# Movement disorders in cerebrovascular disease

*Raja Mehanna, Joseph Jankovic*

**Movement disorders can occur as primary (idiopathic) or genetic disease, as a manifestation of an underlying neurodegenerative disorder, or secondary to a wide range of neurological or systemic diseases. Cerebrovascular diseases represent up to 22% of secondary movement disorders, and involuntary movements develop after 1–4% of strokes. Post-stroke movement disorders can manifest in parkinsonism or a wide range of hyperkinetic movement disorders including chorea, ballism, athetosis, dystonia, tremor, myoclonus, stereotypies, and akathisia. Some of these disorders occur immediately after acute stroke, whereas others can develop later, and yet others represent delayed-onset progressive movement disorders. These movement disorders have been encountered in patients with ischaemic and haemorrhagic strokes, subarachnoid haemorrhage, cerebrovascular malformations, and dural arteriovenous fistula affecting the basal ganglia, their connections, or both.**

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**Parkinson's Disease Center and Movement Disorders Clinic, Department of Neurology, Baylor College of Medicine, Houston, TX, USA**
(R Mehanna MD, Prof J Jankovic MD)

Correspondence to:
Prof Joseph Jankovic, Department of Neurology, Baylor College of Medicine, Houston, TX 77030, USA
**josephj@bcm.edu**

## Introduction

Movement disorders can be divided into hyperkinetic disorders dominated by excessive, abnormal involuntary movements and hypokinetic disorders manifested by paucity or slowness (bradykinesia) of movement.[1] These can be further subdivided into primary, without an identifiable cause, or secondary, due to various disorders. Cerebrovascular diseases represent up to 22% of all secondary movement disorders[2] and abnormal movements develop as residual complications in 1–4% of all patients after strokes (panel).[3–5]

Movement disorders secondary to cerebrovascular diseases are diverse and differ from their idiopathic counterpart not only in their clinical presentation but also in their natural history, prognosis, and treatment. Randomised therapeutic trials of movement disorders related to cerebrovascular diseases are lacking, partly because of their substantial heterogeneity and relative rarity and, therefore, available data stem mainly from retrospective studies and case reports. As our understanding of the mechanisms of movement disorders related to cerebrovascular diseases is changing, a comprehensive and critical review of the published literature on this topic is needed.

## Functional anatomy of the basal ganglia

Since movement disorders related to cerebrovascular diseases are often associated with lesions in the basal ganglia, we provide a brief overview of the motor circuitry of the basal ganglia. Various motor and non-motor cortical areas project primarily to the striatal medium spiny neurons and the subthalamic nucleus, with the globus pallidus pars interna serving as the major output nucleus, which connects back to the cortex via the thalamus. There are two major projections from the striatum: the direct pathway to the globus pallidus pars interna and the indirect projection to the globus pallidus pars interna via the globus pallidus pars externa and the subthalamic nucleus (figure). Modulated by the substantia nigra pars compacta, the indirect pathway exerts surround inhibition and thus facilitates an excitatory drive to muscles responsible for a given movement and suppresses unwanted motor activity not relevant to the primary movement. Thus, hypokinetic disorders are thought to result from overactivation of the indirect pathway leading to an increased output from the globus pallidus pars interna, whereas overactivation of the direct pathway results in low neuronal output from the globus pallidus pars interna leading to hyperkinetic disorders (figure). This model of the basal ganglia and its connections is, of course, an oversimplification of a complex network that, when disrupted, can result in a range of motor abnormalities.[6]

Although the basal ganglia appear to play an important part in normal motor control, severe damage to the basal ganglia caused by acute lesions such as stroke does not always lead to development of a movement disorder. In a review of 820 consecutive patients with deep hemispherical infarct,[7] 13 of whom had pure lenticular infarct, none developed a movement disorder. There are several other reports of basal ganglia infarcts without evidence of any post-stroke abnormal movement.[8–11] In the Lausanne stroke registry of 2500 patients,[3] only 29 (1%) had post-stroke movement disorder attributed to basal ganglia involvement after an ischaemic or haemorrhagic stroke. Why post-stroke movement disorders are relatively rare, even with marked basal ganglia damage, is unclear. This observation, however, raises the possibility of individual susceptibility, remarkable brain plasticity within the basal ganglia anatomical networks partly as a result of parallel processing, and compensatory mechanisms that provide certain resilience and protection against clinically evident loss of motor control.[12,13]

There is emerging evidence that most movement disorders result not from a single lesion, but from a network dysfunction and abnormalities in functional connectivity.[14] For example, akinetic-rigid motor signs of idiopathic Parkinson's disease are characterised by increases in pallidothalamic and pontine metabolic activity, decreases in premotor cortex, supplementary motor area, and a decrease on parietal associative area activity on $^{18}$F-fluorodeoxyglucose PET.[15] These characteristics have been interpreted as the consequence of nigrostriatal dopaminergic deficit and consequent

**Panel: Movement disorders reported after cerebrovascular events**

- Parkinsonism
  - Vascular parkinsonism
  - Progressive supranuclear palsy
  - Isolated freezing of gait
  - Corticobasal syndrome
- Chorea-ballism-athethosis
- Dystonia
- Tremor
- Myoclonus
- Asterixis
- Transient limb shaking
- Stereotypies
- Akathisia
- Tics

