Review

Secondary Movement Disorders: Drug-Related, Systemic, and Neurological Etiologies Across Clinical Spectra

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Review

Secondary Movement Disorders: Drug-Related, Systemic, and Neurological Etiologies Across Clinical Spectra

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Abstract

Movement disorders frequently arise as secondary manifestations of systemic, metabolic, toxic, infectious, vascular, autoimmune, and iatrogenic conditions, yet they remain underrecognized in clinical practice. This narrative review aims to provide a comprehensive and clinically oriented overview of secondary movement disorders, emphasizing common and uncommon etiologies, underlying pathophysiological mechanisms, diagnostic challenges, and management considerations. A broad literature review was conducted using PubMed, Scopus, and Google Scholar, focusing on movement disorders associated with medications, stroke, infections, metabolic abnormalities, demyelinating and autoimmune diseases, neurodegenerative conditions, and systemic illnesses. Secondary movement disorders encompass a wide spectrum of hyperkinetic and hypokinetic phenomenologies, including tremor, dystonia, chorea, myoclonus, parkinsonism, ataxia, and mixed syndromes, often reflecting disruption of basal ganglia–thalamo–cortical and cerebellothalamic networks. Drugs—particularly antipsychotics, antiseizure medications, antidepressants, and antiemetics—represent the most frequent cause, while vascular lesions, infections, and metabolic disturbances constitute important and potentially reversible contributors. Neuroimaging and ancillary testing play a pivotal role in identifying secondary etiologies and distinguishing them from primary neurodegenerative or functional disorders. Recognition of secondary movement disorders is essential, as prompt identification and treatment of the underlying cause may lead to symptom resolution or significant improvement. This review highlights the importance of systematic evaluation, interdisciplinary collaboration, and individualized management strategies, reinforcing the concept that movement disorders often reflect multisystem disease rather than isolated motor pathology.

Keywords: secondary movement disorders; drug-induced movement disorders; parkinsonism; dystonia; tremor; chorea; myoclonus; stroke-related movement disorders; metabolic disorders; neuroimaging

1. Introduction

Movement disorders encompass a heterogeneous group of neurological conditions characterized by abnormal voluntary or involuntary movements, including hypokinetic syndromes such as parkinsonism and hyperkinetic manifestations such as tremor, dystonia, chorea, myoclonus, and tics [1]. Traditionally, movement disorders have been conceptualized as primary neurodegenerative or genetic diseases; however, it is now increasingly recognized that a substantial proportion arise secondary to medications, systemic illnesses, metabolic disturbances, vascular insults, infections, autoimmune processes, and toxic exposures [2]. These secondary movement disorders are of particular clinical importance because many are potentially reversible if the underlying cause is promptly identified and appropriately managed [3,4].

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The pathophysiological basis of movement disorders lies in dysfunction of complex motor networks involving the basal ganglia, cerebellum, thalamus, brainstem, and their cortical connections [5]. Disruption of dopaminergic, GABAergic, glutamatergic, cholinergic, and monoaminergic neurotransmission—whether through structural lesions, metabolic imbalance, immune-mediated injury, or pharmacologic interference—can result in a wide spectrum of phenomenology [6]. Importantly, similar clinical phenotypes may emerge from distinct mechanisms, while a single etiologic factor may produce diverse motor manifestations, contributing to diagnostic uncertainty and frequent misclassification as primary movement disorders [7].

Medication-induced movement disorders represent one of the most common and underrecognized categories of secondary movement disorders [8]. Antiseizure medications, antipsychotics, antidepressants, mood stabilizers, headache therapies, and other centrally acting agents may induce or exacerbate tremor, parkinsonism, dystonia, dyskinesia, myoclonus, and ataxia [9]. These adverse effects may occur acutely or insidiously, may be dose-dependent or idiosyncratic, and often resemble idiopathic neurodegenerative syndromes [10]. Failure to recognize drug-related etiologies may result in unnecessary diagnostic testing, inappropriate escalation of therapy, and avoidable morbidity [11].

Beyond pharmacologic causes, systemic neurological and medical conditions are increasingly implicated in the development of movement disorders. Cerebrovascular disease, particularly lesions involving the basal ganglia, thalamus, cerebellum, or brainstem, can lead to acute or delayed-onset movement disorders, including hemichorea, dystonia, tremor, and vascular parkinsonism [12]. Infections—through direct neurotropism, immune-mediated mechanisms, or post-infectious inflammation—remain important and often treatable causes worldwide [13]. Autoimmune and demyelinating diseases, metabolic abnormalities, hepatic and renal dysfunction, and endocrine disorders further expand the differential diagnosis, emphasizing the need for a broad, systematic approach [14].

Neuroimaging has become indispensable in the evaluation of suspected secondary movement disorders, aiding in lesion localization, etiologic clarification, and differentiation from primary neurodegenerative processes [15]. Advances in structural, functional, and molecular imaging have reinforced the concept of movement disorders as network diseases rather than isolated basal ganglia dysfunction [16]. Similarly, growing interest in biomarkers and pathogenetic pathways—including neuroinflammation, mitochondrial dysfunction, proteostasis failure, and α-synuclein aggregation—has begun to bridge the gap between secondary and primary movement disorder mechanisms [17].

The aim of this review is to provide a comprehensive, clinically oriented overview of secondary movement disorders across a wide range of etiologies, with particular emphasis on drug-induced syndromes, systemic diseases, and structural brain disorders. By integrating clinical phenomenology, pathophysiology, neuroimaging, and management principles, this article seeks to improve recognition of secondary causes, facilitate timely diagnosis, and promote targeted, potentially reversible interventions in patients presenting with movement disorders.

2. Methodology

This work was conducted as a comprehensive narrative review of the literature addressing secondary movement disorders arising from medications, systemic diseases, metabolic abnormalities, vascular disorders, infections, autoimmune conditions, and structural brain lesions. A structured search of the PubMed/MEDLINE, Scopus, and Web of Science databases was performed, including articles published up to December 2025. Search terms combined keywords and Medical Subject Headings related to movement disorders and secondary etiologies, including “movement disorders,” “parkinsonism,” “tremor,” “dystonia,” “chorea,” “myoclonus,” “drug-induced movement disorders,” “secondary parkinsonism,” “stroke,” “infection,” “autoimmune encephalitis,” “metabolic disease,” and “neuroimaging.” Peer-reviewed original studies, systematic and narrative reviews, observational studies, clinical trials, case series, and selected case reports were included when they provided clinically or mechanistically relevant information. Articles were screened by title

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and abstract, followed by full-text review for relevance, with additional studies identified through reference list screening. Data extraction focused on clinical phenomenology, underlying etiologies, proposed pathophysiological mechanisms, neuroimaging findings, management strategies, and clinical outcomes. Given the heterogeneity of study designs and etiologic factors, findings were synthesized qualitatively and organized thematically by causative category to highlight shared mechanisms, diagnostic challenges, and therapeutic implications across secondary movement disorders.

