Gait initiation by patients with lower-half parkinsonism

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Summary

Patients with multiple deep cerebral infarcts and white matter degeneration commonly exhibit a hesitant, shuffling gait, with preserved arm swing. This pattern of walking is called lower-half or lower-body parkinsonism. Gait initiation and turning consist of one or more short, hesitant steps in which the feet shuffle across the floor. This abnormality of gait initiation was studied with quantitative motion analysis in five patients, ages 74-87 years. Five men and five women with normal mobility and comparable ages exhibited three key events of gait initiation: (i) activation of tibialis anterior and inactivation of triceps surae produced bilateral ankle dorsiflexion and a sagittal moment of force that propelled the body anteriorly; (ii) abduction of the swing hip occurred simultaneously with event (i); and (iii) abrupt 3–10° flexion of the support hip and knee occurred nearly simultaneously with events (i) and (ii) and produced a transient reduction in vertical force beneath the support foot. Events (ii) and (iii) produced a coronal moment of force about the ankles that propelled the body toward the support foot. Thus, in normal gait initiation, a smooth sequence of postural shifts propels the body anterolaterally toward the support limb, culminating in a forward step. The patients, by comparison, exhibited errant deviations in their postural shifts of gait initiation, and one or more aborted steps frequently preceded the first complete step. Nevertheless, all patients employed the usual three key events in their initial attempt at stepping, consistent with a normal motor strategy of gait initiation. These results and previous clinical observations suggest that the principal locomotor deficit is an impaired generation of postural shifts that mediate changes from one steady-state posture or movement to another.

Keywords: locomotion; stroke; frontal lobes; basal ganglia; apraxia

Abbreviations: COP = resultant centre of pressure of both feet; COM = total body centre of mass

Introduction

Patients with multiple deep cerebral infarcts commonly walk with short hesitant steps, moderately increased step width, stooped posture, reduced foot-floor clearance and poor balance. Arm swing during walking is often normal or increased in proportion to a reduced stride. This type of shuffling gait has been called lower-half or lower-body parkinsonism because arm swing is preserved (Thompson and Marsden, 1987; FitzGerald and Jankovic, 1989). Most patients with lower-half parkinsonism exhibit large confluent areas of rarefied deep cerebral white matter in combination with multiple small subcortical and occasional cortical infarcts (Thompson and Marsden, 1987; FitzGerald and Jankovic, 1989). The frontal lobes and basal ganglia are usually affected more than other brain regions, and damage in these regions is believed to underlie the gait disturbance (Ishii et al., 1986; Thompson and Marsden, 1987; FitzGerald and Jankovic, 1989). This combination of vascular pathology is often referred to as Binswanger disease or subcortical arteriosclerotic encephalopathy (Tomlinson, 1992).

Many patients with lower-half parkinsonism move their lower extremities with relative ease while seated or recumbent, but their feet appear to stick to the floor upon standing and attempting to walk. Gait is initiated with increased latency and with one or more short, hesitant steps in which the feet shuffle across the floor. Denny-Brown (1958) aptly called this pattern of gait initiation the slipping clutch syndrome. Similar hesitant shuffling occurs when turning and negotiating chairs or obstacles. The characteristics of gait typically improve as walking proceeds (Denny-Brown, 1958; Petrovici, 1968; Thompson and Marsden, 1987; FitzGerald and Jankovic, 1989).

Many authors have viewed the hesitant shuffling gait of
patients with bilateral frontal lobe damage as a form of kinetic apraxia because, in their opinion, the locomotor impairment could not be attributed to existing sensory loss or muscle weakness (Denny-Brown, 1958; Meyer and Barron, 1960; Petrovici, 1968). Denny-Brown (1958) believed that the fundamental pathophysiological disturbance was a perseveration of stance, associated with an instinctive foot grasp. Meyer and Barron (1960) and Petrovici (1968) concurred with Denny-Brown and observed that the primary difficulty was in the initiation of movement. However, the concept of gait apraxia has been challenged because most patients have significant impairment of motor function and cognition. This is particularly true for those patients with lower-half parkinsonism caused by cerebrovascular disease (Thompson and Marsden, 1987) but is also true for most patients with other forms of frontal lobe and basal ganglia damage. For example, six of the seven patients described by Meyer and Barron (1960) had impaired cognition, and all exhibited four or more of the following motor signs: rigidity, action tremor, hyperreflexia, extensor plantar response, unstable stance, hypokinesia and bradykinesia. The aetiology in three of their seven patients was arteriosclerotic cerebrovascular disease, and one of seven had general paresis. The patients in many other studies exhibited similar motor signs, and the aetiology in most was cerebrovascular disease (Denny-Brown, 1958; Petrovici, 1968; Thompson and Marsden, 1987; Yanagisawa et al., 1991). Virtually all patients in prior studies exhibited other clinical signs of frontal lobe damage, such as paratonia, motor perseveration, increased response latency, laboured speech and emotional lability (Denny-Brown, 1958; Meyer and Barron, 1960; Petrovici, 1968; Thompson and Marsden, 1987; Yanagisawa et al., 1991).

