Gait Disturbances in Patients With Pontine Medial Tegmental Lesions

Clinical Characteristics and Gait Analysis

Hiroshi Mitoma, MD; Ryoichi Hayashi, MD; Nobuo Yanagisawa, MD; Hiroshi Tsukagoshi, MD

Objective: To determine the clinical characteristics of gait disorders in patients with pontine medial tegmental lesions.

Design: We compared features of gait disorders between patients with infarcts in the medial tegmentum and those with stroke in other areas of the pons (pathological control subjects) by measuring electromyographic results of lower limb muscles and several biomechanical parameters.

Patients: Two patients with infarcts in the rostral medial tegmentum and 4 control subjects. Two of the control patients had lesions in the pontine base, while the lesions in the other 2 were in the pontine tegmentum and base (combined lesions).

Results: Patients with rostral medial tegmental lesions and controls with pontine base lesions showed unstable walking characterized by irregular angular displacements and foot pressures. However, they differed by the following 3 features. (1) Rostral medial tegmental lesions elicited truncal ataxia without limb ataxia. In comparison, pontine base lesions elicited limb ataxia without truncal ataxia and caused hemiparesis. (2) Instability was more severe and persistent in patients with the former lesions than in those with the latter lesions. Slowness of walking speed and prolongation of the double-support period were clearly observed in the former group. (3) Electromyographic changes characteristic of cerebellar ataxia were clearly evident in patients with rostral medial tegmental lesions. The electromyographic amplitudes of the gastrocnemius and tibialis anterior muscles were almost constant throughout the gait cycle, resulting in the disappearance of the inherent periodic pattern of each muscle.

Conclusion: Medial tegmental lesions in the rostral pons cause prolonged and severe unstable walking that resembles spinocerebellar ataxic pattern, and impairment of the spinocerebellar loop might be the pathomechanism underlying such a gait disturbance.

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Pontine hemorrhage or infarct causes gait disorders characterized by unstable broad-based walking. Possible structures responsible for the pontine stroke-induced gait disturbance include the pyramidal tract, the cerebro-ponto-cerebellar pathway, the medial lemniscus, and the vestibular system. Involvement of a combination of these structures has been considered to determine the clinical picture of such a disturbance.

To elucidate the pathomechanisms of gait disturbances in patients with pontine medial tegmental lesions, we describe 2 patients with infarcts in this region and 4 patients with stroke lesions in other regions of the pons (pathological control subjects). Two of the pathological controls had lesions in the pontine base, and the other 2 had lesions extending in the pontine tegmentum and base. Clinical features were compared in patients with lesions in the medial tegmentum and in the 4 pathological control subjects. Gait analysis was also performed and included recording of electromyographic (EMG) activities of lower limb muscles, angular displacements of the hip and leg joints, and floor reaction forces during free walking.

General Aspects of Gait

Healthy Subjects

An example of a free-walking pattern of a healthy subject is shown in Figure 3. Foot contacts and angular displacements occurred periodically, and displacement of center of body pressure (COP) was straight from one foot to another (Figure 3, A-C). Foot pressures in the vertical direction showed a 2-peak pattern, corresponding to step in and kick off (Figure 3,
SUBJECTS AND METHODS

PATIENTS AND CONTROLS

The clinical features of all patients are summarized in Table 1. Patients 1 and 2 suddenly experienced drunk-enlike walking. On examination a few days later, both patients had great difficulty in sitting and standing due to unsteadiness. Patient 1 did not exhibit hemiparesis, limb ataxia, or sensory impairment. Patient 2 showed a slight weakness of the right upper and lower limbs but no limb ataxia or sensory impairment. Eye movements were normal in both patients. During the following month, the condition gradually improved. However, walking instability persisted, necessitating the use of canes while walking.

On presentation, both patients showed swaying in all directions during upright standing and walking. Tandem gait was impossible. The muscle strength and tonus were normal. Deep tendon reflexes were increased on both sides. The Babinski sign was not observed in patient 1 but was observed in patient 2. Both patients showed no dysmetria, decomposition, or adiadochokinesis in the limbs. Touch, pain, and temperature sensations were intact in both patients. Vibratory and positional sensations were also preserved, and the Romberg sign was not observed. Disturbances of cranial nerves were not present in both patients.