3. Antiseizure Medications and Movement Disorders

Antiseizure medications (ASMs) are widely used in the management of epilepsy, neuropathic pain, and psychiatric disorders [18], yet several agents have been implicated in the development or exacerbation of movement disorders [19]. These adverse effects reflect the broad influence of ASMs on GABAergic, glutamatergic, and ion channel–mediated neurotransmission within basal ganglia–thalamo–cortical circuits [20]. Moreover, patients with drug-resistant epilepsy are more likely to develop abnormal movements [21], and somatosensory symptoms can lead to further challenging in diagnosis [22,23]. Also, some epileptic encephalopathies can have abnormal involuntary movements like Rasmussen encephalitis [24]. Another confounder can be reflexive epilepsy syndromes [25] and even functional seizures [26].

Valproate is the most consistently associated ASM, linked to reversible parkinsonism, postural tremor, and, less commonly, chorea and dystonia [27]. Valproate-induced parkinsonism typically occurs in older adults and may unmask subclinical nigrostriatal dysfunction, with symptoms improving after drug discontinuation [28,29]. Hyperammonemia and mitochondrial toxicity have also been proposed as contributory mechanisms [30,31].

Phenytoin has been associated with cerebellar ataxia [32], tremor [33], choreoathetosis [34], and orofacial dyskinesias [35], particularly in the setting of chronic exposure [36] or supratherapeutic levels [37]. Structural cerebellar changes may persist despite withdrawal [38], highlighting the potential for long-term neurotoxicity [39].

Pregabalin and gabapentin [40], $\alpha 2\delta$ subunit calcium channel ligands [41], are widely used for neuropathic pain [42], epilepsy, and anxiety disorders and are generally considered safe from a movement disorder standpoint [43,44]. However, both agents have been reported to induce or exacerbate abnormal movements, most commonly postural tremor and myoclonus, particularly at higher doses or in patients with renal impairment [45]. Less frequently, cases of ataxia [46], parkinsonism [47], and dyskinesia have been described, likely reflecting dose-dependent modulation of excitatory neurotransmission within cerebellothalamic and basal ganglia circuits [48]. Symptoms are typically reversible with dose reduction or drug discontinuation [49], underscoring the importance of medication review in patients presenting with new or worsening movement disorders.

Newer ASMs are not exempt from motor adverse effects. Levetiracetam has been reported to induce tremor, tics, and rare cases of dyskinesia, likely reflecting altered synaptic vesicle protein 2A–mediated neurotransmission [50]. Topiramate may cause tremor, myoclonus, or, infrequently, dystonia, possibly related to enhanced GABAergic tone and inhibition of glutamatergic pathways [51]. Lamotrigine has been infrequently associated with tremor, myoclonus, and, rarely, exacerbation of parkinsonism [52,53]. Belly dancer dyskinesia is a rare hyperkinetic movement disorder characterized by involuntary [54], rhythmic contractions of the abdominal wall muscles [55]. Baclofen, a GABA receptor agonist [56], is commonly used to treat spasticity and may improve dystonia or painful muscle spasms, but in toxic levels can cause catatonia [57].

Cenobamate, a newer antiseizure medication approved for focal-onset seizures, exerts its effects through dual modulation of voltage-gated sodium channels and positive allosteric modulation of GABA receptors [58]. While clinical trials have primarily reported central nervous system adverse effects such as somnolence, dizziness, and ataxia, postmarketing experience suggests that movement disorders are uncommon but may include tremor and gait instability, particularly during rapid titration or in combination with other sodium channel–blocking agents [59]. Given its potent

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pharmacodynamic profile and potential for drug–drug interactions, careful dose escalation and monitoring are warranted, especially in patients with preexisting movement disorders or cerebellar dysfunction [60].

In contrast, some ASMs may exert therapeutic effects on movement disorders. Clonazepam is commonly used in myoclonus and tics, while carbamazepine may benefit certain paroxysmal dyskinesias [61]. Recognition of ASM-induced movement disorders is critical, as symptoms may mimic primary neurodegenerative or functional syndromes [62]. Also, adverse effect to ASMs is the most common cause of withdrawal of ASMs [63].

4. Depression and Movement Disorders

Depression is highly prevalent across movement disorders and represents both a non-motor manifestation of neurodegenerative disease and a contributor to disability and reduced quality of life [64]. In Parkinson's disease (PD), depression affects up to 40–50% of patients and may precede motor symptom onset, supporting its role as a prodromal feature [65,66]. Similar associations are observed in Huntington's disease, dystonia, and atypical parkinsonian syndromes, where mood disturbances often correlate poorly with motor severity, suggesting partially independent neurobiological mechanisms [67].

The pathophysiology linking depression and movement disorders involves shared dysfunction within frontostriatal, limbic, and monoaminergic networks [68]. Degeneration or dysregulation of dopaminergic, serotonergic, and noradrenergic pathways contributes to both impaired motor control and altered emotional processing [69]. Neuroimaging studies further demonstrate abnormalities in the prefrontal cortex, amygdala, and basal ganglia, reinforcing the concept of movement disorders as network diseases extending beyond motor circuits [70].

Pharmacologic treatment introduces additional complexity. Dopaminergic therapies, including levodopa and dopamine agonists, may improve depressive symptoms through enhancement of mesolimbic dopamine signaling, though fluctuations can contribute to mood instability, apathy, or impulse control disorders [71]. Conversely, antidepressant medications may influence motor function [72]. Selective serotonin reuptake inhibitors (SSRIs) are generally well tolerated but have been associated with tremor, akathisia, or, rarely, parkinsonism. Tricyclic antidepressants and serotonin–norepinephrine reuptake inhibitors may worsen orthostatic hypotension [73] or cognitive impairment [74], particularly in PD [75]. Mirtazapine has been infrequently associated with extrapyramidal symptoms, including tremor, akathisia, dystonia [76], and rare cases of parkinsonism [77], likely reflecting complex serotonergic–noradrenergic modulation of dopaminergic pathways [78]. Buspirone is generally considered motor-neutral but has been infrequently associated with tremor [79] and dystonia [80], rarely, akathisia [81]. Bupropion, a norepinephrine–dopamine reuptake inhibitor used to treat depression [82], has been associated with tremor and, less commonly, myoclonus [83], tics, dystonia, or exacerbation of underlying movement disorders [84].

