Limb Shaking — A Carotid TIA

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SUMMARY Eight patients are described with an unusual form of carotid transient ischemic attack, limb shaking. The basic features included a brief, involuntary, coarse, irregular, wavering movement or tremble involving arm-hand alone, or arm-hand and leg together. In 2 patients limb shaking was the initial manifestation of carotid occlusive disease, and all but one patient had other typical carotid transient ischemic attacks.

Major atheromatous carotid occlusive disease was present in all patients on the side opposite the limb shaking. Four patients had bilateral carotid occlusive disease.

Cerebral ischemia from a carotid territory low-perfusion state may be the pathogenesis of these limb shaking movements, an idea supported by the apparent benefit of surgical revascularization in abolishing or reducing the limb shaking in 6 patients. There was no clinical or EEG evidence to document an epileptiform etiology.

Recognition of this uncommon form of carotid transient ischemic attack may be important in the early diagnosis and treatment of carotid occlusive disease.

IN THIS REPORT, eight patients are described with an uncommon type of carotid transient ischemic attack (TIA) characterized by involuntary limb movements or "shaking." The usual clinical manifestations of carotid occlusive disease, transient hemispheric attacks (THAs) and transient monocular blindness (TMB), are well known but limb shaking, recognized in only a few reports, may be mistaken for focal epilepsy and delay or confuse the early diagnosis of carotid occlusive disease.

Case Reports

Patient 1

An 88-year-old man had a three week history of intermittent episodes of right-sided weakness and speaking difficulty. The episodes, lasting only seconds, involved weakness of the right arm and hand, buckling of the right knee, and speaking difficulty. The usual patterns were right arm and leg, sometimes only the right arm, and rarely the right leg alone. Thick and hesitant speech was associated with some of these events. Involuntary trembling movements of the right arm and hand frequently accompanied the right arm weakness, and prompted a trial of phenytoin treatment. The phenytoin was discontinued, however, when he became toxic.

Upon admission evaluation, blood pressure was 140/90. A detailed neurologic examination was normal. Leg claudication and coronary artery disease were present. There was no history of transient monocular blindness. A CT scan and EEG were both normal.

On admission evaluation, blood pressure was 140/90. The general physical examination was negative except for loud bilateral carotid bruits. The neurologic examination was remarkable for an occasional hesitancy and stammering quality in his speech, and mild right lower face weakness. Strength in the right arm was normal.

During hospitalization four episodes of right arm and hand shaking were observed when the patient was either supine or sitting, all occurring during intravenous heparin anticoagulation. The movements were wavering, shaking excursions of the arm and hand which lasted 4-5 seconds. During one episode, there were 10 second wavering movements of the right hand, at which time he had difficulty holding a fork. He could talk during the shaking, although speech was more hesitant and several verbal paraphasic errors occurred. His right arm could be lifted on its own power to the horizontal during the shaking, but a lateral drift and wavering-shaking movements of a nonrhythmic nature were observed.

Cerebral angiography demonstrated severe stenosis of the left internal carotid artery. Technical difficulties prevented further angiographic studies. Repeat CT scan and EEG were both normal.

The patient underwent prompt carotid endarterectomy and no further TIAS or shaking occurred during one year of follow-up before death from an accident.

Patient 2

A 44-year-old, right-handed man was referred for evaluation of carotid disease. Over 4 months, he experienced many episodes in which his right leg and foot “gave out” due to weakness. He would stumble or fall to the right side because of this, but there had been no loss of consciousness or any other associated symptoms. Angiographic evaluation at his local hospital demonstrated a left internal carotid artery occlusion at the origin. The right carotid was widely patent, and the intracranial circulation was normal. A CT scan was normal.

He was treated with antiplatelet agents (acetylsalicylic acid and dipyridamole), but intermittent, brief episodes of right leg and foot weakness continued. There was no history of transient monocular blindness.

His past medical history was significant for hypertension, leg claudication, and coronary artery disease manifested by angina. He underwent a recent coronary artery bypass graft and had an uneventful recovery.

His general physical examination was negative. Blood pressure was 140/90. A detailed neurologic examination was normal.

During hospitalization for a sternal incision infec-
tion, he experienced shaking episodes of the right arm and leg. One evening, he reached up with his right hand from bed to check the IV bottle and the right hand and foot began shaking. He described coarse, waver-
ing-lateral movements, but no tonic-clonic compo-
nent. The shaking lasted 3–5 minutes, during which time his level of consciousness and awareness re-
mained normal. One hour later when he got up from bed to go to the bathroom, a few minutes of shaking movements affecting his right lower leg and foot oc-
curred, but only to a minor degree in the right hand. Finally, later in the evening upon sitting up in bed, shaking of arm and leg again occurred. His neurologic examination remained normal after these events, and no orthostatic hypotension was documented. An EEG was normal, even with attempts to precipitate the shaking movements by having him stand up quickly. A left extracranial/intracranial bypass graft was performed and at a three month follow-up intermittent right leg weakness continued but only one episode of right arm shaking had occurred.