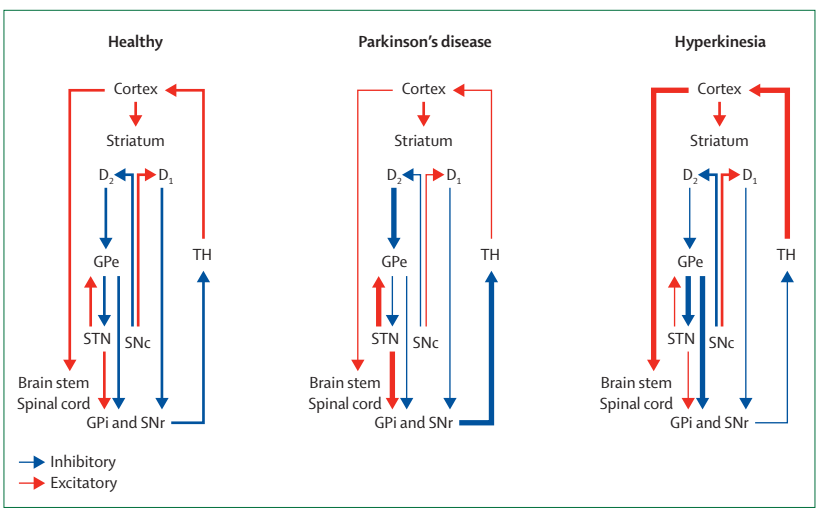


***Figure:*** **Models of basal ganglia dysfunction**
STN=subthalamic nucleus. GPi=globus pallidus pars interna. GPe=globus pallidus pars externa. SNc=substantia nigra pars compacta. TH=thalamus. SNr=substantia nigra pars reticulata. $D_1$=dopamine receptor D1. $D_2$=dopamine receptor D2.

functional abnormalities in the motor cortico-striato-pallido-thalamo-cortical loop and related neural pathways. Parkinson's disease tremor, on the other hand, is characterised by increased metabolic activity in the cerebellum and dorsal pons, the primary motor cortex, and, to a lesser degree, in the caudate and putamen, suggesting a dysfunction of the cerebello-thalamo-cortical pathways.[15,16] Furthermore, resting state functional MRI studies have shown increased functional connectivity between the subthalamic nucleus and cortical motor regions in patients with Parkinson's disease with and without resting tremor.[17] In idiopathic dystonia, however, functional imaging has revealed relative increases in the metabolic activity of the posterior putamen, globus pallidus, cerebellum, and the supplementary motor area.[15]

## Epidemiology

The frequency of post-stroke abnormal movements has been estimated to vary between 1% and 4% of all strokes,[3–5] affecting both sexes equally.[3,4,11] The mean age at onset is typically in the sixth and seventh decades of life, but can range from early childhood to age 90 years.[3,4,11] In a series of 93 patients with a thalamic stroke,[11] 35 of whom developed post-stroke abnormal movements, no difference in age, sex, side of the lesion, or stroke risk factors correlated with the development of the abnormal movements. However, patients who did develop post-stroke abnormal movements had significantly more frequent severe motor ($p<0·05$) and sensory ($p<0·01$) deficits during the acute stage of stroke.[11]

Post-stroke abnormal movements are usually unilateral, and contralateral to the putative brain lesion (83% in one series)[3] or bilateral. However, unilateral abnormal movements ipsilateral to the brain lesion have occasionally been described.[18–21] This phenomenon is, of course, well recognised with ipsilateral cerebellar lesions.[3]

Hemichorea, with or without hemiballism, is the most frequent post-stroke hyperkinetic movement disorder, followed by dystonia.[3,4] In the Lausanne stroke registry,[3] 38% of post-stroke hyperkinetic movement disorders were hemichorea, 17% were dystonia, 10% were limb-shaking, 10% were myoclonus–dystonia, 7% were stereotypies, 7% were asterixis, 3% were myoclonus, 3% were tremor, 3% were hemiakathisia, and 3% were dysarthria and dyskinetic hand.[3] In another registry from Ecuador,[4] 56 (4%) of 1500 patients developed post-stroke abnormal movements: 36% developed chorea, 29% dystonia, 25% tremor, and 11% parkinsonism. Differences in the frequency of various movement disorders between the two registries could be related to the different populations studied as well as to the fact that the Swiss study evaluated patients only for hyperkinetic disorders, and therefore parkinsonism would not be registered. In any case, these were retrospective studies and patients who did not seek medical attention at these centres for new onset movement disorders would have been missed. Thus the frequency of post-stroke movement disorders is likely to be underestimated.

## Prognosis

The natural history and progression of post-stroke movement disorder varies. Post-stroke abnormal movements can occur immediately at the onset of the acute stroke[22–25] or can be delayed and progressive.[11,26–30] Notably, abnormal movements can also be part of the acute stroke presentation, especially in hemiballism and asterixis.[3] The initial movement disorder occurring during or shortly after an acute stroke might be hemiballism, which then evolves into hemichorea, and later to hemidystonia. In some cases, particularly those occurring in childhood, hemidystonia can be associated with hemiatrophy.[31] The latency between the acute stroke

and the onset of abnormal movement varies from less than a day to several years after the stroke.[3–5,10,11,28] Although this latency seems to depend in part on the type of movement disorder, with chorea developing the earliest and parkinsonism the latest in a study of 56 patients (4·3 *vs* 117·5 days, p<0·05),[4] latency still varies widely within each movement disorder.[5] In a study of 26 patients with hemidystonia-hemiatrophy syndrome,[31] ten of whom had a documented stroke before the onset of movement disorder, the mean age at onset of hemidystonia was 14·9 years (range 1–46) and the mean latency from onset of hemiparesis to onset of hemidystonia was 14·7 years (2 weeks to 46 years). Often preceded by a static period, hemidystonia-hemiatrophy syndrome can also represent delayed-onset progressive movement disorder.[11,28] This increasingly recognised, but still poorly understood, phenomenon of delayed-onset progression can occur not only after stroke but also after other static lesions, such as perinatal asphyxia[32,33] and brain injury.[34]