The principle aim of our study was to begin characterizing the abnormalities of gait initiation in lower-half parkinsonism in terms of basic motor physiology. Petrovici’s (1968) EMG analysis of gait initiation by patients with a variety of frontal lobe diseases revealed persistent tonic postural activity in the muscles of the lower extremities and impaired generation of the normal EMG bursts needed for standing and stepping. Petrovici concluded that his patients had ‘a disorder of the postural set’ because they had greatest difficulty making transitions from a static posture (e.g. stance) to locomotor movement (e.g. walking). We now extend Petrovici’s observations by reporting the kinematics, kinetics and EMG patterns of gait initiation in five patients with lower-half parkinsonism caused by cerebrovascular disease.

Methods

This project and its consent form were approved by the Springfield Committee for Research Involving Human Subjects, in accord with federal guidelines of the United States.

Patients

Four men and one woman, aged 74–87 years (mean±SD 79±5 years), with vascular dementia and lower-half parkinsonism participated after giving informed written consent that was co-signed by their next-of-kin. All patients were referred to an outpatient clinic for memory loss and gait impairment. The four men were followed with examinations at least twice yearly, at intervals of ≤6 months for 3–8 years, and all exhibited less than a two-point change in their Mini-Mental Status scores (Folstein et al., 1975) over periods exceeding 1 year. Patient E.S. was seen only twice in a period of 1 month. The clinical characteristics of these patients are summarized in Table 1. Patients E.S. and C.C. had adult-onset diabetes mellitus, and patient C.C. had undergone coronary artery bypass surgery. Patients C.C., J.R. and J.F. had a history of depression. All patients had hypertension. The results of neuroimaging studies are summarized in Table 2. All but one patient (J.R.) had extensive rarefaction of subcortical cerebral white matter, but this patient had the greatest number of cortical and subcortical infarcts.

All patients were able to stand without assistance but were unstable when turning, bending over and responding to a sternal nudge. None could tandem walk, but all could walk at least 10 m without assistance. Each patient exhibited varying degrees of stooped posture, paratonia and short

### Table 1 Clinical characteristics of Binswanger patients and elderly controls

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (m)</th>
<th>HIS*</th>
<th>MMSE†</th>
<th>Duration of gait disorder (years)</th>
<th>Tinetti balance score (max. = 16)</th>
<th>Tinetti gait score (max. = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ES</td>
<td>F</td>
<td>74</td>
<td>72.7</td>
<td>1.47</td>
<td>14</td>
<td>8</td>
<td>4</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>W.T.</td>
<td>M</td>
<td>81</td>
<td>58.2</td>
<td>1.55</td>
<td>13</td>
<td>17</td>
<td>5</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>C.C.</td>
<td>M</td>
<td>79</td>
<td>67.0</td>
<td>1.65</td>
<td>16</td>
<td>20</td>
<td>4</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>J.R.</td>
<td>M</td>
<td>75</td>
<td>59.5</td>
<td>1.55</td>
<td>13</td>
<td>17</td>
<td>10</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>J.F.</td>
<td>M</td>
<td>87</td>
<td>79.5</td>
<td>1.73</td>
<td>9</td>
<td>18</td>
<td>5</td>
<td>11</td>
<td>7</td>
</tr>
</tbody>
</table>

Controls†

| n = 5 | F   | 81 (6) | 65.5 (12.9) | 1.57 (0.04) | 2 (0) | 28 (0) | 0 | 13 (1) | 12 (0) |
|       | M   | 82 (6) | 70.8 (10.7) | 1.65 (0.07) | 3 (1) | 27 (1) | 0 | 14 (1) | 12 (0) |

*Hachinski Ischaemic Score: maximum score = 18; score ≥6 is strong evidence for symptomatic cerebrovascular disease (Hachinski et al., 1975; Wade et al., 1987); †Mini-Mental State Examination: maximum normal score = 30 (Folstein et al., 1975); ‡mean (SD).
Table 2 Summary of neuroimaging studies*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Subcortical white matter abnormality¹</th>
<th>Locations of small infarcts</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Putamen</td>
</tr>
<tr>
<td>E.S.</td>
<td>Bilateral frontal and occipitoparietal</td>
<td>Bilateral</td>
</tr>
<tr>
<td>W.T.</td>
<td>Bilateral frontal and occipitoparietal</td>
<td>Bilateral</td>
</tr>
<tr>
<td>C.C.</td>
<td>Bilateral frontal and occipitoparietal</td>
<td>Bilateral</td>
</tr>
<tr>
<td>J.R.</td>
<td></td>
<td>Right</td>
</tr>
<tr>
<td>J.F.</td>
<td>Bilateral frontal and occipitoparietal, greatest posteriorly</td>
<td></td>
</tr>
</tbody>
</table>

