Ballistic or choreic dyskinesia, or both, on one side of the body (hemiballism-hemichorea) is a clinical entity caused mainly by vascular lesions in the subthalamic nucleus or other structures. There have been few clinical reports of cortical or subcortical white matter lesions associated with involuntary movements. We report four patients who had involuntary movements of relatively short duration and concomitant paresis of the same limb(s). We speculate that subcortical ischemia was a primary cause of these symptoms. We also discuss a possible mechanism of these "positive" motor phenomena occurring after white matter lesions.

**Case Reports**

**Patient 1**

An 82-year-old hypertensive woman suddenly experienced paresthesia of pins and needles in the left arm and the same limb started voluntarily to "hop and jump" an hour later. The dyskinesia consisted of sporadically appearing hovering and flinging movements of the entire left upper limb intermingled with rotation, flexion-extension or pronation-supination of the arm and hand. The face and legs were not involved. These movements were resistant to intravenous administration of glycerol, phenobarbital, diazepam and phenytoin, although sleep induced by the hypnotizing effect of some of these medications was effective in reducing the severity of the movements. The patient was otherwise intact neurologically except for reduced muscle tone in the left extremities. The dyskinesia continued unchanged for 22 hours. As a slowly evolving monoparesis of the left arm became apparent, it decreased in frequency and amplitude and finally disappeared 24 hours after onset. Weakness of the left upper extremity improved in three days without recurrence of the involuntary movements.

Electroencephalography (EEG) was normal without epileptic discharge. CT 20 hours after the onset of the abnormal movements was normal. MRI and angiography were not obtainable.

**Patient 2**

A 75-year-old hypertensive man noticed a sudden tingling numbness of the left half of the body, followed shortly after by flailing involuntary movements of the left extremities. On admission the same day, we observed the following dyskinesia superimposed on mild left hemiparesis:

Weakness of the left upper extremity improved in three days without recurrence of the involuntary movements.

**Abstract:** Four patients presented with hemiballism-hemichorea as a clinical manifestation of white matter ischemia. These patients illustrate "positive" motor phenomena rather than limb weakness as a consequence of cerebral ischemia. In each patient, the involuntary movements disappeared following worsening of paresis. Subcortical white matter infarction in three patients and hemodynamic hypo-perfusion in the cerebral hemisphere contralateral to dyskinetic movements were possible causes. Neuroradiologically, none had pathological changes in the vicinity of the subthalamic nucleus. We presume from these observations that ischemia of the subcortical white matter, without involvement of the basal ganglia or the subthalamic nucleus, may cause hemiballism-hemichorea.

**Résumé:** Hémiballisme-hémichoree induite par ischémie sous-corticale: à propos de quatre cas. Quatre patients se sont présentés avec hémiballisme-hémichoree comme manifestation clinique d’une ischémie de la substance blanche. Ces cas soulèvent une question intéressante mais peu explorée, à savoir l’apparition de phénomènes moteurs positifs plutôt que d’une faiblesse comme conséquence d’une ischémie cérébrale. Les mouvements involontaires sont disparus chez tous les patients suite à une aggravation de la pâsésie. Un infarctus de la substance blanche sous-corticale en était possiblement la cause chez trois patients, alors que chez l’autre le phénomène était possiblement dû à une hypo-perfusion hémodynamique de l’hémisphere cérébral contra-latéral. Au point de vue neuroradiologique, aucun ne manifestait de changements pathologiques dans le voisinage du noyau sous-thalamique. Ces observations nous portent à croire qu’une ischémie de la substance blanche sous-corticale contralatérale, sans atteinte du noyau lenticulaire, du noyau caudé, de l’avant-mur, du noyau amygdalien ou du noyau sous-thalamique, peut causer l’hémiballisme-hémichoree.

the whole left arm swung irregularly and sporadically with large excursions around the shoulder joint, coupled with non-synchronous swift rotations of the forearm and flexion-extension at the elbow and wrist joints. In addition, complex movements of the left leg appeared, consisting of irregular and non-repetitive abduction-adduction, flexion-extension and inversion-eversion at the hip, knee and ankle joints. Repetitive intramuscular administrations of phenobarbital and diazepam failed to control these movements. Two and a half hours after onset, the dyskinetic movements slowly subsided until they completely resolved as the paresis of the left extremities gradually worsened. The left hemiparesis resolved in 10 days without recurrence of abnormal movements.