Trazodone acts as a serotonin 5-HT_2A antagonist and weak serotonin reuptake inhibitor, with secondary α-adrenergic antagonism [85]; altered serotonergic–dopaminergic balance may rarely provoke or worsen extrapyramidal symptoms, including tremor, dystonia, parkinsonism [86], or myoclonus [87].

In hyperkinetic disorders, such as dystonia or tardive syndromes, antidepressants may unmask or exacerbate abnormal movements through serotonergic–dopaminergic interactions [88]. Careful medication selection, individualized risk–benefit assessment, and collaboration between neurology and psychiatry are therefore essential.

5. Headache and Movement Disorders

Headache disorders and movement disorders frequently coexist, suggesting shared neurobiological mechanisms rather than a purely coincidental relationship [89]. Migraine is the most commonly reported headache phenotype among patients with movement disorders, particularly

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Parkinson's disease (PD), dystonia, and essential tremor [90]. Epidemiologic studies indicate that migraine prevalence may be higher in PD patients prior to motor symptom onset, raising the possibility that headache represents an early, non-motor manifestation of neurodegenerative disease [91]. Noteworthy, headache can also be a post-ictal phenomena [92].

The pathophysiological overlap between headache and movement disorders centers on dysfunction within dopaminergic, serotonergic, and trigeminovascular pathways [93]. Dopamine plays a critical modulatory role in migraine biology [94], as evidenced by dopaminergic hypersensitivity during migraine attacks and the frequent occurrence of migraine-associated symptoms such as nausea, yawning [95], and hypotension [96]. This dopaminergic involvement may also explain the association between headache and hyperkinetic disorders, including dystonia and tic disorders, where altered basal ganglia–thalamo–cortical circuits are central [97].

Dystonia-related headache, particularly in cervical dystonia, illustrates a direct structural and functional link between abnormal motor activity and head pain [98]. Sustained muscle contractions, impaired sensorimotor integration, and altered proprioceptive feedback are thought to contribute to secondary headache syndromes in this population [99]. Importantly, treatment of the underlying movement disorder—such as botulinum toxin injections—often leads to significant improvement in headache burden, supporting a causal relationship.

In PD, headache phenotype may evolve over the disease course and be influenced by dopaminergic therapy [100]. Fluctuation-related headache and early-morning headache have been described, reflecting complex interactions between central pain processing and dopamine depletion.

Several medications commonly used in the acute and preventive treatment of headache disorders have been implicated in the development or exacerbation of movement disorders [101]. These adverse effects are typically mediated through dopaminergic antagonism, serotonergic modulation, or maladaptive neuroplastic changes within basal ganglia–thalamo–cortical circuits.

Dopamine receptor antagonists, frequently prescribed for acute migraine-associated nausea, are the most well-recognized offenders. Agents such as metoclopramide and prochlorperazine can induce acute dystonia, akathisia, parkinsonism, and, with repeated exposure, tardive dyskinesia [102]. These risks are heightened in younger patients and with parenteral administration. Although often reversible, tardive syndromes may persist despite drug discontinuation, underscoring the importance of judicious use.

Triptans, while generally considered safe from a motor standpoint, have been rarely associated with tremor, myoclonus, and serotonin toxicity—particularly when combined with selective serotonin reuptake inhibitors or monoamine oxidase inhibitors [103]. In such cases, neuromuscular hyperactivity rather than basal ganglia dysfunction is the predominant mechanism.

Valproate, widely used for migraine prophylaxis [104], has been linked to reversible parkinsonism, particularly in older adults or those with underlying nigrostriatal vulnerability [105]; also, dose-dependent pancytopenia was already observed [106]. Similarly, topiramate may precipitate tremor and, less commonly, myoclonus or dystonia, likely related to altered GABAergic and glutamatergic neurotransmission [107,108].

Emerging migraine therapies are not exempt from neurologic adverse effects. Rare cases of tremor and dyskinesia have been reported with calcitonin gene-related peptide (CGRP) receptor antagonists [109,110], although a direct causal relationship remains uncertain [111]. Flunarizine, used in migraine prophylaxis [112], has been associated with drug-induced parkinsonism and other extrapyramidal symptoms due to dopamine receptor blockade [113].

Opioids can be associated with both headache disorders and movement abnormalities through their effects on central pain and motor pathways [114]. Chronic opioid use is a well-recognized cause of medication-overuse headache, driven by altered nociceptive processing and opioid-induced hyperalgesia [115]. In parallel, opioids may precipitate movement disorders such as myoclonus, tremor, dystonia, or, rarely, parkinsonism, particularly at high doses or in the setting of renal dysfunction and metabolite accumulation [116]. These complications are thought to arise from opioid effects on μ-receptor–mediated neurotransmission, including dysregulation of dopaminergic and

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inhibitory pathways [117]. Recognition is important, as dose reduction or opioid discontinuation often leads to improvement in both headache and movement symptoms.

6. Antipsychotics, Mood Stabilizers, and Movement Disorders

Antipsychotics and mood stabilizers are widely prescribed for psychotic, bipolar, and affective disorders, yet their use is closely linked to the development or exacerbation of movement disorders [118]. These adverse effects arise primarily from disruption of dopaminergic signaling within basal ganglia–thalamo–cortical circuits, as well as broader modulation of serotonergic, GABAergic, and glutamatergic pathways [119].

Antipsychotics, particularly dopamine D_2 receptor antagonists, are the most common cause of drug-induced movement disorders. First-generation antipsychotics are strongly associated with acute dystonia, akathisia, drug-induced parkinsonism, and tardive syndromes [120]. Second-generation antipsychotics demonstrate a lower but variable risk, depending on dopaminergic affinity and serotonergic modulation [121]. Agents such as risperidone and paliperidone retain relatively high extrapyramidal risk, whereas quetiapine and clozapine are considered motor-sparing and are preferred in patients with Parkinson's disease psychosis [122]. Tardive dyskinesia, characterized by involuntary choreiform or dystonic movements, remains a major long-term complication and may persist despite antipsychotic discontinuation [123].