*Patients E.S. and W.T. had CT scans, and patients C.C., J.R. and J.F. had MR scans; ¹Large patchy areas of increased T₂-MRI signal or CT lucency extending from the ventricular wall to the subcortical U fibres.

shuffling hesitant steps, particularly when turning and initiating gait. Rising from a chair frequently required the use of their hands, and their immediate standing balance was poor. Arm swing while walking was increased in proportion to stride, typical of lower-half parkinsonism (Thompson and Marsden, 1987). Patient C.C. shuffled the right lower extremity more than the left, but all other patients walked with symmetrical steps and arm swing. All patients exhibited varying degrees of urinary urgency, and J.R. and J.F. were occasionally incontinent of urine. All but J.F. had mildly laboured speech. Levodopa-carbidopa was tried unsuccessfully in J.R., C.C. and W.T. All patients gave informed consent to participate in the study.

Controls

Five men and seven women were recruited from our community through advertisement for healthy ambulatory people, and each gave informed written consent. All were examined by the same neurologist (R.J.E.). One woman was excluded because she was severely flatfooted, and another was excluded because of mild Alzheimer-type dementia. The clinical characteristics of the remaining five men and five women are summarized in Tables 1 and 3. Their ages ranged from 75 to 92 years, and no control subject had disabling musculoskeletal or neurological disease.

Gait analysis

Gait initiation is defined, in this report, as the sequence of events up to and including the time when the swing (stepping) foot leaves the floor. The onset of gait initiation was identified by a characteristic burst of EMG in the tibialis anterior. This burst could be delineated more accurately than the decrease in tonic gastrocnemius activity that usually preceded the tibialis burst (Elble et al., 1994). Ten trials of gait initiation were recorded from each normal volunteer and from three patients. Patients C.C. and W.T. performed only five trials because of frailty and fatigue. At least one practice trial was conducted. All subjects stood quietly on floor-mounted force plates and were instructed to begin walking briskly in response to a green light, just as they would at a street corner. Patient E.S. was the first person studied. In her experiment, the computer was programmed to begin sampling data
simultaneously with the green light. Unfortunately, she started prematurely in all 10 trials, and only one complete trial of data was recorded. Computer sampling began 0.5 s before the green light in all other experiments.

Each person wore tightly fitting spandex shorts and shirt. Reflective topographic markers were placed on the sacrum, at the level of L5–S1 and bilaterally on the ‘toe’ (between the second and third metatarsal heads), heel (posterior to the calcaneus, 2 cm above the ground), ankle (lateral malleolus of the fibula), knee (lateral femoral epicondyle), hip (greater trochanter), pelvis (anterior–superior iliac spine), shoulder (acromion process), thigh and shank. Motion of these markers was recorded with five 50 Hz computerized infrared stroboscopic cameras (Oxford Metrics Ltd., Oxford, UK). These cameras were capable of recording translational movements of 1.8 mm and angular rotations of 0.25°. Motion of body segments and rotation of joints were computed from the marker data (Vaughan et al., 1992). The location of the total body centre of mass (COM) was computed using anthropometric methods (Winter, 1990).

Two strain-gauge force plates (Advanced Mechanical Technology, Inc., Newton, Mass., USA) recorded the foot–floor reaction forces and moments of force for each foot in the lateral, anteroposterior and vertical directions. These data were used to compute the centre of pressure for each foot (accuracy ±0.5 mm), the resultant centre of pressure of both feet (COP) and the ankle moments of force (Vaughan et al., 1992). Computer sampling of lower extremity EMGs, force plates and the five cameras were activated 0.5 s before the green light, and sampling continued for 5 s.

The EMGs of the tibialis anterior, medial gastrocnemius, rectus femoris, biceps femoris and gluteus medius were recorded bilaterally with 0.8-cm diameter skin electrodes, positioned 2.5 cm apart, longitudinally over the muscle belly. The EMGs and force-plate signals were digitized at 500 samples per second.