Age at the time of the initial injury seems to be an important factor in the phenomenology and latency of the observed movement disorders.[28,35] In a series of 53 patients with delayed onset movement disorders after a static brain injury, including stroke, Scott and Jankovic[28] found that younger age at onset of the injury was associated with a longer latency. For example, the two patients who had a stroke as infants had a latency of 42·8 years before the onset of dystonia ipsilateral to the initial hemiparesis, whereas the adult group had a mean latency of only 1·4 years. Scott and Jankovic[28] also noted that younger age at initial injury was associated with a greater tendency to develop generalised rather than focal or segmental dystonia. Bhatt and colleagues[35] reported a series of six patients in whom anoxic brain injury occurring at age 21 years or younger was complicated by the development of dystonia; by contrast, anoxia at age 33 years or later resulted in an akinetic-rigid syndrome. In a registry from Ecuador, Alarcon and colleagues[4] noted that older stroke patients tended to develop chorea, whereas younger patients were more likely to develop dystonia after stroke.

The pathophysiology of delayed onset movement disorders after a static brain lesion is still poorly understood, but several hypotheses have been suggested, including trans-synaptic neuronal degeneration, inflammatory changes, oxidative stress, ephaptic transmission, remyelinisation, central synaptic reorganisation, denervation supersensitivity, and neuroplasticity.[28,36] Dendritic plasticity and changing patterns of synaptic activity after brain injury could change normal neuronal circuitry and allow the development of delayed onset, progressive movement disorder. Brain metabolism in response to anoxic-ischaemic brain injury also varies with age at the time of injury.[28,37,38] Age-related differences in brain metabolism and neuroplasticity might partly explain the observed relation between age at initial brain insult and subsequent latency and distribution of delayed onset movement disorders. Another possibility is that loss of proprioceptive input from multiple joints might cause impaired synergic stabilisation and contribute to the abnormal movement.[11] Loss of inhibitory influence of the globus pallidus pars interna and substantia nigra pars reticulata on the thalamus has also been postulated to play a part in the pathophysiology.[39]

## Lesion location, cause, and outcome

A vascular event can result in an abnormal movement when several different locations in the brain are affected. In the Lausanne stroke registry,[3] movement disorders secondary to a stroke affecting the basal ganglia were most often secondary to striatal and pallidal lesions (44%), followed by thalamic involvement (37%). Although the basal ganglia are most often involved in post-stroke movement disorders, no specific locations of lesions are reliably predictable of a particular movement disorder.[11,40,41] Furthermore, the same movement disorder can be caused by lesions in different parts of the brain.[3,5,13] For example, hemiballism, traditionally described in lesions of the subthalamic nucleus, can also be associated with lesions in other parts of the brain.[42] Likewise, hemidystonia and hemichorea or hemiathetosis can result from lesions involving areas other than the lentiform nucleus or the caudate, respectively.[3] An isolated lesion correlates with an involuntary movement in less than half of patients.[4] Finally, one cerebrovascular event can cause a combination of different movement disorders in the same patient, such as a combination of chorea, athethosis, and dystonia after thalamic stroke.[10,11]

The most common subtype of stroke leading to abnormal movements is small vessel disease with small deep infarcts,[3,4,25] but cardiac embolism as well as large and medium vessel atherothrombosis have been also reported to cause movement disorders.[4,11,43] Parenchymal or subarachnoid haemorrhages have also been implicated, although less frequently than ischaemic events.[4] Chorea and hemiballism have been described in patients with moyamoya disease.[44] Various hypokinetic and hyperkinetic movement disorders have been also attributed to watershed or border zone infarcts. These infarcts develop in low perfusion areas located at the borders between vascular territories supplied by different arteries, affecting the frontal subcortical region with or without striatal involvement associated with cerebral hypoperfusion.[45]

Patients can develop weakness immediately after the vascular event and the abnormal movement typically emerges during the recovery phase while the motor deficit is improving.[3,4,10,11,39,46,47] Pseudoathetosis associated with proprioceptive deficit can also be a presentation of movement disorder related to cerebrovascular disease,[5,11,39] although it is quite rare.[3,47]

## Vascular parkinsonism

### Clinical presentation

Initially labelled arteriosclerotic parkinsonism by Critchley,[48] parkinsonism of vascular origin was later differentiated from degenerative parkinsonism in neuropathological studies,[49] clinicopathological series,[50] and detailed clinicoanatomic reports.[51] Vascular parkinsonism has been used to describe a heterogeneous group of conditions that manifest clinically in parkinsonian features, but are presumably of vascular cause as suggested by history or multiple strokes. However, a clear history of acute neurological deficits or obvious radiological evidence of previous strokes may not be apparent. In these cases microvascular pathology can be demonstrated at autopsy. Vascular parkinsonism, which accounts for 3–12% of all cases of parkinsonism,[52–54] has an older age at onset than other parkinsonian syndromes.[55] In our database of 10 153 patients, vascular parkinsonism was the second most frequent cause of parkinsonism, after Parkinson's disease (table 1).