Mood stabilizers also exert clinically relevant motor effects. Valproate is associated with postural tremor and reversible parkinsonism, particularly in older adults or with prolonged exposure [124]. Lithium commonly causes fine tremor and [125], at toxic levels, may provoke ataxia, myoclonus [126], or choreiform movements; chronic use has also been linked to irreversible cerebellar dysfunction in rare cases [127]. Carbamazepine and oxcarbazepine may induce ataxia, tremor, or nystagmus, reflecting sodium channel blockade and cerebellar involvement [128]. Lamotrigine is generally well tolerated but has been rarely associated with tremor or myoclonus [52].

Methylphenidate may induce or exacerbate tics, chorea, dystonia, or stereotypies through dopaminergic overstimulation, particularly in susceptible individuals [129].

7. Stroke and Movement Disorders

Stroke is an important but underrecognized cause of secondary movement disorders [130], resulting from disruption of motor control networks involving the basal ganglia, thalamus [131], cerebellum [132], and their cortical connections [133]. Cerebrovascular disorders are among the most common causes of neurological disorders [134]. Although relatively uncommon, post-stroke movement disorders provide key insights into human motor circuit function and often pose diagnostic and therapeutic challenges [135]. Atrial fibrillation increases the risk of cardioembolic stroke involving basal ganglia and thalamic circuits [136–138], thereby serving as an important vascular contributor to secondary movement disorders [139]. A study from Brazil revealed significant increase of cardiovascular risk factors related to the development of stroke and movement disorders [140]. But, not only ischemic also hemorrhagic strokes were already described with movement disorders [141,142].

The most frequently observed post-stroke movement disorders are hemichorea and hemiballismus [143], typically arising from lesions affecting the subthalamic nucleus, caudate nucleus, putamen, or thalamus [144]. These hyperkinetic syndromes usually present acutely or subacutely following ischemic stroke and are contralateral to the lesion [145]. Pathophysiologically, loss of inhibitory basal ganglia output leads to excessive thalamocortical excitation and involuntary movements [146]. While often self-limited, symptoms may persist and require pharmacologic intervention.

Sleep disorders, movement disorders, and stroke are tightly interconnected through shared neuroanatomical and vascular mechanisms [147]. Conditions such as REM sleep behavior disorder and restless legs syndrome frequently coexist with Parkinsonian syndromes and may precede clinical

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onset [148]. Disordered sleep also increases vascular risk via hypertension and inflammation [149], while stroke can disrupt motor and sleep-regulatory circuits, leading to secondary parkinsonism, insomnia, hypersomnia, and worsened functional recovery, with important implications for prognosis, prevention, and multidisciplinary neurological care strategies [150].

Other movement disorders associated with stroke include dystonia, tremor, myoclonus, and parkinsonism, which may present weeks to months after the vascular insult [151]. Delayed onset is thought to reflect maladaptive plasticity and reorganization within motor circuits rather than direct tissue injury alone [152]. Vascular parkinsonism, classically associated with bilateral subcortical ischemic changes [153], manifests with predominant lower-body bradykinesia, gait disturbance [154], and postural instability, often with poor levodopa responsiveness [155]. Other possible clinical manifestations involve the development of abnormal eye movements [156]. Limb-shaking TIA is a rare manifestation of cerebral hypoperfusion [157], characterized by transient, involuntary limb movements that can mimic focal seizures and abnormal movements [158]. Seizures may occur in IPAH secondary to cerebral hypoperfusion [159], hypoxemia, syncope-related anoxic injury, or complications of severe right-sided heart failure and elevated pulmonary pressures [160]. Basal ganglia involvement in artery of Heubner stroke leading to movement disorder [161].

Lesion location plays a major role in phenotypic expression [162]. Thalamic and brainstem strokes are commonly linked to tremor and Holmes tremor [163], whereas cerebellar infarcts may result in ataxia and action tremor [164]. Noteworthy, significant dysphagia can be observed [165]. Corpus callosum infarcts can lead to astasia [166]. Neuroimaging is essential for diagnosis, aiding lesion localization and exclusion of neurodegenerative or functional etiologies [167]. Stroke involving the precentral 'hand knob' region can result in focal hand weakness and secondary movement disorders due to disruption of primary motor cortex output [168]. On the other side, there is also the cortical (spastic) foot drop [169,170]. A rare clinical presentation in which venous outflow obstruction leads to elevated intracranial pressure or focal brainstem/temporal involvement, resulting in an ipsilateral peripheral facial nerve palsy [171]. More common clinical manifestations include seizures, occurring in more than half of the individuals affected by cerebral venous sinus thrombosis [172].

8. Infections and Movement Disorders

Infections represent an important and potentially reversible cause of secondary movement disorders, arising through direct neurotropism, immune-mediated mechanisms, vascular injury, or post-infectious neuroinflammation [173]. Both acute and delayed movement abnormalities have been described across bacterial, viral, and parasitic infections, often reflecting involvement of the basal ganglia, thalamus, cerebellum, or brainstem [174]. Noteworthy, aspiration pneumonia is a leading cause of morbidity and mortality in Parkinson's disease [175].

Viral infections are among the most commonly implicated. Human immunodeficiency virus (HIV) can produce a wide spectrum of movement disorders [176], including chorea, dystonia, tremor, and parkinsonism, through direct central nervous system involvement or opportunistic infections [177]. Post-encephalitic parkinsonism, historically associated with encephalitis lethargica [178], has also been reported following infections with influenza [179–181], Japanese encephalitis virus [182], measles [183,184], dengue virus [185–187], West Nile virus [188], and more recently SARS-CoV-2 [189,190]. These syndromes may emerge weeks to months after infection, suggesting immune-mediated or neurodegenerative mechanisms [191].

Herpesvirus infections, particularly HSV and VZV, can involve basal ganglia and thalamic structures [192], resulting in acute or post-infectious movement disorders such as chorea, tremor, parkinsonism, and others abnormal eye movements [193].