Patients 3 and 4 showed mild gait disturbances compared with patients with medial tegmental lesions. Although they were unable to walk unsupported due to weakness and instability of the contralateral limbs in the short-term, both patients were able to walk unaided during the present examination. They showed slight hemiparesis. Patients 5 and 6 showed severe and long-term unstable gait similar to those with medial tegmental lesions and used canes during walking. Both patients showed slight hemiparesis and hyperreflexia in contralateral limbs. Muscle tonus was normal in both patients. Moderate dysmetria, decomposition, and adiadochokinesis were noted on the contralateral side (patient 5) or on both sides (patient 6). Both patients exhibited severe truncal ataxia. Patient 5 showed impairments of touch, vibratory, and positional sensations on the contralateral side. The Romberg sign was observed in patient 5. Patient 6 showed no sensory disturbances. Patient 5 had ipsilateral facial palsy, horizontal nystagmus to the ipsilateral side, and slurred speech.

The interval between the stroke and the present examination was similar in patients 1 and 2 (average, 32.5 months) and control patients (mean±SD, 37.8±23.1 months). In gait analysis, to minimize speed-dependent changes, we also studied healthy elderly subjects (6 men and 6 women; mean±SD age, 75.4±5.8 years) whose

<table>
<thead>
<tr>
<th>Patients</th>
<th>Age, y/Sex</th>
<th>Diagnosis</th>
<th>Lesion Side</th>
<th>Time Since the Attack, mo</th>
<th>Gait Disorder Severity</th>
<th>Hemiparesis (Brunnstrom Recovery Stage)</th>
<th>DTR (rt/lt)†</th>
<th>Babinski Sign (rt/lt)</th>
<th>Limb Ataxia</th>
<th>Truncal Ataxia</th>
<th>Touch Sense</th>
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<td>48/M</td>
<td>Hemorrhage</td>
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<td>24</td>
<td>Mild</td>
<td>+ rt (5)</td>
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<td>+ rt&gt;lt</td>
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<td>24</td>
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<td>+ rt (5)</td>
<td>↑↑</td>
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<td>+ rt&gt;lt</td>
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<td>+ rt (5)</td>
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<td>lt</td>
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* The tonus was normal in all patients. DTR indicates deep tendon reflexes; rt, right side; lt, left side; –, not observed; +, observed; ↑, augmented; N, normal; and ↓, decreased.
† At the biceps brachii and triceps brachii muscles, the knee and the ankle.

B). Electromyographic recordings showed periodic phasic muscle activities (Figure 3, D).

Patients With Medial Tegmental Lesions

Figure 4. A, demonstrates the gait pattern of patient 1. Displacement of COP was irregular, and a small loop was observed when COP shifted from one foot to another. Hyperreflexia and the Babinski sign were present on the contralateral side (patient 4) or on both sides (patient 3). Moderate dysmetria, decomposition, and adiadochokinesis were present in both limbs in both patients, whereas truncal ataxia was not evident. In patient 3, sensations of touch, pain, and temperature were decreased on the contralateral side, with the exception of the face. However, the vibratory sensation and proprioception were intact, and the Romberg sign was not observed. Patient 4 showed no abnormalities of sensations without impairment of touch sensation in the left side of the face. Both patients had no nystagmus but showed a slightly slurred speech.

Patients 5 and 6 showed severe and long-term unstable gait similar to those with medial tegmental lesions and used canes during walking. Both patients showed slight hemiparesis and hyperreflexia in contralateral limbs. Muscle tonus was normal in both patients. Moderate dysmetria, decomposition, and adiadochokinesis were noted on the contralateral side (patient 5) or on both sides (patient 6). Both patients exhibited severe truncal ataxia. Patient 5 showed impairments of touch, vibratory, and positional sensations on the contralateral side. The Romberg sign was observed in patient 5. Patient 6 showed no sensory disturbances. Patient 5 had ipsilateral facial palsy, horizontal nystagmus to the ipsilateral side, and slurred speech.

The stride was shortened, and the double-support period was prolonged. The floor reaction forces in the vertical axis formed multipeak patterns on both sides. The range of angular displacement was small in the left and right hips, knees, and ankles, and the pattern was different in each cycle. Phasic EMG activity was noted in both proximal muscles throughout the gait cycle; however, the periodic EMG pattern associated with the gait...
preferable walking speed was slow (mean ± SD, 63.1 ± 16.3 cm/s). Informed consent was obtained from all subjects.