The classical form of vascular parkinsonism is described as lower body parkinsonism[56] because it affects predominantly the legs with broad-based, shuffling, and often freezing gait and postural instability.[57] It is usually bilateral, non-tremulous, and frequently associated with pyramidal signs, pseudobulbar palsy, incontinence, dementia, diabetes, and hypertension.[25,56,58–62]

In an Italian multicentre, cross-sectional study,[62] 158 patients with possible Parkinson's disease or vascular parkinsonism were enrolled to study the relation between cerebrovascular disease and parkinsonism. Vascular parkinsonism was defined as insidious onset of symmetrical lower body parkinsonism, although 59% had asymmetric onset. Only 48% responded to levodopa; poor response was associated with symmetric onset of motor symptoms, worse disease severity, absence of dyskinesia, and increased number of vascular risk factors. The frontal lobe was most involved with vascular lesions, and 70% had abnormal dopamine transporter (DAT) SPECT or DAT scan. Patients with hypertension, vascular lesions in the basal ganglia and in the periventricular regions, and those who had normal DAT uptake had the worst prognosis.

| | Number of patients |
|---|---|
| Parkinson's disease | 7467 |
| Vascular parkinsonism | 816 |
| Progressive supranuclear palsy | 616 |
| Multiple system atrophy | 562 |
| Dementia with Lewy bodies | 332 |
| Other cases of parkinsonism | 314 |
| Drug-induced parkinsonism | 282 |
| Corticobasal degeneration | 224 |
| Hemiparkinsonism | 85 |
| Post-encephalitic parkinsonism | 22 |
| Carbon monoxide poisoning | 9 |
| Manganese poisoning | 9 |
| Brain tumour | 6 |
| Familial basal ganglia calcification | 5 |

***Table 1*: Clinical diagnoses in 10 153 patients presenting with parkinsonism at Baylor College of Medicine, 1988–2013**

Vascular parkinsonism can be sometimes difficult to distinguish from the postural instability and gait disorder (PIGD) subtype of idiopathic Parkinson's disease.[63,64] PIGD, however, usually manifests in narrow-based rather than wide-based gait, greater upper body involvement, and more cognitive impairment compared with vascular parkinsonism, and the bradykinesia and rigidity usually improve with levodopa.

The coexistence of lower body parkinsonism on examination and cerebrovascular disease on imaging with evidence of stroke-like lesions in or near the basal ganglia should raise the possibility of vascular parkinsonism,[65] but consensus on diagnostic criteria is lacking.[54] Diagnosis of vascular parkinsonism is supported by preserved olfactory function[66] and normal DAT scan.[62,67–69] However, DAT deficit can be present in vascular parkinsonism, but tends to be more symmetric than in Parkinson's disease.[70] Although up to a half of patients with vascular parkinsonism improve with levodopa,[71] a robust response would favour the diagnosis of idiopathic Parkinson's disease.[5]

### Stroke location and cause

Vascular parkinsonism has been mostly associated with unilateral[23,24,72] or bilateral[23,25,51] infarcts in the striatum, lentiform nucleus, or pons. Most frequently, the lesions observed are multiple lacunes affecting the basal ganglia and hemispheric white matter, but sparing the substantia nigra.[58] A study of 14 patients[73] showed that extensive white matter lesions on imaging correlated well with the development of vascular parkinsonism, particularly if there was a marked involvement of the striatum. However, vascular parkinsonism could be secondary to cerebral microinfarcts that might not be detected by conventional structural MRI.[74,75] Parkinsonism has also been described in Binswanger's disease (a form of leukoencephalopathy caused by hypoxia-ischaemia of distal watershed periventricular territories associated with ageing, hyperviscosity, and increased fibrinogen concentrations),[76] CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy), and moyamoya disease (spontaneous occlusion of the circle of Willis with hypertrophied lenticulostriate collateral vessels in the basal ganglia).[44] Temporally, two forms of vascular parkinsonism have been described: one with acute onset, associated with basal ganglionic infarcts, and another with insidious onset, associated with more chronic and diffuse subcortical white matter ischaemia and usually involving the striatum, lentiform nucleus, or pons.[4,5,25,58,77] The second form of progression is seen in about two-thirds of patients with vascular parkinsonism, and has a more relentless rather than stepwise progression.[55]

### Prognosis and treatment

Overall, only a third to half of patients with vascular parkinsonism improve with conventional dopaminergic therapy.[53,56,59,71,78] Patients with vascular lesions in or close to the nigrostriatal pathway and rare cases with abnormal DAT SPECT imaging are more likely to improve with levodopa.[62,79,80] In a series of six patients with acute vascular parkinsonism, only one recovered completely after 2 years and the other five progressively worsened.[4] The response to carbidopa-levodopa was moderate at best and did not last more than 6 months. Four of the five patients had early fluctuations and dyskinesias between 1 and 9 months after starting levodopa.[4] On the other hand, in another series of six patients with hemiparkinsonism after anterior cerebral artery infarction, spontaneous improvement was reported in all despite no response to carbidopa-levodopa.[46] Some patients with vascular parkinsonism had a transient improvement in their gait after removal of 35–40 mL of CSF,[81] but this finding has not been confirmed by others. Management should focus on physical and occupational therapy as well as detection and treatment of atherosclerosis, hypertension, diabetes mellitus, and other stroke risk factors.[5]

## Atypical forms of vascular parkinsonism

A new form of vascular parkinsonism secondary to a dural arteriovenous fistula has been described.[82–87] In addition to cognitive decline, this form of parkinsonism can also be associated with headache, tinnitus, vertigo, ataxia, urinary incontinence, or visual disturbance. Antiparkinsonian drugs, including levodopa, are usually not beneficial,[83,84,87] but motor and cognitive symptoms improve after surgical or endovascular treatment of the vascular malformation in almost half of cases. The suggested mechanism includes hypoperfusion oedema secondary to the vascular abnormality.