Bacterial infections can similarly affect motor circuits [194]. Sydenham chorea, a post-streptococcal autoimmune disorder, is characterized by involuntary choreiform movements and remains a prototypical example of infection-induced movement disorder [195]. Tuberculosis and neurosyphilis may present with tremor, dystonia [196], or parkinsonism [197] due to meningoencephalitis [198], vasculitis [199], or intracranial granulomas involving deep gray matter

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structures [200,201]. In neurosyphilis, the Jarisch–Herxheimer reaction may transiently worsen or unmask movement disorders [202] due to acute inflammatory responses following antibiotic initiation [203]. Bartonella henselae infection can cause central nervous system involvement with rare secondary movement disorders, including chorea, tremor, GBS [204], or parkinsonism [205].

A chronic infection caused by Mycobacterium leprae that can produce peripheral neuropathy and immune-mediated complications [206], occasionally manifesting with tremor, neuropathic pain, or secondary movement abnormalities due to nerve damage [207]. An aggressive angioinvasive fungal infection that may cause cranial neuropathies [208], cavernous sinus thrombosis, cerebral infarction or hemorrhage, seizures [209], altered mental status, and focal neurological deficits due to vascular invasion and rapid tissue necrosis is zygomycosis [210].

Parasitic infections, particularly toxoplasmosis [211] in immunocompromised individuals, frequently involve the basal ganglia and are associated with hemichorea [212], dystonia, and tremor [213], especially in cases of rhombencephalitis [214]. Neurocysticercosis may also produce a range of hyperkinetic [215] and hypokinetic movement disorders depending on lesion location and inflammatory burden [216,217]. African trypanosomiasis may cause extrapyramidal movement disorders due to basal ganglia involvement [218]. Neuroschistosomiasis can produce movement disorders through inflammatory involvement of the brain and spinal cord [219].

Antibiotics have been implicated in a range of secondary movement disorders through mechanisms that include neurotoxicity, metabolic disturbance, and immune-mediated effects [220]. β-lactams (particularly high-dose penicillins and cephalosporins) and fluoroquinolones can precipitate tremor [221], myoclonus [222,223], chorea [224], idiopathic intracranial hypertension [225], and seizures, especially in older adults or patients with renal impairment, likely due to GABAergic inhibition and excitatory neurotransmission imbalance [226,227]. Metronidazole is classically associated with reversible cerebellar ataxia, dysarthria, and tremor, often accompanied by characteristic dentate nucleus hyperintensities on MRI [228]. Macrolides and sulfonamides have rarely been linked to extrapyramidal symptoms, including dystonia and parkinsonism, potentially via drug–drug interactions or metabolic toxicity [229]. Additionally, antibiotic exposure may unmask underlying movement disorders or contribute indirectly through antibiotic-associated encephalopathy leading to asterixis [230].

9. Demyelinating Disease and Movement Disorders

Demyelinating diseases, particularly multiple sclerosis (MS), are increasingly recognized as causes of secondary movement disorders [231]. Tremor is the most common manifestation in MS [232], frequently presenting as postural or intention tremor due to cerebellar and cerebellothalamic pathway involvement. Other reported movement disorders include dystonia, chorea, myoclonus, parkinsonism, and ataxia, reflecting demyelinating lesions affecting the basal ganglia, thalamus, brainstem, or spinal cord [233]; which can especially be seen in recurrent cases [234]. Movement symptoms may fluctuate with disease activity and often correlate with lesion location on neuroimaging. Recognition of demyelination-related movement disorders is important, as targeted immunomodulatory therapy and symptomatic treatment may lead to clinical improvement [235].

Autoimmune diseases are an increasingly recognized and potentially treatable cause of movement disorders [236]. Immune-mediated inflammation or antibody-driven disruption of basal ganglia, cerebellar, or cortical networks can produce a wide spectrum of phenotypes, including chorea, dystonia, tremor, myoclonus, ataxia, and parkinsonism [237,238]. Classic examples include Sydenham chorea [239], anti–NMDA receptor encephalitis, GAD-65 [240], MOG [241], anti–LGI1–associated faciobrachial dystonic seizures [242], and movement disorders linked to lupus [243], antiphospholipid syndrome, Sjögren syndrome [244], and paraneoplastic syndromes [245]. Symptoms may be acute or subacute, fluctuate over time, and coexist with cognitive, psychiatric, or systemic features [246]. Prompt recognition is critical, as immunotherapy—including corticosteroids, intravenous immunoglobulin, plasma exchange, or targeted biologics—can lead to significant neurological improvement and prevention of long-term disability.

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10. Dementia and Movement Disorders

Dementia syndromes frequently coexist with movement disorders, reflecting overlapping neurodegenerative processes involving cortical, subcortical, and brainstem networks [247]. Parkinsonian features are common in dementia with Lewy bodies (DLB) and Parkinson's disease dementia (PDD), while extrapyramidal signs, gait disturbance, and myoclonus may also occur in Alzheimer's disease (AD), particularly in advanced stages [248]. In contrast, frontotemporal dementia may present with motor neuron disease or atypical parkinsonism, including corticobasal syndrome [249].

Pharmacologic treatment of dementia can significantly influence motor function [250]. Cholinesterase inhibitors (donepezil, rivastigmine, galantamine [251]) are a mainstay of therapy for AD, DLB, and PDD and may modestly improve cognitive and behavioral symptoms [252]. In DLB and PDD, cholinesterase inhibitors often improve neuropsychiatric symptoms without worsening parkinsonism, though tremor and syncope have been reported [253]. Memantine, an NMDA receptor antagonist, is generally motor-neutral but may rarely exacerbate confusion or gait instability [254]. Noteworthy, cholinesterase inhibitors have already been associated with different movement disorders such as dystonia, myoclonus [255], and parkinsonism .

Management of psychosis and agitation introduces further complexity. Dopamine-blocking antipsychotics can precipitate severe parkinsonism, rigidity, and neuroleptic sensitivity reactions in DLB [256]. Motor-sparing agents such as quetiapine, clozapine, and pimavanserin are therefore preferred when antipsychotic therapy is required [257]. Recognition of medication-related motor effects is essential, as inappropriate pharmacologic choices may significantly worsen functional outcomes in patients with dementia [258].