Patient 3

A 64-year-old, right-handed man was referred for evaluation of episodic right hand movements. Five years before admission, a brief episode of left transient monocular blindness occurred, and angiography demon-
strated left internal carotid artery occlusion. He was asymptomatic for two years but then a prolonged, five-
hour episode of speech disturbance prompted warfarin anticoagulation. Two years later, warfarin was discon-
tinued because of intra-abdominal bleeding. Antiplate-
let agents were initiated after his right leg became transiently weak upon arising from a chair. Three months before evaluation, he noted the onset of involuntary jerking movements of the right distal arm, hand, and fingers described as a “flap.” Twenty-
five to thirty brief episodes, lasting seconds, had oc-
curred without any obvious precipitating conditions. A trial of phenytoin had no effect on the movements. In the two months before evaluation, 2 distinct half-hour episodes occurred in which speech became “slowed” and the right hand was clumsy and weak when he attempted to reach for change in his pocket. Another episode occurred in which only speech was slowed for minutes. He was hospitalized, but “flapping” move-
ments continued despite intravenous heparin anticoag-
ulation. He was transferred for further evaluation.

On admission examination, his blood pressure was 140/90. The general physical examination was normal except for bilateral subclavian artery distribution bruits, and a faint bruit along the left common carotid. The neurologic examination was normal.

CT scan and EEG were normal. Cerebral angiography demonstrated left internal carotid occlusion with filling of the left middle cerebral artery territory from the vertebrobasilar circulation via the posterior communicat-
ing artery. The right internal carotid was patent.

He continued to have brief episodes several times per month of right hand “flapping” occasionally asso-
ciated with speaking difficulty over the next year and a half while on antiplatelet medications. He died sud-
denly of a cardiac arrest, 1½ years later.

Patient 4

A 48-year-old woman was admitted for transient right-sided weakness and speaking difficulty. Intermit-
ten episodes of darkening vision in the left eye had occurred over the past year. During the three months prior to admission she had 3 episodes of right arm and hand numbness with speaking difficulty, and one epi-

dose of right leg numbness. An angiogram performed at another hospital demonstrated occlusion of the left internal carotid artery at its origin. She was treated with acetylsalicylic acid and sulfipyrazone, but fol-
lowing another brief episode of right leg weakness she was hospitalized.

On admission examination blood pressure was 160/110. The general physical and neurologic exami-
nations were normal. A 10 minute episode of right foot and leg jerking movements occurred in which her foot “moved around in circles.” An EEG subsequently showed left temporal slow activity, but no epileptiform features. A CT scan was normal. Right leg weakness associated with trembling movements lasting 3–4 minutes occurred one additional time. She was switched to warfarin anticoagulation, but intermittent episodes of right hand and leg movements continued and warfarin was discontinued. A trial of phenytoin did not affect the attacks of right-sided weakness and shaking.

She became aware of reading difficulty and re-eval-
uation demonstrated dyslexia and right homonymous hemianopia. Repeat angiography showed the left internal carotid occlusion with filling of the left middle cerebral artery territory through left ophthalmic and internal maxillary collateral channels. The right carotid artery was patent. Another EEG showed left tempo-
ral slow activity, but no epileptiform features. She underwent a left extracranial/intracranial bypass graft and no further episodes of weakness, speaking diffi-
culty, or shaking have occurred in over seven years.

Patient 5

A 50-year-old woman was evaluated for intermittent hand “twitching” and slurred speech. Eleven months previous several brief episodes of lightheadedness and right hand weakness occurred. On one occasion she experienced slurred speech for 20 minutes. In a sepa-
rate event vision in the left eye darkened momentarily. Cerebral angiography demonstrated severe stenosis of the left internal carotid artery just above its origin, and a tandem stenosis of the carotid siphon. There was subtotal occlusion of the right internal carotid artery with only a trickle of antegrade flow, but no significant intracranial contribution. Both middle cerebral artery territories were supplied via the posterior communicat-
ing artery from the vertebrobasilar circulation. She was treated with warfarin anticoagulation. Two months later she experienced transient lightheadedness and slurred speech in association with bilateral hand twitching. CT scan was normal. Warfarin anticoagula-
tion was changed to antplatelet treatment using acetyl salicylic acid and dipyridamole. Another episode of bilateral hand twitching prompted hospital evaluation.