Magnetic resonance imaging was performed using a 0.5-T superconducting system (repetition time, 3000 milliseconds; echo time, 100 milliseconds). Slice planes were perpendicular to the long axis of the brainstem, with a section thickness of 5 mm and an interval length of 6 mm. Topographic localization of the vascular lesion was determined using transverse anatomical templates. Infarcts in patients 1 and 2 were located in the medial tegmentum of the rostral pons (Figure 1, A, and Figure 2, A and B). Herein, we defined these lesions as medial tegmental lesions according to the definition of Silverstein,2(pp13-53) Patients 3 and 4 had lesions located in the pontine base (Figure 1, B, and Figure 2, C), These lesions were defined as paramedian or lateral pontine lesions.2(pp13-53) In patient 5, the area of infarct extended from the paramedian area to the medial tegmentum, while in patient 6, the ischemic area was located in the lateral area and the medial tegmentum (Figure 1, C, and Figure 2, D). Furthermore, a magnetic resonance imaging scan showed associated infarcts in 3 patients: a focal lesion in the thalamus (in patients 2 and 3) and diffuse cerebrobral white matter abnormalities (in patient 6).

**GAIT ANALYSIS**

Gait analysis was performed in all patients but patient 6. Subjects were asked to walk at their own ordinary speed without support along a 6-m walkway, in which 2 force plates (2 m long and 80 cm wide) were serially arranged (model 1812A; Anima, Tokyo, Japan). The pressure exerted on the force plate by each foot was measured in 3 dimensions. Changes in angles around the hip, knee, and ankle joints in the sagittal plane were measured using electrogoniometers. Angular displacement values were expressed relative to the leg position in a relaxed standing posture.

Electromyograms of the adductor magnus, gluteus medius, biceps femoris, vastus lateralis, gastrocnemius or soleus (GC), and tibialis anterior (TA) muscles were recorded bilaterally using surface silver–silver chloride surface electrodes placed 3 cm apart. Electromyographic signals were passed through a band-pass filter of 20 to 500 Hz. A multichannel telemeter box (model 511X; NEC-Sanei, Tokyo, Japan) was attached to the lower back and used to transmit data of angular changes and EMG signals.

Data were stored on a microcomputer (model PC-9801; NEC-Sanei) after analog-to-digital conversion. The sampling rate for floor reaction forces and angular displacements was 100 Hz, while that of EMGs was 1000 Hz.

A gait cycle was divided into 4 phases: the first double-support period (phase 1), the single-support period (phase 2), the second double-support period (phase 3), and the swing period (phase 4). Electromyographic signals were rectified and integrated at a time constant of 50 milliseconds. The integrated EMG was summed through each phase and then divided by the duration of that phase (the time-averaged EMG was calculated in microvolts per second).

Data were expressed as the mean ± SD. In comparing the data of patients with those of healthy controls, the value recorded in the patient was considered pathological when it exceeded the mean ± 2 SD of controls, according to the method by Knutsson and Richards.6 Using an unpaired t test, we examined statistical differences of spatiotemporal gait parameters and angular displacements between the right and left legs in each subject.

<table>
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<tr>
<th>Pain and Temperature Senses</th>
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<th>Cranial Nerve Disturbance</th>
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**Pathological Control Subjects**

Figure 4, B, shows the gait pattern of patient 3. Displacement of COP was irregular, the stride was short, and the single-support period of the right leg was shorter than that of the left leg. The amplitude of the floor reaction forces in the vertical axis decreased on the right side, and the floor reaction forces in the vertical axis showed a multipeak pattern on both sides. The range of angular displacements cycle was not clear in both distal muscles. Patient 2 showed a walking pattern similar to that of patient 1.

**Figure 1.** Lesion sites in each type, superimposed in 4 schematic planes of the pons: 1, section through the decussation of the trochlear nerve; 2, section through the entrance of the trigeminal nerve; 3, section through the principal sensory nucleus and motor nucleus of the trigeminal nerve; and 4, section through the vestibular and the facial nerve nuclei. A, Medial tegmental lesions. B, Paramedian or lateral pontine lesions. C, Combined lesions. Inset numbers represent patients 1 through 6; L, left side; and R, right side.
decreased in the right knee and ankle joints, and their patterns were irregular. The EMG amplitudes of the right leg muscles were low, compared with those of the left leg. Patient 4 exhibited similar EMG and kinematic patterns.

The walking pattern of patient 5 showed features of walking of patients with medial tegmental lesions and of those with paramedian or lateral lesions (data not shown). Displacement of COP was irregular, with a small loop. Stride was shortened, and the single-support period was shorter in the right leg than in the left leg. The floor reaction forces in the vertical axis showed multipeaks on both sides, while the floor reaction forces in the vertical axis of the right leg decreased in amplitude. Patterns of angular excursions were different on each gait cycle on both sides, and angular displacements of the knee and ankle joints decreased in amplitude, especially on the right side. The periodic EMG pattern associated with the gait cycle was not clear in the distal muscles of both sides, while the activity of right leg muscles was low compared with that of the left leg.