11. Metabolic Abnormalities and Movement Disorders

Metabolic abnormalities represent an important and often reversible cause of movement disorders [259]. Disruptions in glucose regulation [260], electrolytes (copper [261], calcium [262,263], sodium [264]), hepatic [265] or renal function [266], thyroid [267], and mitochondrial metabolism can interfere with basal ganglia and cerebellar networks [268], producing chorea, dystonia, myoclonus, ataxia, or parkinsonism [269]. Classic examples include hypoglycemia-induced chorea, nonketotic hyperglycemia–related hemiballismus [270], Wilson disease [271], hepatic encephalopathy [272,273], and uremic movement disorders [259]. Early recognition of these metabolic etiologies is essential, as timely correction frequently leads to substantial clinical improvement and may prevent irreversible neurological damage [274].

Glutaric aciduria type I (GA-I) is a rare autosomal recessive metabolic disorder caused by deficiency of glutaryl-CoA dehydrogenase [275], leading to accumulation of glutaric and 3-hydroxyglutaric acids that are toxic to the basal ganglia. Movement disorders are a hallmark of GA-I, most commonly presenting in infancy or early childhood with acute or subacute dystonia following intercurrent illness or metabolic stress [276]. Chronic sequelae include severe generalized dystonia, choreoathetosis, and axial hypotonia, often associated with striatal injury on neuroimaging [277].

Adrenoleukodystrophy (ALD) is a rare X-linked peroxisomal disorder caused by pathogenic variants in the ABCD1 gene, leading to impaired transport and accumulation of very-long-chain fatty acids (VLCFAs) in the nervous system and adrenal cortex [278]. Neurological manifestations vary by phenotype and age of onset and may include progressive spastic paraparesis, dystonia [279], ataxia, tremor, and parkinsonian features, reflecting involvement of corticospinal tracts and deep gray matter. Forms include childhood cerebral ALD, adult cerebral ALD, and adrenomyeloneuropathy (AMN), the latter often presenting with slowly progressive spastic gait and sensory disturbances.

12. Challenging Cases in Movement Disorders

Challenging cases in movement disorders often arise from phenotypic overlap, fluctuating symptoms, and rare etiologies that mimic more common conditions. Patients may present with mixed

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hyperkinetic and hypokinetic features, episodic symptoms, or atypical age of onset, complicating diagnosis and delaying appropriate treatment [280]. Structural, metabolic, genetic, autoimmune, and neurodegenerative causes frequently intersect, requiring a systematic and multidisciplinary diagnostic approach. A rare presentation in which elongation or calcification of the styloid process compresses the cervical sympathetic chain or carotid artery, resulting in ipsilateral ptosis, miosis, and anhidrosis without other focal neurological deficits [281].

Alternating hemiplegia of childhood (AHC) exemplifies the complexity of such cases. AHC is a rare neurodevelopmental disorder, typically caused by pathogenic variants in ATP1A3, characterized by recurrent episodes of hemiplegia that alternate sides and may be accompanied by abnormal eye movements, dystonia, choreoathetosis, and autonomic features [282]. Symptoms usually begin before 18 months of age and can transiently resolve with sleep, often leading to misdiagnosis as epilepsy, migraine, or functional neurological disorder [283]. Over time, many patients develop persistent movement abnormalities, cognitive impairment, and gait disturbance, further blurring diagnostic boundaries [284]. Dislocation or subluxation of the crystalline lens due to zonular weakness, commonly associated with systemic disorders such as Marfan syndrome [285], homocystinuria, Weill-Marchesani syndrome, or ocular trauma [286].

Rabbit syndrome is a rare, drug-induced movement disorder characterized by fine, rapid, rhythmic vertical movements of the lips and perioral muscles, resembling a rabbit's chewing motion, while sparing the tongue [287]. It is most commonly associated with chronic exposure to antipsychotic medications, particularly typical neuroleptics, and is considered a form of extrapyramidal side effect distinct from tardive dyskinesia [288]. Symptoms often worsen with stress and improve with anticholinergic treatment, highlighting its underlying dopaminergic–cholinergic imbalance [289].

Pisa syndrome is a rare postural movement disorder characterized by a persistent, involuntary lateral flexion of the trunk, often with axial rotation, giving the appearance of leaning to one side [290]. It is most commonly associated with exposure to dopaminergic-blocking agents such as antipsychotics, but may also occur in patients with Parkinson's disease, dementia with Lewy bodies, or following the use of dopaminergic medications, cholinesterase inhibitors, or antiemetics [291]. The pathophysiology is thought to involve an imbalance between dopaminergic and cholinergic systems affecting axial muscle control. Pisa syndrome can fluctuate with posture and walking and is often partially reversible with medication adjustment, particularly withdrawal of the offending drug, optimization of dopaminergic therapy, or cautious use of anticholinergic agents [292].

Movement disorders during pregnancy present unique diagnostic and therapeutic challenges due to physiological changes, fetal safety considerations, and evolving symptom profiles [293]. Pregnancy can exacerbate preexisting movement disorders such as Parkinson's disease, dystonia, or tics [294], while also giving rise to pregnancy-specific conditions like chorea gravidarum, historically associated with rheumatic fever and now more commonly linked to autoimmune disease or antiphospholipid syndrome [295]. Eclampsia can be associated with posterior reversible encephalopathy syndrome [296]. Hormonal fluctuations, altered dopamine sensitivity, and changes in drug pharmacokinetics may influence symptom severity [297]. Management requires careful balancing of maternal functional needs with fetal risk, often favoring nonpharmacologic strategies or medications with established safety profiles, alongside close multidisciplinary collaboration between neurology and obstetrics.

Substances of abuse are an important and often underrecognized cause of movement disorders, acting through direct neurotoxicity, neurotransmitter imbalance, or withdrawal effects [298]. Cocaine and amphetamines can precipitate chorea, dystonia, tics, or stereotypies through excess dopaminergic stimulation, while chronic use may lead to persistent dyskinesias [299]. Alcohol is associated with tremor, ataxia, myoclonus, and, in withdrawal states, hyperkinetic movements [300]. Opioids may induce myoclonus and rigidity, particularly with high doses or metabolite accumulation [301]. Cannabis has been linked to tremor and, rarely, acute dystonia, whereas inhalants and synthetic drugs can cause irreversible basal ganglia injury with secondary

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parkinsonism or dystonia [302]. Recognition of substance-related movement disorders is essential, as symptoms may improve with cessation, detoxification, and supportive neurological management.