Admission blood pressure was 170/85. The general examination revealed only a right carotid bifurcation bruit, and the neurologic examination was normal. Repeat angiography showed no change. Repeat CT scan and EEG were normal. Warfarin anticoagulation was restarted.

For one year she remained symptom free then two, 20 minute episodes of bilateral hand twitching occurred in association with difficulty “getting words out.” She remained alert during these episodes. On another occasion, the hand movements persisted despite her attempts to hold down the arm. The movements were not related to position. She underwent a left extracranial/intracranial bypass graft and the twitching movements referable to her right side no longer occurred. Later, a right extracranial/intracranial bypass graft was performed for recurrent left-sided twitching, and in a two month follow-up, no further movements occurred.

Patient 6

A 53-year-old woman was evaluated for transient right arm and leg weakness associated with speech difficulty. Seven weeks prior to admission, the right leg buckled and weakened for several minutes. Subsequently, several transient episodes of right arm weakness occurred with difficulty getting words out. On two occasions, she experienced brief bilateral visual obscurations, but there was no history of definite transient monocular blindness.

On admission to the hospital, blood pressure was 150/90. The general examination was notable for bilateral carotid bifurcation and ocular bruits. The neurologic examination was normal. Cerebral angiography demonstrated severe stenosis of the left supraclinoid internal carotid artery and occlusion of the extracranial portion of the right internal carotid artery. Both hemispheres received flow from the verteobasilar circulation via the posterior communicating artery. She was treated with intravenous heparin and later switched to warfarin anticoagulation. Four brief episodes of right hand shaking occurred while therapeutically anticoagulated. An EEG showed only intermittent left temporal slow activity, but no epileptiform patterns. A CT scan was normal.

Recurrent episodes of right arm and leg weakness continued on warfarin anticoagulation, and she was readmitted to hospital two months later for a left extracranial/intracranial bypass graft. On the first postoperative day, a transient episode of aphasia and right arm weakness occurred in association with a drop in her blood pressure. Repeat CT scan showed a left frontal, watershed area infarction. She was discharged on phenytoin, acetyl salicylic acid, and dipyridamole, but one month later she suddenly became aphasic with left conjugate gaze preference, right homonymous hemianopia, and right arm weakness. CT scan showed a new left middle cerebral artery territory infarction.

Repeat angiography was unchanged and she was discharged to a rehabilitation center on warfarin anticoagulation. In the ensuing 24 months, no further neurologic events have occurred.

Patient 7

A 61-year-old man was evaluated for episodic left arm and hand trembling. Four months prior to admission, left arm trembling first occurred in association with bilateral forearm and leg weakness. Several weeks later, he experienced transient left arm weakness, slurred speech with numbness and drooling from the left side of his mouth. There was no history of transient monocular blindness.

On admission evaluation, his blood pressure was 155/110. The general physical examination was negative. Neurologic examination showed mild left lower face weakness and pyramidal dysfunction of the left hand. During hospitalization, several 3–5 minute episodes of left arm shaking were observed. Attempts at controlling the shaking by holding onto the forearm did not stop the movements. During limb shaking he remained alert with normal sensation. Also, two separate episodes of aphasia and right face weakness occurred in association with vigorous antihypertensive therapy. A CT scan demonstrated a small infarction in the right centrum semiovale. Angiography disclosed bilateral extracranial internal carotid artery occlusions. The middle cerebral artery territories were supplied via the posterior communicating arteries from the verteobasilar circulation, and from retrograde ophthalmic flow.

He was treated with antihypertensive medications, acetyl salicylic acid and dipyridamole. During 14 months of follow-up, no further TIAs or limb shaking have occurred.

Patient 8

A 68-year-old man was evaluated for transient left arm and leg “shaking.” Six months prior to admission, he experienced two brief episodes of left arm and leg “shaking” which resulted in residual paresthesias of the left side of his body. Two months prior to admission, transient left arm and leg “shaking” recurred. The movements were described as non-rhythmic, flailing type lateral excursions of the arm and hand followed by similar movements of the left leg. No weakness or impairment in consciousness were present, and he was noted to converse normally during the episode. There was no history of transient monocular blindness.

On hospital admission, his blood pressure was 190/90. The general physical examination was negative. The neurologic examination was remarkable for left visual neglect, levitation of the outstretched left arm, and clumsiness of fine finger movements in the left hand. Pin and light touch were diminished on the entire left side of the body, and joint position sense was decreased on the left fingers and toes.

