of excitatory commands but on the structured selection of a single motor program from among competing alternatives. Voluntary movement requires the simultaneous facilitation of the intended action and suppression of adjacent or competing motor representations. As proposed in models of basal ganglia function, motor output emerges from the competition between excitatory and inhibitory pathways, with the desired movement facilitated and alternative motor programs actively suppressed [23]. In the cortex, this focusing mechanism is expressed physiologically as surround inhibition—the suppression of excitability in cortical representations adjacent to the activated muscle [24]. This mechanism, long recognized in sensory systems for sharpening discrimination, appears to serve an analogous function in the motor system: to prevent overflow and ensure selective execution of finely tuned motor commands.

The importance of this mechanism is particularly evident in individuated finger movements, where cortical representations lie in close proximity and precise control is required. Complex manual tasks—typing, musical performance, handwriting—require that excitation be precisely focused while neighbouring muscles remain suppressed. In healthy individuals, transcranial magnetic stimulation (TMS) demonstrates that during index finger flexion, motor-evoked potentials (MEPs) in a surrounding, non-synergistic muscle are transiently suppressed at movement initiation despite a concurrent rise in spinal excitability [24]. This suppression is temporally specific: it occurs during the preparatory and early phasic phases and disappears during tonic maintenance [25]. The inhibition is supraspinal, as spinal excitability (H/M ratio or F-wave measures) increases rather than decreases during this phase.

In FHD, and particularly in WC, this focusing mechanism fails. During movement initiation, MEPs in surrounding muscles are not suppressed; instead, excitability may remain unchanged or even increase [24,25]. Importantly, the excitability of the synergist muscle representation is preserved [19,25]. Thus, the problem is not insufficient activation of the intended movement, but failure to suppress competing representations.

Hallett promoted the concept of surround inhibition as a physiological explanation for the characteristic motor overflow seen in dystonia. Subsequent work sought to identify the mechanisms underlying surround inhibition. Short intracortical inhibition (SICI), a TMS protocol investigating a GABA-A–mediated inhibitory circuit within primary motor cortex, emerged as a candidate contributor. In healthy subjects, SICI is modulated across movement phases and may participate in suppressing non-involved muscles. In FHD, SICI is reduced during movement initiation in the surround muscle [25], implicating impaired intracortical GABAergic inhibition. Magnetic resonance spectroscopy demonstrated reduced GABA concentrations in contralateral sensorimotor cortex and lentiform nucleus in FHD [26], although subsequent studies were less consistent.

However, not all inhibitory circuits appear to generate surround inhibition. Long-latency afferent inhibition does not differ in a manner that explains the surround deficit [27]. Digital short afferent inhibition may even increase during movement in FHD [28], possibly reflecting altered cholinergic–GABAergic balance or compensatory mechanisms. These findings suggest that surround inhibition cannot be attributed to a single inhibitory pathway but reflects coordinated regulation across multiple inhibitory systems. The basal ganglia remain relevant to this framework. Through the relative activity of direct and indirect pathways, striatal output modulates thalamocortical drive and contributes to the selection and suppression of motor programs [29]. Although contemporary models emphasize distributed network dynamics rather than a simple opposition of two pathways, the principle that motor selection depends on balanced facilitation and suppression remains valid [30]. Dysfunction of inhibitory components within these circuits—including possible deficits of striatal GABAergic interneurons—could reduce suppression of competing motor programs and thereby impair surround inhibition.

Molecular imaging provides converging support for impaired inhibitory control. Reduced GABA-A receptor binding has been demonstrated in the left sensorimotor cortex in FHD [31], a region directly implicated in surround inhibition. The same study showed reduced binding in the cerebellar vermis and increased binding in inferior prefrontal cortex, the latter correlating negatively with disease duration and interpreted as compensatory reorganization within associative networks. These observations place inhibitory dysfunction within cortical and subcortical nodes of motor control circuits and, together with broader neurophysiological and imaging evidence implicating altered cerebellar activity and connectivity, further support the view that dystonia reflects dysfunction across distributed motor networks rather than within a single anatomical substrate [32].

Taken together, the evidence converges on a unifying principle: normal movement selection depends on transient, spatially precise suppression of competing motor representations during preparation and initiation. This suppression is supraspinal, temporally regulated, and mediated by interacting GABAergic circuits within cortex, basal ganglia, and cerebellum. In FHD, synergist excitation remains largely intact, but inhibitory focusing fails. The result is not excessive motor drive, but a breakdown of competitive suppression—allowing unintended motor programs to intrude upon the selected action. Dystonia therefore also represents a disorder of motor selection rather than of motor generation itself.