EEG was normal. CT performed 6 hours after the onset revealed a reduced density at the right fronto-parietal subcortical white matter and centrum semiovale, which was more clearly depicted on the follow-up CT (Figure 2a) on day 8. On brain MRI on day 19, the corresponding region appeared high-signal on a T2-weighted image (T2WI) without involving the overlying cortex, basal ganglia or subthalamic nucleus (Figure 2b, c). Angiography revealed subtotal occlusion of both internal carotid arteries at the cervical bifurcations. Single photon emission computed tomography (SPECT) using 99mTc hexamethyl-propylene-amine oxime (HM-PAO) on day 11 showed hypoperfusion at the right hemisphere (Figure 2b, c). Tracer uptake appeared normal in the basal ganglia and subthalamic areas. The patient refused angiography.

**Patient 3**

An 86-year-old woman was hospitalized because of a sudden right-sided weakness. Subsequently, the following dyskinesia was observed: the entire right arm was sporadically and violently rotated in an invasive-evasive fashion at the shoulder joint with the elbow slightly flexed. These movements lasted for a few minutes, then were substituted by dyskinesia of the right leg: coarse and irregular extension-flexion at the hip and knee joints which lasted another few minutes. The involuntary movements of the right extremities continued for an hour and gradually subsided after aggravation of right hemiparesis.

EEG was normal except for a small amount of theta waves in the left centro-parietal region. A plain CT examined 3 hours after the onset revealed a fresh infarction confined to the white matter of the precentral and premotor cortices on the right side (arrows). (B) The centrum semiovale (arrow heads) appear intact.
Figure 2 — (Patient 2): (A) Plain CT shows an extensive low density area in the right centrum semiovale (arrow), (B) which appears high-signaled (arrow) on T2-weighted image (TR 3000 msec, TE 100 msec) on MRI (Toshiba MRT-50A, 0.5 tesla). (C) The subthalamic regions (arrow heads) appear intact on MRI.

Figure 3 — (Patient 3): Plain CT (A) and MRI (B) show a diffuse subcortical infarction in the left centrum semiovale (arrows), which may antedate the episode of acute stroke. The subthalamic regions (arrow heads in (C) appear normal.)

A cortical or subcortical lesion is an extremely rare cause of non-epileptic dyskinetic disorders and includes infarction at the superior frontal and precentral gyri, or nonspecific atrophy of the postcentral gyrus contralateral to dyskinesia.

In patient 1, although a CT examination alone may be inadequate to exclude the possibility of subthalamic involvement, we considered that a newly appearing subcortical white matter infarct of the right precentral gyrus was responsible both for involuntary movements and monoparesis of the left arm. In
In patients 2, 3 and 4, as in patient 1, transient ischemia or minute pathological changes in the subthalamic nucleus, which would escape MRI detection, cannot be excluded totally from the possible etiologies of hemiballism-hemichorea. However, the probability that such undetected lesions exist is low since they are unlikely to produce concomitant paresis.

Differential diagnoses include Huntington’s chorea, chorea acanthocytosis, senile chorea, or dyskinesias of metabolic and toxic etiologies. However, the acute clinical course and unilateral involvement in our patients indicated a vascular origin. Post-apoplectic partial seizure may be differentiated by lack of compatible EEG abnormalities.

Normal motor sequences are controlled by complex positive and negative feedback circuits (Figure 5). In essence, the precentral and prefrontal cortices send excitatory projections to the corpus striatum. For the promotion of ongoing movements, the striatum inhibits the medial globus pallidus (MGP) and substantia nigra reticulata (SNr), which, as a consequence, disinhibit the thalamus and cortex (positive feedback loop). For inhibition of undesirable movements, the striatum suppresses the lateral globus pallidus and inhibitory projections to the subthalamic nucleus, which exerts excitatory action on the MGP and SNr. These latter structures then inhibit the thalamus and cortex (negative feedback loop). An alternative excitatory pathway which projects from the precentral motor cortex directly to the subthalamic nucleus has been identified.

According to this theory, selective disruption of the negative feedback loop would result in “disinhibition” of the motor cortex and occurrence of involuntary movements. It is possible that hemiballism-hemichorea occurs when a lesion is situated anywhere in this loop, whether it is in the basal ganglia, subthalamic nucleus, or subcortical white matter. When more of the motor cortex or the pyramidal tract is involved, motor paresis may eliminate any dyskinesia.

Although subcortical white matter infarction is common, resultant hemiballism-hemichorea is only rarely encountered, probably because selective disruption of the negative loop may seldom occur, or because most lesions may cause paresis, precluding involuntary dyskinesia.
REFERENCES