CT scan showed a deep, right parietal infarction. Cerebral angiography demonstrated bilateral extracranial internal carotid artery occlusions. Hemisphere filling occurred by several collateral routes including
retrograde ophthalmic flow and leptomeningeal collaterals from the posterior cerebral artery. An EEG showed right parietal-temporal slow activity, but no epileptiform patterns. He was discharged on phenytoin, acetyl salicylic acid, and dipyridamole.

Two months later, he underwent a right extracranial-intracranial bypass graft because of recurrent light-headedness associated with visual blurring. In a 26 month follow-up, no further episodes of weakness or shaking have occurred.

**Summary of Patients**

The present patients included 5 men and 3 women, ranging in age from 44–88 years with an average age of 60 years. All patients had multiple episodes of transient, involuntary limb movements described as "shaking," "trembling," "twitching," "flap," or "wavering." The movements were usually brief, lasting seconds to minutes, occasionally longer. Limb position was not an obvious precipitating factor since movements were described under circumstances of supine, sitting, or even standing postures with the limb at rest or in an anti-gravity position. Only Patient 2 experienced positionally related limb movements when he arose from supine to sitting or standing in the absence of documented orthostatic hypotension, a condition described by Caplan and Sergay. The limb movements were not accounted for on the basis of a sensory loss or primary cerebellar deficit since all patients had normal cerebellar functions and all but one (Case 8) had normal sensation.

The movements affected only arm and hand in 4 patients, arm and leg together in 3 patients, (including 1 patient with separate leg shaking), and 1 patient had simultaneous bilateral hand movements. The features of the movements were coarse and irregular, often with lateral excursions maximally located in the distal portion of the affected extremity. Only occasionally was the extremity weak at the time the movements were occurring. On several occasions, associated symptoms such as speaking difficulties and weakness occurred at the same time as the movement disorder.

The movements lacked the features of focal epilepsy. Definite rhythmic, tonic-clonic jerking, head-eye turning, or impaired consciousness were never present. No patients had a history of convulsive phenomena. Epileptiform activity was never seen in patient EEGs between attacks, but unfortunately, no EEG was performed during an attack. A trial of anticonvulsant medication in 5 patients did not alter the movements.

All patients had major atheromatous carotid occlusive disease on the side opposite the movement disorder. Six patients had extracranial carotid disease (5 occlusion, 1 stenosis), one patient had extracranial carotid stenosis with a tandem lesion producing occlusion at the top of the carotid, and one patient had only intracranial carotid siphon stenosis. Bilateral carotid occlusive disease was present in four of seven patients undergoing bilateral carotid angiography, all four with occlusions.

Seven patients had other typical carotid territory TIAs, either hemispherical or ocular, in addition to the shaking spells (table 1). Two patients had their initial TIA as a shaking attack, while the others had either a hemispherical attack or transient monocular blindness as the first clinical manifestation of their carotid disease.

A permanent neurologic deficit (stroke) was not a necessary feature accounting for the shaking movements. Only 3 patients had a mild deficit at the time they presented with shaking spells, and 2 of the 3 had small hemisphere infarcts on CT scan. The remaining 5 patients had only TIAs and normal CT scans at initial presentation. Two of these patients, however, developed stroke later in their course.

Surgical revascularization (endarterectomy or extracranial-intracranial bypass grafting) in 6 patients appeared to have a beneficial effect in reducing or abolishing the limb movements (table 1). **Discussion**

These patients corroborate earlier observations that transient limb movements may be a manifestation of carotid occlusive disease. In his observations on carotid TIAs, Fisher noted that "a frank, focal convulsive seizure is a great rarity, but the patient may speak of the affected parts as trembling, shaking, twisting, drawing up, or moving irregularly." Case 4 in Russell and Page's report had involuntary jerking movements of the left arm and leg lasting two minutes in association with presumptive carotid occlusive disease. Yanagihara and Klass provided the most extensive report on this condition, describing six patients with involuntary limb movements in association with either severe stenosis or occlusion of the carotid opposite the side of movements. The authors suggested that cerebral ischemia was the probable pathogenetic mechanism. Their patients responded favorably (decrease or absence of shaking movements) to surgical revascularization in the form of either carotid endarterectomy or extracranial/intracranial bypass grafting.