4. Why does a seemingly motor disorder implicate the sensory system?

Motor control depends on the continuous integration of sensory input with motor intention. Voluntary movement is not generated solely by descending commands; it is shaped by ongoing tactile, proprioceptive, and multimodal feedback that refines motor preparation, calibrates force and timing, and contributes to the refinement of selected motor program. The motor system operates within a closed sensorimotor loop in which afferent signals modulate cortical and subcortical excitability even before movement begins. Hallett was among the first to articulate that dystonia might reflect a disturbance of sensorimotor integration rather than a purely motor abnormality, explicitly raising the question of whether dystonia could be considered, at least in part, a sensory disorder [1].

This conceptual shift was grounded in a clinical observation: the sensory trick (or *geste antagoniste*). In dystonia, especially focal forms such as WC and cervical dystonia, a light touch to a specific body region can transiently reduce abnormal posturing [1]. In some cases, even anticipation of touch attenuates dystonia, suggesting that altered sensory input—not passive mechanical stabilization—modifies central motor processing. The sensory trick therefore implies that aberrant motor output remains dynamically modifiable by afferent input, pointing toward instability within sensorimotor circuits rather than fixed motor overactivity.

Physiological studies provided converging support for this idea. Median nerve somatosensory-evoked potentials (SEPs) in dystonia demonstrate enhanced N30 amplitudes without latency changes [33].

The N30 is thought to reflect activation of motor-related cortical regions, including supplementary motor area and premotor cortex, and its amplitude is modulated by motor state. Increased N30 amplitude therefore suggests abnormal excitatory influences within motor networks during early sensory processing. Rather than being confined to perception alone, sensory input appears to abnormally engage motor circuits at very short latency.

Behavioural investigations further revealed subtle but reproducible perceptual abnormalities. Spatial discrimination refers to the ability to distinguish two stimuli as separate in space—for example, recognizing that two points applied to the skin occupy different locations [34]. Temporal discrimination refers to the ability to perceive two sequential stimuli as occurring at distinct moments rather than merging into a single percept. In FHD, thresholds for both spatial and temporal discrimination are elevated, meaning that larger separations or longer intervals are required for accurate perception [35,36]. Importantly, these abnormalities are not restricted to the symptomatic limb but are also present in the unaffected limb and in other focal dystonia [37]. Similar findings in some unaffected relatives raise the possibility that altered sensory processing may represent an endophenotypic trait rather than a consequence of abnormal movement [38].

At the cortical level, early mapping studies reported disorganization of finger representations in primary somatosensory cortex in FHD, with enlarged and overlapping receptive fields interpreted as maladaptive plasticity induced by repetitive use [39]. Electrophysiological studies using paired-pulse SEP paradigms showed reduced suppression of specific SEP components at short intervals, correlating with elevated temporal discrimination thresholds and indicating impaired intracortical inhibitory processing in S1 [40]. Moreover, somatosensory paired associative stimulation induced exaggerated and dysregulated plastic responses in S1 of dystonia patients [41], linking abnormal inhibition to abnormal plasticity. These exaggerated responses suggest not merely increased plasticity, but impaired homeostatic regulation of synaptic modification within sensorimotor cortex [29]. In the context of repetitive and highly trained motor behaviours, reduced inhibitory control combined with abnormal plasticity may promote unstable sensorimotor encoding at the level of skill representation, even if primary somatotopic maps remain structurally intact. Together, these findings supported the influential hypothesis that task-specific dystonia reflects distorted hand somatotopy. However, this interpretation has recently been questioned, at least in musician's dystonia, where high-resolution functional imaging demonstrated intact generic finger representations within primary sensorimotor cortex [42]. These results suggest that the core disturbance may not be a gross breakdown of primary cortical maps, but rather a dysfunction of higher-order encoding or network-level integration.