**KINEMATIC FACTORS**

Patients with medial tegmental lesions and those with combined lesions showed a widened stance, reduced walking speed, and a prolonged double-support period beyond the mean±2 SD values of controls (Table 2). In contrast, patients with lesions in the paramedian or lateral pons showed small changes in spatiotemporal parameters, being within the mean±2 SD, except for a prolonged double-support period in patient 3 and a widened stance in patient 4. In patients with paramedian or lateral pons lesions and combined lesions, the single-support periods of legs on the contralateral side of lesions were significantly shorter than those of ipsilateral legs (P<.01).

Angular displacements decreased in both sides in patients 1 and 2; the decreases in knee and ankle joints were
beyond the mean−2 SD of the control (Table 3). No significant differences between the right and left legs were observed (P>.05), except for the knee joint of patient 2. In patients with lesions in the paramedian or lateral pons, angular displacements on the side contralateral to the lesion were significantly smaller than those on the ipsilateral side (P<.01). Furthermore, the decrease in the contralateral knee joint of patient 3 and those of the contralateral knee and ankle joints in patient 4 exceeded the mean−2 SD of the controls. In patient 5 with combined lesions, angular displacements of the knee and ankle joints were small, beyond the mean−2 SD of the control on both sides, and such a decrease was significantly smaller on the contralateral side than on the ipsilateral side (P<.01).

**MUSCLE ACTIVITIES**

**Figure 5.** A, shows time-averaged EMGs during free walking in patients 1 and 2. The periodic activity pattern associated with the gait cycle was observed in each proximal muscle but was not seen in each distal muscle. The amplitude of the time-averaged EMG of the GC...
muscle was higher than the mean+2 SD of the control during phases 1 and 4. No difference was observed between right and left legs.

In patients 3 and 4 (Figure 5, B), the amplitudes of the time-averaged EMGs of the GC muscle during phase 2 and of the TA muscle during phases 1, 2, and 3 on the contralateral side of the lesion were lower than the mean−2 SD of the control. In contrast, the amplitude of the time-averaged EMG was augmented on the ipsilateral side. The EMG amplitudes of the adductor magnus muscle during phases 1 and 4; the gluteus medius muscle during phases 2 and 4; the vastus lateralis muscle during phases 2, 3, and 4; the biceps femoris muscle during phase 3; and the GC muscle during phases 1 and 2 were higher than the corresponding mean+2 SD of the control.

In patient 5, the amplitude of the time-averaged EMG of each muscle was low on the contralateral side of the lesion (data not shown). The amplitude of the gluteus medius, vastus lateralis, and TA muscles during phase 1 was less than the mean−2 SD of the control. On the ipsilateral side, the

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**Figure 4.** Sample record of gait in patient 1 with a medial tegmental lesion (A) and in patient 3 with a lesion in the paramedian or lateral pons (B). a indicates displacement of the center of pressure; b, floor reaction forces in the vertical axis (Fz) and anterior-posterior axis (Fy); c, angular displacements of the hip, knee, and ankle; and d, electromyograms of the hip and lower leg muscles. In b and c, the solid line shows the trace of floor reaction forces and angular displacements of the right foot; dotted lines trace those of the left foot. L indicates left leg, R, right leg.
time-averaged EMGs of the proximal muscles showed a normal pattern throughout the gait cycle. In ipsilateral distal muscles, there was no clear periodic pattern due to augmented activity of the GC muscle during phases 1, 3, and 4, and of the TA muscle during phases 2, 3, and 4.

**COMMENT**

**CHARACTERISTICS OF PONTINE STROKE-INDUCED GAIT DISORDERS**

Patients with rostral medial tegmental lesions and pontine base lesions showed a common feature of unstable walking with irregular angular displacements and foot pressures. Palliyath et al. defined features of cerebellar ataxic gait as irregularity of stepping and lack of coordination of limb movements. Thus, both types of gait disturbances exhibited a cerebellar ataxic pattern. However, our studies showed the following 3 differences in ataxic symptoms or gait disturbances induced by lesions of the rostral medial tegmentum and pontine base.