Our patients provide additional documentation of this unusual condition and its strong relationship to carotid occlusive disease. We agree with Yanagihara and Klass that transient, focal cerebral ischemia is the likely mechanism producing the movements. An ischemic mechanism based on a carotid low-perfusion state is strongly suggested by 1) the angiographically documented severe extracranial and/or intracranial carotid occlusive disease, 2) the similarity of recurrent movements which may reflect repeated bouts of ischemia affecting the same brain region in the distal field of the obstructed carotid artery, and finally, 3) surgical revascularization (endarterectomy or extracranial/intracranial bypass grafting) in 6 of the 8 patients led to a cessation or reduction in shaking spells, suggesting correction of a low-perfusion condition. However, the small number of patients involved and the short follow-up caution against an endorsement of surgical treatment as unequivocally effective.

A seizure mechanism, either primary or related to transient cerebral ischemia, as an explanation for the shaking movements cannot be entirely dismissed. Ex-
Experimental evidence suggests that seizure activity can be recorded from cortical neurons subjected to ischemic effects. Some investigators have even found a high incidence of true epilepsy associated with carotid occlusive disease, while in other studies epilepsy has been conspicuously absent as a manifestation of either carotid TIAs or acute carotid stroke. In the present patients, non-epileptiform EEGs between shaking spells, and the negative response to anticonvulsants don't eliminate the possibility of seizures. EEG recordings during a shaking episode, unfortunately not available in these patients, would have gone a long way toward settling this issue, and should be sought in the evaluation of future patients.

Limb shaking is an unusual and not well recognized form of carotid territory TIA. Recognition of these shaking movements and their relationship to major carotid occlusive disease may lead to more prompt diagnosis and appropriate treatment.

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References


<table>
<thead>
<tr>
<th>Patient no., sex, age</th>
<th>Limb shaking</th>
<th>Transient hemispheric attacks (THAs)</th>
<th>Transient monocular blindness (TMB)</th>
<th>Carotid occlusive disease</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 M, 88</td>
<td>r arm-hand</td>
<td>aphasia &amp; weakness r arm, hand &amp; leg</td>
<td>no</td>
<td>IICA extracranial stenosis</td>
<td>/ carotid endarterectomy</td>
<td>limb shaking &amp; THAs abolished at 1 yr. followup</td>
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<tr>
<td>2 M,44</td>
<td>r arm, hand &amp; foot</td>
<td>weakness r leg</td>
<td>no</td>
<td>IICA extracranial occlusion</td>
<td>/ EC/IC bypass graft</td>
<td>THAs continued but only 1 episode of limb shaking in 3 mos. followup</td>
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<td>3 M, 64</td>
<td>r arm-hand</td>
<td>aphasia &amp; weakness r hand</td>
<td>1 eye</td>
<td>IICA extracranial occlusion</td>
<td>Antiplatelet medications</td>
<td>all TIAs stopped but limb shaking continued for ½ yrs. of followup</td>
</tr>
<tr>
<td>4 F, 48</td>
<td>r leg-foot</td>
<td>aphasia &amp; weakness r leg-hand, r leg weakness</td>
<td>1 eye</td>
<td>IICA extracranial occlusion</td>
<td>/ EC/IC bypass graft</td>
<td>all TIAs and limb shaking abolished in 7 yr. followup</td>
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<tr>
<td>5 F, 50</td>
<td>simulta-neous r &amp; l hands</td>
<td>aphasia &amp; weakness r hand</td>
<td>1 eye</td>
<td>/ IICA extracranial stenosis &amp; tandem siphon stenosis, r ICA occlusion</td>
<td></td>
<td>all TIAs &amp; limb shaking abolished in 7 mos. followup</td>
</tr>
<tr>
<td>6 F, 53</td>
<td>r hand</td>
<td>Aphasia &amp; weakness r arm, hand, leg; aphasia &amp; weakness r arm</td>
<td>no</td>
<td>/ supraclanoid ICA ICA stenosis, r ICA extracranial occlusion</td>
<td>/ EC/IC bypass graft</td>
<td>postop. L MCA territory stroke then no further neurological events in 32 mo. followup</td>
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<td>l arm-hand</td>
<td>slurred speech, weakness r arm &amp; numbness l face; Aphasia &amp; weakness r face</td>
<td>no</td>
<td>r &amp; l ICA extracranial occlusions</td>
<td>antiplatelet medications</td>
<td>THAs and shaking abolished in 14 mo. followup</td>
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<td>8 M, 68</td>
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<td>no</td>
<td>no</td>
<td>r &amp; l ICA extracranial occlusions</td>
<td>r EC/IC bypass graft</td>
<td>THAs &amp; shaking abolished in 26 mos. followup</td>
</tr>
</tbody>
</table>

ICA = Internal Carotid Artery
MCA = Middle Cerebral Artery
EC/IC = extracranial/intracranial

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