**Performance-oriented assessments**

Balance and gait of each patient and control were quantified by a physiotherapist and by a neurologist (R.J.E.) using the Tinetti Assessment Scale (Tinetti et al., 1986). Ability to rise from a chair, sit in a chair, stand with eyes open, maintain stable stance in response to a sternal nudge and turn 360° were rated on an ordinal scale of 0 (abnormal), 1 or 2 (normal). Sitting balance and standing with eyes closed were scored 0 (abnormal) or 1 (normal). The maximum normal balance score was 16. Similarly, gait initiation, step length, step height, step symmetry, step continuity and step width were rated on an ordinal scale of 0 or 1, and walking path and truncal sway were rated 0, 1 or 2. The maximum normal gait score was 12.

**Quantitative measurement of muscle strength**

A hydraulically driven, computer-controlled device (KinCom® model 125E-plus, Chattecx Corporation, Chattanooga, Tenn., USA) with an accuracy of ±2 N was used to measure isometric muscle strength of right knee extension from a position of 60° flexion and of right ankle dorsiflexion and plantarflexion from 10° plantarflexion. Two trials were conducted after one practice trial. Subjects were asked to develop and sustain a maximum contraction for 3 s. The best of two trials was selected for analysis. Only three patients (C.C., J.R. and J.F.) were studied in this manner.

**Statistical analysis**

Only five patients were studied, and limited data were obtained from patient E.S. Furthermore, the patients differed greatly in their degree of locomotor impairment, and they did not perform equal numbers of trials. Consequently, we tested the hypothesis that the sample mean of each patient variable was drawn from the population of control values. The control mean and standard deviation of each variable were estimated separately for the normal women and men using the pooled data from all 48 trials by the women and all 50 trials by the men. The 95% confidence limits of the control mean (x) for each variable was computed as x±(SD±L±√n), where SD was the control group’s standard deviation, n was the number of trials for a particular patient and t was the Student’s t value (P = 0.05, d.f. = n−1). The patient mean for each variable was compared with the control mean and its confidence limits. Only the data from the four male patients were suitable for statistical analysis, and we compared their means with those of our male controls. The control values for the women are reported, but were not used in statistical comparisons.

**Results**

The normal volunteers had no difficulty with the experimental protocol. The ten trials were performed correctly by all volunteers, although two women started one trial prematurely. Thus, 98 control trials were suitable for analysis. The means and standard deviations of the principal kinematic and kinetic variables are summarized in Table 4.

All patients had poor concentration and significant dementia, as measured with the Mini Mental Status exam (Table 1), and they were easily distracted by the laboratory hardware and personnel. Therefore, the patients were reminded repeatedly before each trial to begin walking when the green light appeared. All patients had substantial clinical impairment of gait and balance, as measured with the Tinetti Assessment Scale (Table 1, Tinetti gait and balance scores). Two patients (C.C. and W.T.) were sufficiently frail and unstable that only five trials of gait initiation were attempted. Patient E.S. was too insecure to perform the task without the support of her husband’s arm, and her impaired concentration and dementia, which included simultanagnosia, were so severe that she repeatedly began walking before the green light. Consequently, only one complete trial and one nearly
Table 4 Summary of motion analysis data

<table>
<thead>
<tr>
<th></th>
<th>Normal volunteers</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Women</td>
<td>Men</td>
</tr>
<tr>
<td>Response time (s)</td>
<td>0.21 (0.11)</td>
<td>0.22 (0.08)</td>
</tr>
<tr>
<td>Initial step magnitude (m)</td>
<td>0.36 (0.07)</td>
<td>0.48 (0.09)</td>
</tr>
<tr>
<td>Time to start of initial step (s)</td>
<td>0.50 (0.17)</td>
<td>0.41 (0.08)</td>
</tr>
<tr>
<td>Initial lateral COP excursion (s)</td>
<td>0.35 (0.11)</td>
<td>0.29 (0.05)</td>
</tr>
<tr>
<td>Initial posterior COP excursion (s)</td>
<td>0.38 (0.12)</td>
<td>0.29 (0.05)</td>
</tr>
<tr>
<td>Initial lateral COP excursion (m)</td>
<td>0.037 (0.013)</td>
<td>0.052 (0.014)</td>
</tr>
<tr>
<td>Initial posterior COP excursion (m)</td>
<td>0.031 (0.015)</td>
<td>0.042 (0.016)</td>
</tr>
<tr>
<td>Right knee angle during quiet stance (deg)</td>
<td>9.4 (4.8)</td>
<td>7.7 (3.7)</td>
</tr>
<tr>
<td>Right hip angle during quiet stance (deg)</td>
<td>8.4 (5.3)</td>
<td>4.8 (6.7)</td>
</tr>
<tr>
<td>Support knee rotation (deg)</td>
<td>4.8 (3.6)</td>
<td>6.0 (2.6)</td>
</tr>
<tr>
<td>Support hip rotation (deg)</td>
<td>3.2 (2.3)</td>
<td>3.6 (2.3)</td>
</tr>
<tr>
<td>Drop in vertical force beneath support foot (N)</td>
<td>118.9 (47.9)</td>
<td>143.7 (52.5)</td>
</tr>
<tr>
<td>Rise in lateral shear beneath swing foot (N)</td>
<td>13.0 (5.2)</td>
<td>20.4 (6.0)</td>
</tr>
<tr>
<td>Peak coronal moment of force (Nm)</td>
<td>27.8 (11.9)</td>
<td>37.1 (12.3)</td>
</tr>
<tr>
<td>Peak sagittal moment of force (Nm)</td>
<td>25.4 (10.1)</td>
<td>34.0 (18.0)</td>
</tr>
</tbody>
</table>