Progressive supranuclear palsy is characterised by ophthalmoparesis (affecting preferentially downward gaze), akinesia, rigidity, postural instability with early falls, and pseudobulbar symptoms with dysarthria and dysphagia. Vascular progressive supranuclear palsy, secondary to multiple infarcts involving the basal ganglia, thalamus, or rostral brainstem,[88] is the second most frequent cause of this syndrome after idiopathic progressive supranuclear palsy. By contrast with idiopathic progressive supranuclear palsy, vascular progressive supranuclear palsy is more likely to have asymmetric and predominantly lower-body involvement, cortical and pseudobulbar signs, dementia, and bowel and bladder incontinence.[89] It also has a much higher frequency of stroke risk factors and vascular lesions on MRI. Two patients with a presentation suggestive of progressive supranuclear palsy were found to have Binswanger's disease and responded to levodopa.[90]

Isolated freezing of gait secondary to vascular disease has been anecdotally reported, with one case secondary to a bilateral pedunculopontine nuclei stroke[91] and one case secondary to dural arteriovenous fistula causing congestion of the globus pallidus.[92]

Corticobasal syndrome is characterised by asymmetric levodopa-resistant parkinsonism associated with cortical dysfunction such as apraxia, dementia, aphasia, and other movement disorders. It has rarely been reported as secondary to extensive diffuse chronic ischaemic cerebral disease.[93,94]

## Chorea, ballism, and athetosis

### Clinical presentation

Chorea consists of involuntary, continual, and irregular movements that flow randomly from one body part to another, and ballism is a form of flinging high-amplitude and coarse chorea.[1] Hemichorea and hemiballism share similar pathophysiology and are treated with the same drugs.[42] Athetosis can be viewed as a slow form of chorea that consists of non-patterned, writhing movements.[1] Hemichorea, hemiballism, and athetosis can develop during the acute stage of stroke,[30] although in many cases they emerge as the motor deficit improves. Hemiballism can be an acute stroke manifestation, like hemiplegia or aphasia.[95]

Athetosis is uncommonly seen after a stroke, and is often a component of a mixed involuntary movement disorder such as dystonia–athetosis or choreo-athetosis.[11,13,96] It should be distinguished from pseudo-athetosis, which is due to a loss of proprioception that could be secondary to a thalamic infarct[11,96,97] as well as a spinal cord lesion or neuropathy.[97]

### Stroke location and mechanisms

Overall, hemichorea has been reported mostly after vascular events affecting the lentiform nucleus or the thalamus (table 2).[3,4,30,100,101] One patient with right upper limb chorea was found to have a cavernous malformation in the head of the contralateral caudate nucleus,[161] and another had hemichorea secondary to contralateral carotid stenosis that resolved after endovascular carotid stenting.[162] Despite the traditional belief that the subthalamic nucleus is the typical anatomical correlate for hemiballism, this hyperkinetic movement disorder has been described following lesions in other locations.[3,4,42,104,118–120] The subthalamic nucleus was the site of pathology in only six (27%) of 22 patients with hemiballism in one radiological series: 27% of patients had lesions in other basal ganglia structures but not the subthalamic nucleus, three (14%) patients had temporal or cortical lesions, or both, and seven (32%) patients had normal imaging.[163]

### Prognosis and treatment

Although usually self-limited, post-stroke chorea and ballism can substantially increase morbidity through injury and impaired coordination and might, therefore, need short-term treatment. Pharmacological therapy consists chiefly of antidopaminergic therapy with