First, patients with lesions in the medial tegmentum of the rostral pons showed unstable walking accompanied by truncal ataxia, whereas those with lesions in the pontine base showed unstable gait with limb ataxia. Second, instability was more severe and persistent in the...
former group than in the latter group of patients. Slowness of walking speed and prolongation of the double-support period, which are compensatory reactions for instability in walking and reflect the severity of the gait disorder, were clearly observed in the former group. Third, EMG features characteristic of cerebellar ataxic gait were clearly observed in the walking of patients with rostral medial tegmental lesions. In our previous study, features of cerebellar ataxic gait included augmented activity of the GC and TA muscles during those phases associated with lack of muscle recruitment during normal walking. Based on these changes, the periodic activity pattern associated with the gait cycle was not clear in the GC and TA muscles. These features were observed in patients with lesions in the cerebellar vermis and in those with lesions in the hemisphere, although they were more marked in the former patients than in the latter (H.M., R.H., N.Y., and H.T., unpublished data, 1993). The present results showed that in the walking by patients with medial tegmental lesions, GC and TA muscle activities were almost similar in each phase, resulting in the disappearance of the periodic activity pattern of each muscle associated with the gait cycle. Considered together, the gait abnormalities of patients with medial tegmental lesions in the rostral pons closely resembled the unstable walking caused by impairment of the cerebellar vermis, whereas those of patients with pontine base lesions resembled the unstable walking caused by deficits of the cerebellar hemisphere.

In addition to differences in ataxia, another distinguishable feature was that truncal ataxia was the only symptom in patients with medial tegmental lesions in the pons. Lesions in the rostral medial tegmentum elicited EMG and kinematic abnormalities on both sides. How-
ever, no hemiparesis, sensory impairment, or vestibular symptoms were noted in these patients. In contrast, in walking by patients with pontine base lesions, limb ataxia on the contralateral side was accompanied with hemiparesis.

POSSIBLE STRUCTURES RESPONSIBLE FOR GAIT DISORDERS INDUCED BY ROSTRAL MEDIAL TEGMENTAL LESIONS

Differences in impairment of cerebellar afferent or efferent fibers would explain the difference in ataxia between rostral medial tegmental lesion– and pontine base lesion–induced gait disorders.

The cerebellar vermis receives proprioceptive information from the periphery through the spinocerebellar tracts, and in turn sends output signals carried by vestibulospinal and reticulospinal neurons in the tegmentum field via cerebellar nuclei.10 This cerebellospinal loop is known to play a crucial role in the regulation of stepping and stabilization.11,12 Hayashi et al13 reported that a 3-Hz postural oscillation, a symptom of cerebellar vermis, occurred in patients with strokes in the dorsal pons and indicated that such impairment could cause symptoms related to the spinocerebellar loop. The present pattern in rostral medial tegmental lesions included severe and persistent truncal ataxia without limb ataxia, and walking closely resembled ataxic gait caused by impairment of the vermis. Stroke-related lesions closely overlapped the area where axons of the fastigial and interpositus nuclei neurons are distributed.14-16 Thus, it appears that lesions in the medial tegmentum of the rostral pons damage the output fibers from the cerebellar nuclei on the vestibulospinal and reticulospinal tracts, so as to endow the spinocerebellar ataxic nature in this dorsal lesion–induced gait disorder.

The cerebellar hemisphere receives input signals from the cerebral cortex via the pontine nucleus, and sends output signals to the cerebral cortex through the dentato-thalamo-cortical pathways.10 This loop is recruited for adaptive control of voluntary movements.10 Our patients with lesions in the pontine base showed ataxic hemiparesis, and the features of the ataxia resembled cerebellar hemisphere ataxia. Thus, the cerebrospinal and cerebro-ponto-cerebellar pathways would have been damaged at the pontine base in these patients,3 which was confirmed by magnetic resonance imaging observations. Such segregated impairments of cerebellar afferent or efferent fibers in the pons appear to adequately explain the features of pontine stroke–induced gait disturbances.

Figure 5. A, Comparison of time-averaged electromyograms (EMGs) recorded during walking of patients 1 and 2 with medial tegmental lesions with those of healthy subjects. Open bars indicate the time-averaged EMGs of healthy subjects (n=24). B, Comparison of time-averaged EMGs recorded during walking of patients 3 and 4 with lesions in the paramedian or lateral pons with those of healthy subjects. Open bars indicate the time-averaged EMGs of healthy subjects (n=24).
CONCLUSIONS

We reported herein that lesions in the medial tegmentum of the rostral pons led to unstable gait, which was not associated with hemiparesis, limb ataxia, sensory impairment, or vestibular-related symptoms. The severe instability persisted for more than 2 years after the stroke. The EMG and kinematic properties of this type of gait abnormality were of spinocerebellar nature. Impairment of the spinocerebellar loop might be one of the underlying pathomechanisms of marked instability.

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Reprints: Hiroshi Mitoma, MD, Mitoma Neurological Clinic, 1-2-10 Minami-Ikebukuro, Toshima-ku, Tokyo 171-0022, Japan.

REFERENCES


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Roger N. Rosenberg, MD
Editor

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