*P < 0.05; mean (SD) computed from 10 trials for Patients J.R. and J.F., five trials for patient W.T. and four trials for C.C.; five women, 10 trials each for three and nine trials each for two; five men, 10 trials each; initial attempt at stepping was aborted; measured at the time of peak lateral excursion of the COP.

complete trial of gait initiation were captured by the computer, and these data were not analysed statistically.

All normal volunteers exhibited the stereotypic pattern of gait initiation described previously for healthy younger volunteers (Elble et al., 1994). The centres of pressure and gravity were always 2-10 cm anterior to the ankles during quiet stance. Gait initiation began with a postorolateral movement of the COP toward the swing foot. The COP then moved laterally toward the support foot as the swing foot began leaving the ground. This movement of the COP was associated with coronal and sagittal moments of force that propelled the total body COM toward the support foot and into forward motion (Fig. 1). This sequence of postural shifts was produced by predictable EMG patterns of muscle activity in all of the elderly controls (Fig. 2). During quiet stance, the gastrocnemius was tonically active and tibialis anterior was silent. This combination of muscle activities produced tonic ankle plantarflexion, which counterbalanced the slight forward lean of normal stance. Gait initiation began with decreased activity in gastrocnemius, followed by an abrupt activation of tibialis anterior and vastus lateralis. This combination of muscle activities caused ankle dorsiflexion, posterior movement of the COP and a forward pendular movement of the body about the ankles. The lateral movement of the COP toward the swing foot was produced by activation of the swing hip abductors and by 3-10° flexions of the support knee and hip. Swing hip abduction produced increased (negative) lateral shear between the foot and floor, and the support knee and hip flexions produced a transient reduction in vertical foot-floor reaction force (Fig. 3).

All patients intermittently exhibited a hesitant shuffling pattern of gait initiation (slipping clutch syndrome). This abnormality was most predictable after turning 180°, and these turns were executed with multiple shuffling short steps. However, all patients exhibited variable performance of these tasks, particularly the least disabled patients J.R. and J.F. In all 10 trials, J.R. and J.F. successfully initiated gait in the allotted time of 4.5 s (Table 4), and a complete step was achieved with the first postorolateral excursion of the COP. These initial steps were much shorter than those of controls (Table 4), but the principal events of gait initiation, seen in controls, were evident in all trials: (i) activation of tibialis anterior and inactivation of triceps surae, in association with
Fig. 2 EMGs from the control trial of gait initiation shown in Fig. 1. EMGs were recorded from the support (left) and swing (right) lower extremities. During quiet stance, the tibialis anterior (TA) was quiet and the gastrocnemius (GA) was tonically active. Variable tonic activity was present in the vastus lateralis (VL) and medial hamstrings (MH), depending upon the degree of knee flexion during quiet stance. Activity in GA suddenly decreased at the onset of gait initiation, and this was followed shortly by an abrupt activation of TA and vastus lateralis (vertical broken lines).

bilateral ankle dorsiflexion, produced a sagittal moment of force that propelled the body anteriorly; (ii) abduction of the swing hip occurred simultaneously with event (i) and produced a coronal moment of force about the ankles that propelled the body toward the support limb; (iii) abrupt flexion of the support hip and knee occurred nearly simultaneously with events (i) and (ii), and produced a transient reduction in vertical force beneath the support foot, thereby contributing to the coronal moment of force toward the support limb. The time from onset of gait initiation (i.e. activation of tibialis anterior) to the beginning of stepping movement was significantly prolonged for J.F. and J.R. (Table 3, time to start of initial step).