| | Most frequent location of stroke | Other reported locations | Treatment options |
|---|---|---|---|
| Vascular parkinsonism | Striatum, lentiform nucleus or pons, unilateral[23,24,72] or bilateral[51,22,25] | Frontal cortex and subcortical region[4,46,98,99] | Levodopa improves symptoms in a third to half of patients;[56,78,71,53,59] physical therapy, occupational therapy, control of cardiovascular risk factors[5] |
| Vascular progressive supranuclear palsy | Multiple infarcts involving the thalamus or the basal ganglia[88] | .. | Physical therapy, occupational therapy, control of cardiovascular risk factors |
| Vascular corticobasal syndrome | Extensive diffuse chronic ischaemic cerebral disease[93,94] | .. | Physical therapy, occupational therapy, control of cardiovascular risk factors |
| Chorea | Lentiform nucleus or thalamus[3,4,30,100,101] | Subthalamus, striatum, posterior limb of internal capsule, corona radiata, frontal lobe, parietal lobe, temporal cortex, external capsule, and pons[3,5,102,103] | Neuroleptics, traditional (haloperidol, pimozide, perphenazine, and fluphenazine), or newer generation (such as olanzapine, quetiapine, clozapine);[3,104–110] tetrabenazine;[111] clonazepam;[109] sodium valproate;[109] topiramate;[112] stereotactic lesion or DBS of Vop or Vim[5,113–117] |
| Hemiballism | Thalamus or striatum more frequently than subthalamus[3,4,42,104,118–120] | Anterior cortical parietal artery without involvement of deep structures[121] | As for chorea |
| Dystonia | Lenticular, especially putamen[4,12,40,41,47,96] | Thalamus,[122–124] caudate, internal capsule, frontal lobe, parieto-occipital lobe,[3,5,125] brainstem,[26] cerebellum,[126,127] spinal cord,[128] and cerebellothalamic pathways[129] | Botulinum toxin,[130] anticholinergic drugs,[47] benzodiazepines,[3] baclofen, typical and atypical neuroleptics,[47,131] tetrabenazine,[132] stereotactic lesion or DBS of the globus pallidus or thalamus[47] |
| Cerebellar outflow tremor | Posterior thalamus or lesions disrupting the dentatorubrothalamic pathway [5,39,133–135] | Subthalamus, striatum, frontal or parietal lobes, or deep nuclei of the cerebellum.[4,5,136] | Propranolol and primidone, alone or in combination;[5,137] clonazepam;[5] sodium valproate;[5] ceruletide;[138] lisuride;[139] levetiracetam;[140] anticholinergics;[133] adding weight to the affected limb;[141] levodopa (if resting tremor);[142,143] DBS of Vim,[5,144] Vop,[113] or both[145] |
| Holmes tremor | Dentatorubrothalamic, cerebellothalamic, or nigrostriatal pathways[30,136,146,147] | .. | As for cerebellar outflow tremor |
| Myoclonus (usually focal or segmental) | Midbrain, pons, or thalamus[4,5,13,29] | .. | Clonazepam; sodium valproate, levetiracetam, piracetam, primidone, tetrabenazine, acetazolamide, botulinum toxin[131,141,148] |
| Asterixis | Thalamus, basal ganglia, frontoparietal cortex, cerebellum and brainstem[3,5,46,149,150] | .. | Usually improves spontaneously after a few days[150] |
| Transient dyskinesias | Severe carotid or vertebro-basilar stenosis[3,13,151–154] | .. | Endarterectomy[3,13,151] |
| Stereotypies | Parietal, lenticulo-striatal, thalamic, midbrain, or left middle cerebral artery territory;[3,155–157] most often unilateral | Large infarcts in the territory of the internal carotid artery, middle cerebral artery, or the anterior cerebral artery[157] | Clonazepam, tetrabenazine[131,132] |
| Akathisia | Posterior thalamus[3] | .. | Clonazepam[3] |
| Tics | Striatum, globus pallidus, frontal or parietal cortex[158,159] | Left frontal lobe haemorrhage secondary to an arteriovenous malformation[160] | Clonidine;[160] neuroleptics[44] |

DBS=deep brain stimulation. Vop=nucleus ventralis oralis posterior of the thalamus. Vim=nucleus ventralis intermedius of the thalamus.

***Table 2:*** **Anatomical location and treatment of post-stroke movement disorders**

traditional and newer generation of neuroleptics that act by blocking dopamine receptors (table 2).[3,104–110] These agents, although usually effective, carry the risk of drug-induced parkinsonism and tardive dyskinesia and, therefore, tetrabenazine, a dopamine-depleting agent, has become the preferred choice because it does not cause tardive dyskinesia.[111,131,132] Clonazepam and sodium valproate have also been used with some success[109] as well as topiramate, tested in a small number of patients.[112] Stereotactic lesion as well as deep brain stimulation to the thalamic nuclei ventralis oralis posterior or intermedius seems to confer excellent control of medically refractory post-stroke hemichorea-athetosis and hemiballism.[5,113–116] Unilateral pallidotomy was also reported to be beneficial in one patient with contralateral post-stroke monochorea.[117] Superficial temporal artery-middle cerebral artery bypass has been reported to improve chorea in patients with moyamoya disease.[45]

In a small series of ten to 20 patients with chorea or ballism, complete improvement was reported in 45–56% on one or two of these drugs[3,108] and partial improvement was reported in 37–45% of patients.[3,108] In another series of 18 patients with focal chorea or hemichorea, two (11%) spontaneously recovered completely and 15 (83%) partially.[4]

Non-ketotic hyperglycaemic chorea, which is rarely the initial manifestation of diabetes mellitus, can mimic basal ganglia stroke.[164] It usually improves with aggressive metabolic control normalising blood glucose, although tetrabenazine might also be helpful.[165]

## Dystonia

### Clinical presentation

According to a recent consensus update, "dystonia is a movement disorder characterised by sustained or intermittent muscle contractions causing abnormal, often repetitive, movements, postures, or both. Dystonic movements are typically patterned, twisting, and may be tremulous. Dystonia is often initiated or worsened by voluntary action and associated with overflow muscle activation [involuntary activation of muscles that are not required to perform a given movement]".[166] The second

most frequent post-stroke movement disorder after chorea,[3] post-stroke dystonia, is frequently delayed in onset.[28,30] Post-stroke dystonia can be focal, affecting one hand, one foot, or the cranial area,[4,11,122,126] and can be segmental or even generalised; typically it is unilateral (hemidystonia) and contralateral to the lesioned hemisphere.[3,4,11,13,47] When the initial insult occurs in childhood, hemidystonia can be associated with hemiatrophy, the so-called hemidystonia-hemiatrophy syndrome.[31] Another variant of post-stroke dystonia is dystonic myoclonus. This disorder was described in three patients as a "jerky dystonic unsteady hand" secondary to lesions in the posterior thalamic nuclei due to posterior choroidal artery infarcts.[167] Myoclonus dystonia was also described in four patients with infarcts in the ventral intermediate and posterior thalamic nuclei.[123] Dystonic tremor or dystonic athetoid movements[5,13,168] and paroxysmal dystonia have also been described after stroke.[169,170] Post-stroke dystonia is often associated with hypertonicity due to underlying spasticity and the term spastic dystonia is sometimes used to describe this combination of dystonia-spasticity.