Bardet–Biedl syndrome is a rare genetically heterogeneous ciliopathy characterized by retinal dystrophy [303], obesity, polydactyly, hypogonadism, renal abnormalities, and cognitive impairment, with occasional but clinically significant movement disorder manifestations [304]. Although movement disorders are not core diagnostic features, affected individuals may develop tremor, ataxia, parkinsonism, dystonia, or impaired motor coordination, likely reflecting cerebellar involvement, basal ganglia dysfunction, or neurodevelopmental abnormalities related to defective ciliary signaling [305]. Marden–Walker syndrome is a rare congenital disorder characterized by distinctive facial dysmorphism, congenital joint contractures (arthrogryposis), and severe neurodevelopmental impairment [306]. Affected individuals typically have mask-like facies, blepharophimosis, micrognathia, hypotonia, and profound developmental delay, often with associated cerebellar or other central nervous system abnormalities. Management is supportive and requires multidisciplinary care [307]. Cervical spondylotic myelopathy and radiculopathy [308] is an important mimic of amyotrophic lateral sclerosis [309].

13. Neuroimaging in Movement Disorders

Neuroimaging has become a cornerstone in the evaluation and understanding of movement disorders, providing critical insights into disease mechanisms, diagnosis, and progression [310]. Structural, functional, and molecular imaging techniques allow in vivo assessment of basal ganglia–thalamo–cortical networks that underlie both hypokinetic and hyperkinetic syndromes. Also, neuroimaging can be useful to differentiate PD from drug-induced parkinsonism [311].

Structural MRI plays a foundational role in excluding secondary causes of movement disorders and identifying disease-specific patterns [312]. In atypical parkinsonian syndromes, characteristic findings such as putaminal atrophy and the “hot cross bun” sign in multiple system atrophy, midbrain atrophy in progressive supranuclear palsy, and asymmetric cortical atrophy in corticobasal degeneration aid diagnostic differentiation [313]. Advanced MRI techniques, including susceptibility-weighted imaging and neuromelanin-sensitive sequences [314], further enhance visualization of nigrostriatal degeneration and iron deposition.

Functional neuroimaging, particularly dopamine transporter (DaT) single-photon emission computed tomography (SPECT) [315], is widely used to distinguish neurodegenerative parkinsonism from non-dopaminergic movement disorders such as essential tremor or drug-induced parkinsonism [316]. Positron emission tomography (PET) enables more detailed interrogation of dopaminergic, serotonergic, and cholinergic pathways, contributing to phenotypic stratification and therapeutic decision-making [317].

In hyperkinetic disorders, including dystonia and chorea, functional MRI and PET studies demonstrate aberrant sensorimotor integration, network-level dysregulation, and abnormal plasticity rather than focal structural lesions [318]. These findings support the conceptualization of movement disorders as network diseases rather than isolated basal ganglia dysfunction [318,319]. The emerging concept of “network kernels” has provided new perspectives on brain connectivity [320].

Cardiac 123I-metaiodobenzylguanidine (MIBG) scintigraphy is a valuable functional imaging modality for assessing postganglionic sympathetic cardiac innervation and has emerged as an important biomarker in parkinsonian disorders [321]. Reduced myocardial MIBG uptake reflects cardiac sympathetic denervation, a hallmark of synucleinopathies such as Parkinson’s disease (PD), dementia with Lewy bodies [322], and pure autonomic failure [323,324]. In contrast, patients with atypical parkinsonian syndromes, including progressive supranuclear palsy and multiple system atrophy [325], typically demonstrate preserved or less severely reduced MIBG uptake, enhancing its utility in differential diagnosis [326]. Importantly, abnormal MIBG findings may precede motor symptom onset, supporting its role as an early non-motor biomarker of disease [327]. Despite limitations related to availability, cardiac comorbidities, and medication effects, MIBG scintigraphy

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provides complementary information to dopaminergic imaging and underscores the systemic nature of neurodegenerative movement disorders [328].

Emerging neuroimaging modalities, such as connectomics, diffusion tensor imaging, and hybrid PET/MRI, are redefining disease classification and offering potential biomarkers for early diagnosis and treatment response [329]. As neuroimaging continues to evolve, its integration with clinical phenotyping and genetic data promises to advance precision medicine in movement disorders [330].

14. Management of Movement Disorders

The management of movement disorders is multifaceted and requires an individualized approach based on the underlying diagnosis, symptom profile, disease stage, and patient goals [331]. Treatment strategies broadly encompass pharmacologic therapy, non-pharmacologic interventions, and surgical approaches, with an increasing emphasis on multidisciplinary care.

Pharmacologic management remains the cornerstone for most movement disorders. In Parkinson's disease (PD), dopaminergic therapies—including levodopa [332], dopamine agonists, monoamine oxidase-B inhibitors, and catechol-O-methyltransferase inhibitors—aim to restore dopaminergic tone and improve motor symptoms [333]. In hyperkinetic disorders, such as dystonia, chorea, and tics, treatment often targets neurotransmitter imbalance using anticholinergics, GABAergic agents, dopamine depletors, or antidopaminergic medications [334]. Botulinum toxin injections play a central role in focal dystonia and certain tremor syndromes, offering targeted symptom relief with minimal systemic effects; especially for oromandibular forms of dystonia [335] and spasticity [336]. Pimavanserin, a selective 5-HT_2A inverse agonist [337], treats Parkinson's disease psychosis without dopaminergic blockade and therefore does not worsen parkinsonism [338].

Amantadine may precipitate or worsen myoclonus through NMDA receptor antagonism and altered dopaminergic modulation [339], particularly in renal impairment or dose accumulation [340]. Fatigue is a common, multifactorial non-motor symptom in Parkinson's disease, driven by dopaminergic and non-dopaminergic dysfunction, sleep disturbance, mood disorders [341], and autonomic impairment; management focuses on optimizing dopaminergic therapy, treating contributing factors such as depression or sleep disorders, encouraging graded exercise, and selectively using agents such as modafinil or methylphenidate in refractory cases [342].

Vitamin deficiencies and excesses are increasingly recognized as reversible contributors to movement disorders, influencing basal ganglia and cerebellar function [343]. Deficiencies in vitamins such as B_{12}, E, and thiamine have been associated with parkinsonism, ataxia, dystonia, and chorea, while hypervitaminosis—particularly vitamin B_6—may lead to sensory neuropathy and abnormal movements [344]. Vitamin D deficiency has been associated with increased risk and severity of Parkinson's disease [345].

Non-pharmacologic interventions, including physical, occupational, and speech therapy, are essential components of comprehensive care. These therapies address gait instability, balance impairment, dysarthria, and functional decline, and are increasingly supported by evidence demonstrating improved mobility and quality of life [346].