Patients C.C. and W.T. did not complete a step with the first posterolateral excursion of the COP. Instead, a very short complete step was achieved after one or more aborted attempts at stepping, in which the intended swing foot completely failed to leave the ground (slipping clutch syndrome). Nevertheless, the initial aborted step in each trial of C.C. and W.T. contained the three principal events of gait initiation. The two analysable trials of Patient E.S. also contained these events. Patients E.S., J.R. and J.F. exhibited gait initiation with initial aborted steps much less frequently than patients C.C. and W.T., but their initial successful steps were always very short. The steps of all patients lengthened and became less hesitant as walking proceeded.

A representative example of start hesitation with aborted steps is shown in Fig. 4 (patient W.T). The initial trajectory of the COP was posterolateral, toward the intended right swing foot. The necessary postural shifts for gait initiation occurred within 1.6 s of the onset of recording (1.1 s after the signal to go), but the step was aborted before significant movement of the right foot. The COP then fluctuated erratically as gait initiation proceeded with a shift of the
COP back toward the right foot (Fig. 4; 2.0 s). By 3.0 s, the COP had shifted back and forth between the right and left feet, producing two incomplete steps with little forward movement of the feet and COM.

The initial aborted step in Fig. 4 contained the key events of normal gait initiation. It began with activation of tibialis anterior (Fig. 5), which produced bilateral ankle dorsiflexion and posterior movement of the COP. Abduction of the swing hip occurred nearly simultaneously with activation of tibialis anterior (Fig. 6). Hip abduction produced an increased (more negative) lateral shear beneath the swing foot, and flexion of the support hip and knee produced a transient drop in the vertical force beneath the support foot (Fig. 6). These joint flexions and swing hip abduction produced the lateral movement of the COP toward the swing foot, which underlay the coronal moment of force that propelled the COM toward the support foot.

The lower extremity EMGs of our patients differed from controls in several respects. Gait initiation always began with bilateral activation of tibialis anterior, but the amplitudes of...
Fig. 5 The lower extremity EMG data are shown for the trial of gait initiation in Fig. 4 (Patient W.T.). Gait initiation began at 1.13 s (vertical broken lines). TA = tibialis anterior, GA = gastrocnemius, VL = vastus lateralis and MH = medial hamstrings.

activation exhibited considerable variability and right-left asymmetry (Fig. 5). Furthermore, the tonic activity in gastrocnemius during quiet stance tended to persist during tibialis activation. The vastus lateralis and medial hamstrings were always tonically active during quiet stance, consistent with the greater hip and knee flexion (i.e. flexed posture) of our patients (Table 4, right hip and knee angles during quiet stance). This tonic activity tended to obscure the phasic EMG bursts of gait initiation (Fig. 5) and precluded precise determinations of EMG latencies except for tibialis anterior.

Visual inspection of the COP trajectories revealed obvious differences between the patients and controls. The COP trajectories of our controls always flowed smoothly as they coursed posterolaterally toward the swing foot and then laterally toward the support foot. Control trajectories varied from trial to trial only in their overall amplitudes and in the degree to which the COP moved anteriorly during its lateral excursion from swing foot to support foot. By contrast, even the least disabled patients (J.R. and J.F.) produced COP trajectories with errant deviations from trial to trial (Fig. 7).

The response times for patients W.T. and C.C. were greatly prolonged, and these were the most disabled patients for which response times could be measured (Table 4). The patients’ mean response times correlated strongly with their total Tinetti scores (gait plus balance), but the sample size was too small for statistical significance ($r = -0.813$; $n = 4$; $F = 3.91$, $P = 0.187$). Patient J.R.’s mean response time was within normal limits, and J.F.’s mean response time was nearly normal (Table 4).

The means (SD) of the principal kinematic and kinetic variables of gait initiation are shown in Table 4 for the four patients who initiated gait without assistance. The time intervals from tibialis anterior activation to maximum initial lateral and posterior excursions of the COP toward the swing foot were normal or very mildly prolonged for all patients. Thus, the initial peak sagittal and coronal moments of force about the ankles occurred within roughly normal limits of time, once gait initiation had begun. However, all but the least impaired patient (J.F.) produced an initial sagittal moment of force and posterior COP movement that were...
**Gait initiation**

Fig. 6 The vertical foot-floor reaction forces, lateral foot-floor shear forces, support limb angles and anteroposterior (AP) and COP coordinates are shown for the initial aborted step of gait initiation in Fig. 4 (Patient W.T.). Activation of tibialis anterior occurred at 1.13 s (vertical broken lines). The three key events of gait initiation are evident even though a complete step was not achieved (compare with Fig. 3).

Fig. 7 Four COP trajectories from patient J.R. are shown to illustrate the initial posterolateral movement of the COP toward the swing foot. Gait initiation began at the arrows. Note the errant COP excursions and inter-trial variability (compare with Fig. 1).