### Stroke location and mechanisms

Lenticular lesions are the most common lesions that result in dystonia, especially when they involve the putamen.[4,12,40,41,47,96] In addition to thalamic nuclei ventralis intermedius and posterolateral thalamic lesions,[10,123,149] dystonia has been also described after vascular lesions in the brainstem-diencephalic junction; the latter manifests in blepharospasm, oromandibular dystonia, or other combinations of cranial dystonia.[26] Recurrent hemidystonia has been reported in some cases of childhood or adult moyamoya disease.[5,171]

### Prognosis and treatment

The treatment of stroke-induced dystonia is symptomatic and does not differ from that for idiopathic dystonia. Botulinum toxin can be useful in focal or segmental dystonia and is frequently used to treat spasticity-dystonia that occurs after a stroke or in patients with cerebral palsy.[130] In patients with hemidystonia or more generalised dystonia, systemic drugs, intrathecal baclofen, and deep brain stimulation, or a combination of the different approaches, are often needed to provide at least partial relief (table 2). Surgical procedures targeting the globus pallidus or the thalamus, whether ablative or deep brain stimulation, seem to yield the best response,[47] but the most effective target remains to be determined. Deep brain stimulation targeting the internal capsule improved the controlateral stroke-induced foot dystonia in one patient, with sustained benefit at 2 years.[116]

In 15 patients with focal or hemidystonia, five (33%) spontaneously improved completely and ten (67%) partially.[4] Myoclonic dystonia seems particularly resistant to drugs or spontaneous remissions.[3]

## Tremor

### Clinical presentation

Tremor is a rhythmic, oscillatory movement produced by alternating or synchronous contractions of antagonist muscles that can occur at rest or during action. Action tremor is further divided into postural, seen when holding the limb against gravity, or kinetic, seen when moving the limb.[1] Post-stroke tremor generally occurs on action although some patients can have a mixture of rest, postural, and kinetic tremor.[11,39,46,101] It is most often multifocal or segmental and unilateral rather than focal or generalised.[4,5,39,46,101,133] Isolated post-stroke tremor that is unaccompanied by other abnormal movements is rare.[11,39,133,172] Although typically a delayed consequence of a stroke, tremor as part of the acute stroke presentation has been anecdotally reported.[3]

### Stroke location and mechanisms

Tremor is most often caused by lesions in the posterior thalamus or lesions disrupting the dentatorubro-thalamic, cerebellothalamic, or nigrostriatal pathways[5,39,133–135] (table 2). Tremor occurring only when writing (task-specific writing tremor) has been described after a small frontal cortical infarct.[173] A rest tremor that becomes more severe on maintaining a posture and most severe during movement is called rubral, midbrain, or Holmes tremor, although a lesion of the red nucleus has not always been identified.[136,146] Strokes involving the posterior circulation can involve the thalamus, producing slow (1–3 Hz) rest and postural tremors, referred to as myorhythmia.[39] The presence of myorhythmia, which superficially resembles Parkinson's disease tremor, but is slower in frequency, usually indicates a vascular or other lesion in the thalamus, rostral brainstem (particularly the substantia nigra), inferior olivary nucleus, and the cerebellum.[174] Lesions involving the Guillain-Mollaret triangle linking the dentate nucleus, red nucleus, and inferior olivary nucleus, can be also associated with palatal myoclonus, another slow, 1–3 Hz, rhythmical movement, often associated with hypertrophy of the inferior olivary nucleus.[175]

### Prognosis and treatment

Post-stroke tremor is particularly refractory to pharmacotherapy (table 2).[5] Propranolol and primidone, alone or in combination, are the first line treatment in essential tremor, but are rarely beneficial in the post-stroke cerebellar-outflow tremors.[5,137] Adding weight to the affected limb (eg, wrist weights) can also dampen the tremor.[141] In severe cases, deep brain stimulation targeting the thalamic nuclei ventralis intermedius,[5,144] ventralis oralis posterior,[113] or both,[145] or the lenticular fasciculus[176] could be the best therapeutic options. In a series of 14 patients with focal or unilateral tremor, four (29%) improved completely and ten (71%) partially.[4]

## Myoclonus

### Clinical presentation

Myoclonus refers to sudden, involuntary, brief, irregular, jerk-like contractions (positive myoclonus) or inhibition of contraction (negative myoclonus) of certain muscle groups. Segmental myoclonus consists of rhythmical contractions of muscles anatomically related to brainstem nuclei or a spinal cord segment.[177]

### Stroke location and mechanisms

Post-stroke myoclonus is usually focal or segmental with lesions in the midbrain, pons, or thalamus.[4,5,13,29] Secondary palatal myoclonus, a form of focal myoclonus affecting primarily the levator veli palatini muscle, has been described in pontine or bulbar strokes,[13] but was also reported in one patient with lateral thalamic infarction.[178] Generalised post-stroke myoclonus has not been reported.[5,13]

### Prognosis and treatment

Myoclonus is most frequently treated with clonazepam and sodium valproate, but levetiracetam, piracetam, primidone, tetrabenazine, acetazolamide, and botulinum toxin injections can be tried.[131,141,148] Several drugs in combination might be needed (table 2).