In summary, in dystonia, sensory signals abnormally engage motor circuits at early latency, perceptual resolution is subtly degraded, intracortical inhibition within S1 is reduced, and plastic responses are exaggerated. Even if primary somatotopic maps remain structurally intact in dystonia, their functional regulation appears altered. In this context, sensory input may fail to provide the spatial and temporal signals necessary to stabilize motor selection. The system remains excitable but insufficiently regulated—vulnerable to interference, overflow, and intrusion of competing motor representations. Dystonia thus reflects not only disordered motor output, but instability within the sensorimotor loop that normally shapes and refines voluntary action.

5. Conclusion

What began as careful description of abnormal muscle recruitment evolved into a coherent physiological framework in which impaired inhibition, abnormal premotor shaping, dysregulated plasticity, and altered sensory modulation interact across cortical and subcortical circuits (Fig. 1). Within this network perspective, dystonia and functional dystonia—another major focus of Hallett's research—may appear clinically similar, and neither demonstrates overt structural pathology; both can be understood as disorders of information flow within distributed brain systems. A crucial difference emerges in their stability [43,44]. Once established, most adult-onset focal dystonia rarely remit. Although interventions can modulate the network—botulinum toxin by altering peripheral input, retraining strategies by modifying learned motor patterns, and deep brain stimulation by directly influencing circuit

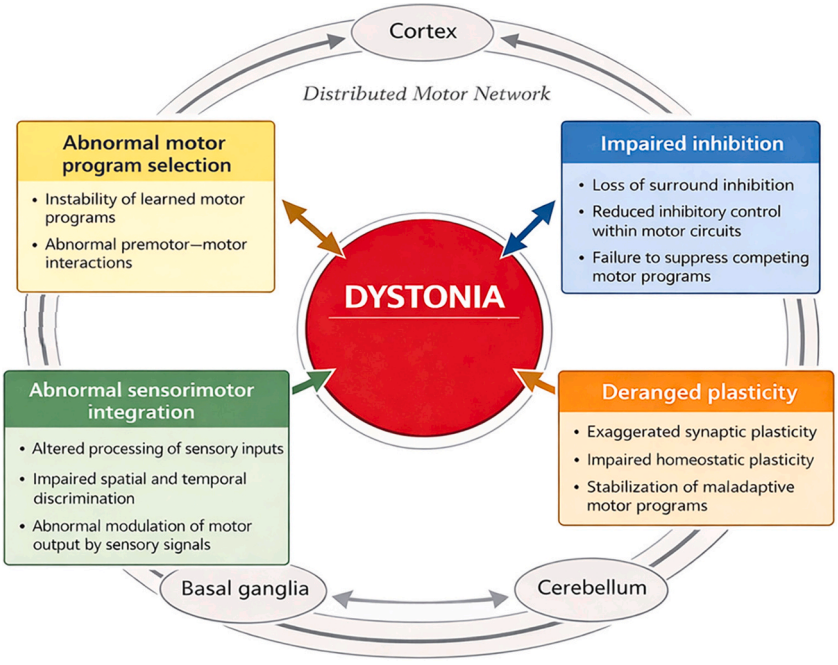

Fig. 1. Conceptual framework of physiological mechanisms contributing to dystonia. Dystonia is conceptualized as a disorder emerging from dysfunction within a distributed motor network involving sensorimotor cortex, basal ganglia, and cerebellum. Interacting abnormalities—including impaired inhibition, abnormal motor program selection, altered sensorimotor integration, and deranged plasticity— disrupt the refinement and execution of motor commands, leading to the abnormal movements and postures characteristic of dystonia.

activity—the underlying abnormal state typically reasserts itself when the intervention is withdrawn. This persistence has led to the view that many forms of dystonia reflect abnormal plasticity acquired over time and subsequently stabilized within motor networks, particularly in highly trained motor systems such as handwriting, but maintained in a manner that resists reversal. In contrast, functional dystonia may improve rapidly, suggesting that the decisive factor is not outward phenomenology but the durability of the underlying network changes [43]. The therapeutic challenge therefore extends beyond suppressing abnormal output: it is to understand how pathological motor memories are maintained at the network level, why forgetting fails, and how more durable rebalancing of sensorimotor circuits might be achieved. In this sense, Hallett's work provides more than a catalogue of physiological findings; it offers a conceptual architecture linking phenomenology to mechanism, mechanism to network reorganization, and ultimately to future directions in rational treatment.

CRediT authorship contribution statement

Anna Latorre: Writing – original draft, Conceptualization. **Lorenzo Rocchi:** Writing – original draft, Conceptualization. **Kailash Bhatia:** Writing – review & editing, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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