The normal elderly volunteers in this study exhibited three key events in gait initiation: (i) activation of tibialis anterior and inactivation of triceps surae produced bilateral ankle dorsiflexion; (ii) abduction of the swing hip, which occurred substantially below normal limits. By contrast, the initial mean coronal moment of force and lateral COP movement toward the swing foot were below normal limits but were more nearly normal than the sagittal moment and posterior COP excursion.

All patients exhibited variable degrees of frailty and mild generalized weakness. Patient J.R. had mild spastic weakness of the left upper extremity and patient C.C. was slightly more weak and hypertonic in the right upper and lower extremities. The results of quantitative muscle testing are summarized in Table 5. The patients produced weaker knee extensions, ankle plantarflexions and ankle dorsiflexions than the male controls. Quantitative measurement of ankle plantarflexion was less reliable than the other measurements because the apparatus was sensitive to forces developed at the knee and hip and because the apparatus was awkward for this particular task.

**Table 5** Quantitative measures of muscle strength in three male patients*

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<tr>
<th></th>
<th>Knee extension</th>
<th>Ankle dorsiflexion</th>
<th>Ankle plantarflexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women (n = 5)</td>
<td>370 (54)*</td>
<td>97 (16)</td>
<td>288 (109)</td>
</tr>
<tr>
<td>Men (n = 5)</td>
<td>522 (65)*</td>
<td>182 (54)</td>
<td>370 (103)</td>
</tr>
<tr>
<td>Patient C.C.</td>
<td>333</td>
<td>130</td>
<td>103</td>
</tr>
<tr>
<td>Patient J.R.</td>
<td>273</td>
<td>62</td>
<td>76</td>
</tr>
<tr>
<td>Patient J.F.</td>
<td>398</td>
<td>120</td>
<td>287</td>
</tr>
</tbody>
</table>

*Peak muscle contraction in Newtons; †mean (SD).
simultaneously with event (i); (iii) abrupt 3–10° flexion of the support hip and knee, which occurred nearly simultaneously with events (i) and (ii). Event (i) produced a sagittal moment of force (torque) that propelled the body into forward motion. Events (ii) and (iii) propelled the body laterally, toward the support limb, so that the swing foot could leave the ground. Thus, purposeful postural shifts of gait initiation propelled the body from stable stance into forward motion and culminated in a forward step. These results are identical to those found previously in a younger population of normal volunteers (Elble et al., 1994).

All patients, but no controls, exhibited erratic errant excursions in their COP during gait initiation. The COP moves when ground reaction forces beneath the feet change during passive and active changes in posture and movement. Our patients were not subjected to exogenous perturbations of posture, so their erratic COP excursions reflected an impaired production of the self-initiated postural shifts in gait initiation.

The lower extremity EMGs contained abnormally sustained activity in gastrocnemius, vastus lateralis and medial hamstrings. This observation is similar to that of Petrovici (1968) and of Yanagisawa et al. (1991). The EMG activity in these muscles frequently contained irregular bursts, but we did not find the action tremor observed by Yanagisawa et al. (1991). Our 4.5-s recordings were much shorter than those of Yanagisawa et al. (1991), so perhaps longer recordings would have revealed tremor in one or more patients. Clinically, symptomatic tremor was not present in our patients or in those reported in previous studies (Thompson and Marsden, 1987; FitzGerald and Jankovic, 1989).

The initial postural shifts by our patients were qualitatively similar to those of normal people but were far more variable in amplitude and direction. All but one very impaired patient produced initial coronal moments of force that were reduced, but were still comparable to those of controls. However, all but the least impaired patient produced greatly reduced sagittal moments of force, indicating a reluctance or inability to propel the body into forward motion. The postural shifts of gait initiation are potentially destabilizing, particularly in the sagittal plane. Consequently, compensatory or precautionary changes in motor execution possibly underlay the reduced sagittal moments of force in our patients. Similarly, the short shuffling steps of our patients may have been, to some extent, a compensatory adjustment to postural instability. Our patients were weaker than the male controls, so reduced muscle strength could have contributed too.

The postural shifts of gait initiation by our patients began in <1 s after the signal to go. Therefore, simple hesitation was not the principal abnormality. The transition from stance to walking was delayed most by errant postural excursions that were followed frequently by one or more abortive attempts at stepping (slipping clutch syndrome). These abnormal postural shifts were ill-suited for normal stepping. Thus, impaired postural control appeared to contribute significantly to the prolongation of gait initiation in our patients.