Deep brain stimulation (DBS) is an established surgical therapy for selected patients with medication-refractory movement disorders [347]. DBS involves the implantation of electrodes into specific subcortical targets, most commonly the subthalamic nucleus or globus pallidus internus in PD, and the ventral intermediate nucleus of the thalamus in essential tremor [348]. In appropriately selected patients, DBS significantly reduces motor fluctuations, dyskinesias, and tremor, while allowing for medication reduction [349]. In dystonia, particularly primary and generalized forms, globus pallidus internus DBS can lead to sustained and progressive symptom improvement.

15. Future Studies

Future studies should aim to better define the mechanistic links between metabolic, genetic, and neurodegenerative processes underlying movement disorders, with particular emphasis on

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biomarkers that enable early and accurate diagnosis. Longitudinal, multicenter cohorts integrating clinical phenotyping, neuroimaging, and molecular data will be critical to delineate disease trajectories and identify predictors of progression and treatment response.

A key area of interest is the continued development and validation of seeding amplification assays (SAA) for α-synuclein [350]. Recent studies have demonstrated high sensitivity and specificity of α-synuclein SAA in cerebrospinal fluid for Parkinson's disease and related synucleinopathies, even in prodromal stages [351]. Future work should evaluate the performance of SAA across diverse populations, atypical parkinsonian syndromes, and secondary parkinsonism, including cases associated with metabolic or systemic conditions. Standardization of assay protocols and exploration of peripheral tissues—such as skin, olfactory mucosa, and gastrointestinal biopsy specimens—may further improve feasibility and accessibility.

Additionally, future investigations should assess whether SAA positivity correlates with specific movement disorder phenotypes, disease severity, or rate of progression, and whether it can serve as a stratification tool for clinical trials [352]. Combining SAA results with other biomarkers, including neurofilament light chain, inflammatory markers, and neuroimaging metrics, may enhance diagnostic precision and allow for more individualized therapeutic approaches [353].

Disease-modifying trials in Parkinson's disease (PD) remain a central focus of future research, aiming to slow or halt neurodegeneration rather than provide solely symptomatic relief [354]. Current investigational strategies target key pathogenic mechanisms, including α-synuclein aggregation, mitochondrial dysfunction, neuroinflammation, lysosomal impairment, and deficits in proteostasis [355]. Approaches under study include immunotherapies against misfolded α-synuclein, small molecules enhancing autophagy or lysosomal function, gene therapies addressing pathogenic variants such as GBA or LRRK2, and agents modulating oxidative stress and neuroinflammatory pathways [356]. Advances in biomarker development—particularly α-synuclein seeding amplification assays, neurofilament light chain, and advanced imaging—are increasingly enabling better patient stratification, earlier intervention, and more sensitive outcome measures [357]. Future disease-modifying trials will likely rely on biomarker-driven enrichment of at-risk or prodromal populations and adaptive trial designs, with the goal of shifting PD management toward earlier, personalized, and biologically targeted interventions [358].

Emerging evidence suggests that metformin and other repurposed medications may confer neuroprotective effects in movement disorders through anti-inflammatory, mitochondrial, and metabolic mechanisms [359]. Observational studies have linked metformin use with reduced risk and slower progression of Parkinson's disease, while agents such as statins, GLP-1 receptor agonists [360], urate-elevating therapies, calcium channel blockers, and immunomodulatory drugs have shown signals of disease modification in preclinical and early clinical studies [361]. Future research should prioritize well-designed randomized trials, biomarker-guided patient selection, and mechanistic studies to clarify therapeutic potential and define optimal intervention windows.

Lower serum uric acid levels have been consistently associated with increased risk and faster progression of Parkinson's disease [362]. Given its antioxidant properties, uric acid has been explored as a potential neuroprotective biomarker and therapeutic target, although interventional strategies aimed at urate elevation have thus far yielded mixed clinical results, underscoring the need for refined patient selection and biomarker-guided trials [363].

Future studies should increasingly focus on proteostasis, the cellular network that maintains protein synthesis, folding, trafficking, and degradation, as a unifying mechanism underlying many movement disorders [364]. Disruption of proteostatic pathways—including the ubiquitin–proteasome system, autophagy–lysosomal function, and molecular chaperones—contributes to the accumulation of misfolded and aggregation-prone proteins such as α-synuclein, tau, and huntingtin, which are central to both neurodegenerative and secondary movement disorders [365]. Translational research is needed to clarify how age, metabolic stress, inflammation, and genetic variation impair proteostasis in vulnerable motor networks such as the basal ganglia and cerebellum. Future studies should also explore proteostasis-targeted biomarkers and therapeutics, including enhancers of

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autophagy, modulators of protein clearance, and interventions that restore proteome balance, with the goal of shifting treatment strategies toward disease modification and neuroprotection rather than purely symptomatic control.

16. Conclusions

Secondary movement disorders constitute a broad and clinically significant spectrum of conditions arising from medications, systemic illnesses, metabolic and endocrine abnormalities, vascular lesions, infections, autoimmune disorders, and toxic exposures. Despite their etiologic diversity, these disorders converge on shared pathophysiological disruptions of basal ganglia–thalamo–cortical and cerebellothalamic networks, mediated through alterations in dopaminergic, GABAergic, glutamatergic, and monoaminergic signaling. Clinically, secondary movement disorders frequently mimic primary neurodegenerative syndromes, leading to diagnostic challenges and potential delays in appropriate treatment. Careful clinical phenomenology, comprehensive medication and systemic evaluation, and targeted use of neuroimaging and laboratory testing are therefore essential to differentiate secondary causes from primary movement disorders.

A key clinical implication of this review is the potential reversibility of many secondary movement disorders when the underlying cause is promptly identified and addressed. Withdrawal of offending drugs, correction of metabolic derangements, treatment of infections or autoimmune processes, and optimization of vascular or systemic disease often result in substantial motor improvement and prevention of long-term disability. Advances in neuroimaging and biomarker research are further refining diagnostic precision and reinforcing the concept of movement disorders as network-based diseases rather than isolated basal ganglia pathologies. Future studies aimed at integrating clinical phenotyping with molecular biomarkers and network-level imaging may improve early recognition, guide targeted interventions, and ultimately bridge mechanistic understanding across primary and secondary movement disorders.

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