## Asterixis

Asterixis is a negative myoclonus manifested by brief lapses of anti-gravity muscle contractions producing flapping movement of the outstretched limbs. Post-stroke asterixis is usually unilateral and affects the upper limb predominantly.[3,13,46,150] However, bilateral asterixis has also been described.[46,150] Asterixis typically develops during the acute phase of the stroke[3,46] and has been reported after lesions of the basal ganglia, thalamus, frontoparietal cortex, cerebellum, and brainstem.[3,5,46,149,150] It is typically self-limited and does not need pharmacotherapy.[5]

## Transient paroxysmal hyperkinesias

Some movement disorders associated with cerebrovascular disease occur as paroxysmal, transient, and intermittent involuntary movements that can be difficult to characterise phenomenologically. Transient limb shaking attacks have been described as flapping or trembling episodes lasting a few seconds or minutes and often accompanied by paresis of the involved limb.[179,180] Often wrongly attributed to seizures or paroxysmal dyskinesias,[170] these transient episodes are usually associated with severe carotid or vertebrobasilar stenosis, documented to be secondary to decreased cerebral blood flow, and represent forms of transient ischaemic attacks.[3,13,151–154] They might be precipitated by events that induce cerebral hypoperfusion such as standing up or coughing.[179] Electroencephalograms do not show any epileptiform activity during the events,[5] distinguishing these events further from epileptic seizures. Symptoms can disappear after endarterectomy.[3,13,151] One patient with moyamoya disease was reported to develop transient dyskinesia with frontoparietal cortical and subcortical hypoperfusion on SPECT and improved after superficial temporal artery-middle cerebral artery bypass.[45]

## Stereotypies, akathisia, and tics

Stereotypies are involuntary, coordinated, repetitive, patterned, and rhythmic seemingly purposeless movements.[1] Very few cases of stroke-induced stereotypies have been reported,[3,155,156] some being part of the acute stroke presentation.[3] These cases were usually attributed to parietal, lenticulostriatal, thalamic, midbrain, or left middle cerebral artery territory infarcts (table 2). Most patients had unilateral lesions with contralateral stereotypies. Some improved with clonazepam, but they might be refractory to benzodiazepines, amantadine, and anticholinergics.[156] Tetrabenazine, while the most effective drug in the treatment of tardive stereotypies, has not been evaluated in patients with post-stroke stereotypies.

Punding is a form of complex stereotypy and has been reported in a patient with brainstem stroke in the form of endlessly writing, copying, and organising recipes as well as purchasing and hoarding food. The patient improved with 150 mg per day of sertraline.[181]

Akathisia is defined as an intense inner urge to move and marked motor restlessness that is partially relieved by voluntary activity. Although usually drug-induced or as part of tardive dyskinesia, one patient with hemiakathisia 5 months after an acute stroke involving the contralateral posterior thalamus was reported.[3] His symptoms improved with clonazepam.

Tics consist of involuntary twitches (motor tics) or sounds (phonic tics). Post-stroke tics have rarely been reported after lesions to the striatum, globus pallidus, and frontal or parietal cortex.[158,159] Post-stroke tics can be treated with clonidine[160] or dopamine receptor antagonists such as risperidone or fluphenazine if needed.[44]

## Conclusions and future perspectives

Future studies should use the most advanced imaging and neurophysiological techniques to better characterise the anatomical and clinical correlation between the location of stroke-related lesions and the phenomenology of the movement observed. These techniques might also provide insights into the pathophysiology of stroke-related movement disorders. Since these disorders are relatively uncommon, except for vascular parkinsonism, compared with the general prevalence of cerebrovascular disease, multicentre studies will be needed to study the natural history and treatment of these hypokinetic and hyperkinetic movement disorders.

Although strokes are the most common cause of neurological morbidity, involvement of the basal ganglia or brainstem by a cerebrovascular lesion rarely leads to the development of a movement disorder. Furthermore, there is no strict anatomical or clinical correlation

### Search strategy and selection criteria

We reviewed available English language literature in PubMed between 1983 and 2012 using the search terms "stroke", "vascular", "hemorrhage", "infarction", "malformation", "fistula", and each of the different movement disorders, "parkinsonism", "tremor", "dystonia", "chorea", "ballism", "athetosis", "myoclonus", "akathisia", "tics", or "stereotypies". Articles were also identified through searches of the authors' own files.

between the location of the lesion or lesions and the phenomenology of the movement observed. Although post-stroke hyperkinetic movement disorders are relatively uncommon, vascular parkinsonism is probably the second most common form of parkinsonism and is a major cause of disability. Control of stroke risk factors is essential in reducing the incidence of movement disorders related to cerebrovascular disease. The quality of life of patients with post-stroke movement disorders can be meaningfully improved with symptomatic therapies tailored to their specific needs.

#### Contributors

RM contributed to review of the literature and the original draft. JJ contributed to review of the literature, editing, and critique.

#### Conflicts of interest

JJ has received research support Allergan, Allon Therapeutics, Biotie, Ceregene, Chelsea Therapeutics, Diana Helis Henry Medical Research Foundation, EMD Serono, Huntington's Disease Society of America, Huntington Study Group, Impax Pharmaceuticals, Ipsen, Lundbeck, Medtronic, Merz Pharmaceuticals, Michael J Fox Foundation for Parkinson Research, National Institutes of Health, National Parkinson Foundation, Neurogen, St Jude Medical, Teva Pharmaceutical Industries, University of Rochester, Parkinson Study Group, has been a consultant or advisory committee member for Allergan, Chelsea Therapeutics, EMD Serono, Lundbeck, Merz Pharmaceuticals, Michael J Fox Foundation for Parkinson Research, Neurocrine Biosciences, Teva Pharmaceutical Industries, and has participated in editorial boards for Elsevier, Medlink, Neurology, Neurology in Clinical Practice, Neurotoxin Institute, Scientiae, and UpToDate. RM declares that he has no conflicts of interest.

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