The contributions of postural dyscontrol and impaired movement cannot be distinguished completely in our patients because postural adjustments are an integral part of gait initiation and most other aspects of locomotion (Massion, 1992). Our patients did not exhibit pure postural instability, and patients with spinal, sensory and cerebellar dys-equilibrium do not exhibit slipping clutch syndrome (Nutt et al., 1993). Furthermore, our patients, like those in previous studies (Petrovici 1968; Thompson and Marsden, 1987; Yanagisawa et al., 1991), had far less difficulty moving their lower extremities while recumbent or seated, situations in which the demands of postural control are less. Patients like ours exhibit greatest difficulty when starting, turning, sitting and rising from a chair, and the characteristics of gait frequently improve as walking proceeds. Therefore, the principal locomotor deficit appears to be an impaired generation of postural shifts that mediate changes from one steady-state posture or movement to another.

Denny-Brown (1958), Meyer and Barron (1960) and Petrovici (1968) reasoned that the usual strategy or motor plan of gait initiation was probably preserved in patients with gait apraxia. In support of this hypothesis, our patients exhibited the three principal events of normal gait initiation. Further evidence of a preserved motor plan is the peculiar variability in performance by our patients and those of previous studies (Yanagisawa et al., 1991). The occasional nearly normal performance of such patients suggests that the underlying deficit is not as hopelessly untreatable as the term apraxia suggests.

Our patients had radiological evidence of frontal lobe and basal ganglia damage, and their neurological signs were consistent with impairment of these structures. The frontal lobes, in conjunction with the basal ganglia and cerebellum, play a critical role in motor planning and execution (Brooks, 1995; Tanji, 1994). Studies in laboratory animals indicate that the frontal lobes participate in the planning and execution of gait initiation (Mori and Takakusaki, 1988), and clinicopathological studies have repeatedly implicated lesions of the mesial frontal lobes and basal ganglia in patients with a hesitant, short-stepped, shuffling gait (Yakovlev, 1954; Denny-Brown, 1958; Meyer and Barron, 1960; Thompson and Marsden, 1987; Nutt et al., 1993). The motor, supplementary motor and premotor areas of the frontal cortex project to the basal ganglia, and these cortical areas and the basal ganglia also connect with bulbospinal locomotor pathways and with brainstem nuclei that project to the cerebellum (Mori and Takakusaki, 1988; Tanji, 1994). Consequently, it is easy to envision how frontal lobe pathology could produce a spectrum of locomotor impairment ranging from relatively pure postural instability (frontal dysequilibrium) to isolated akinesia. This spectrum of impairment has been observed, for example, in patients with cerebrovascular disease and progressive supranuclear palsy (Achiron et al., 1993; Riley et al., 1994). The akinetic syndrome has been called frozen gait (Yanagisawa et al., 1991), primary progressive freezing gait
(Achiron et al., 1993) and gait ignition failure (Atchison et al., 1993).

Our study was limited to patients with lower-half parkinsonism due to cerebrovascular disease. Similar studies of patients with sensory ataxia, cerebellar ataxia, progressive supranuclear palsy, Parkinson disease and focal neurological damage are needed to determine the specificity of our results. In addition, such studies should incorporate the tricks that have been used to facilitate gait initiation in patients with frontal lobe and basal ganglia disease. For example, an inverted walking stick (FitzGerald and Jankovic, 1989; Atchison et al., 1993) and stripes on the floor (Yanagisawa et al., 1991) are helpful to some patients, and Petrovici (1968) found that rhythmic commands and music facilitated walking in two of his five patients. Why these tricks are not helpful in all patients is unclear, but the extent to which locomotion is impaired by postural instability versus akinesia may be important. It has been reported in recent studies that postural instability played a greater role in vascular patients than in patients with idiopathic Parkinson disease (Yanagisawa et al., 1991; Trenkwalder et al., 1995). Patients with vascular parkinsonism benefited more from postural support (Yanagisawa et al., 1991), and their postural stability was more sensitive to disturbances of visual and somatosensory feedback (Trenkwalder et al., 1995). Consequently, concomitant vestibulopathy, peripheral neuropathy, poor vision and musculoskeletal pathology could impair locomotion in these patients to a surprising degree. Such contributing conditions are frequently treatable and are particularly common in older people.

Acknowledgements

The authors wish to thank Suzanne Elble, for editing this manuscript and performing the data reduction, Denyse Herrmann for performing the Tinetti and muscle strength assessments and Stephen W. Hill for providing valuable technical assistance in collecting the motion analysis data. This work was supported by grant AG10837 from the National Institute on Aging and by a grant from the Whitaker Foundation. The authors gratefully acknowledge the use of Memorial Medical Center’s out-patient rehabilitation facilities.

References


Received February 20, 1996. Revised May 25, 1996. Accepted June 14, 1996