A Clinical Guide to Assess the Role of Lower Limb Extensor Overactivity in Hemiplegic Gait Disorders

A. Yelnik, MD; T. Albert, MD; I. Bonan, MD; I. Laffont, MD

Background and Purpose—The aim of this study was to assess the role of knee and ankle extensor overactivity in the hemiplegic gait observed in stroke victims and to propose a clinical guide for selecting patients before treatment of a supposed disabling spasticity.

Methods—A standardized physical examination procedure was performed in 135 consecutive stroke patients. All patients were able to walk without human assistance. The period after stroke ranged from 3 to 24 months (mean, $11.5 \pm 7.25$ months). Spasticity was evaluated with the stroke victim in sitting position and during walking. Overactivity of the quadriceps was considered disabling when inducing inability to flex the knee during the swing phase despite adequate control of knee flexion in sitting and standing positions; overactivity of the triceps surae was considered to be disabling when heel strike was not possible despite good control of the ankle flexion in sitting position; triceps retraction was also considered.

Results—Disabling overactivity was observed in 56 (41.5%) patients: 11 times for the quadriceps femoris, 21 times for the triceps surae, and 21 times for both muscles. It was considered to be the main disorder impairing gait among only 16 (12%) patients: 9 for the quadriceps alone, 3 for the triceps alone, and 4 for both. Sitting spasticity of the lower limb was not predictive of disabling overactivity during walking.

Conclusions—Extensor muscle overactivity is one of the components of gait disorders in stroke patients. The difficulty in assessing spasticity and its real causal effect in gait disturbances are discussed. A clinical guide is proposed. (Stroke. 1999;30:580-585.)

Key Words: gait ■ hemiplegia ■ muscle overactivity ■ muscle spasticity ■ stroke
known to vary greatly, especially according to position, increasing in the standing position. While observing how a subject with an abnormal motor pattern walks, it is not easy to distinguish the respective contribution of spasticity, weakness, dystonia, synergistic extension, and loss of sensitivity. Physical examination is usually the only way to evaluate the role of spasticity in hemiplegic gait disorders and must thus be systematically performed.

We conducted a clinical analysis of hemiplegic gait. Our purpose was to assess the role of spasticity among other neurophysiological disorders in order to propose a clinical guide for selecting patients before treatment. Because it is impossible to measure the stretch reflex clinically, attention was focused on muscle overactivity, a symptom, rather than on spasticity, one of its physiological components.

### Subjects and Methods

#### Patients

This study was based on a prospective follow-up of all the patients discharged from our department of physical medicine and rehabilitation who had suffered a single inaugural cerebral stroke confirmed by CT scan. Patients who could not walk without human assistance were excluded, as were those with an orthopedic disorder involving the lower limb or a past history of neurological disease.

#### Methods

A standardized examination, reported in the Appendix, was carried out at each consultation after discharge. The results reported here are those of the last follow-up consultation not exceeding 2 years postdischarge. Data recorded were sex; age at stroke; type and site of the lesion according to CT scan performed at diagnosis; number of months since the stroke; ability to walk without aid, with cane, or with crutch or tripod cane; and walking distance less than 10 meters, 10 to 50 meters, or more than 50 meters at a time. Sensitivity was assessed as normal, impaired, or anesthesia. Motor testing was performed with stroke patients in sitting position; 17 (12.6%) patients were in group 0 (strongly impaired), 48 (35.5%) in group 1 (impaired), 70 (51.9%) in group 2 (good); When motor testing was performed with stroke patients in standing position, 27 (20%) were found to be in group 0, 49 (36.3%) in group 1, and 59 (43.7%) in group 2.

Anesthesia was present in 2 patients, impaired sensitivity in 57 (42.2%).

#### Neurological Examination

Motor testing was performed with stroke patients in sitting position; 17 (12.6%) patients were in group 0 (strongly impaired), 48 (35.5%) in group 1 (impaired), 70 (51.9%) in group 2 (good); When motor testing was performed with stroke patients in standing position, 27 (20%) were found to be in group 0, 49 (36.3%) in group 1, and 59 (43.7%) in group 2.

Anesthesia was present in 2 patients, impaired sensitivity in 57 (42.2%).

Spativity was tested with stroke patients in sitting position: Tonus of the quadriceps was normal in 66 (48.9%) patients, slightly increased in 68 (50.4%), and strongly increased in 1; tonus of the triceps surae was normal in 46 (34.1%) patients, slightly increased in 72 (53.3%), and strongly increased in 17 (12.6%).

Dystonia during walking was observed on the tibialis anterior (with or without tibialis posterior) in 11 (8.1%) patients and was considered disabling 7 times. On toe flexors dystonia was observed in 16 (11.8%; twice associated with tibialis anterior) patients with shoes off; however, among them only 1 case was considered disabling with shoes on. On extensor hallucis longus it was observed in two cases.

As regards orthopedic disorder, 22 patients had a triceps surae retraction, 5 of them had been surgically treated.

Disabling overactivity was observed in 56 (41.5%) patients: 14 times for the quadriceps femoris, 23 times for the triceps surae, and 19 times for both of them (Table 1). Overactivity was considered to be the main trouble impairing gait in only 12% of the patients: 9 for the quadriceps alone, 3 for the triceps alone, and 4 for both of them. Disabling overactivity is related to the existence of spasticity in sitting position (P<0.001), but the existence of a spasticity in sitting position does not infer disabling overactivity during walking.
walking related to sitting spasticity

during swing phase can be also due to other causes such as a difficult to assess clinically, and the insufficient knee flexion in sitting position. Nevertheless, quadriceps overactivity is to be disabling when inducing inability to flex the knee position and then while walking. Overactivity was considered as useful for 3 (2.3%) patients, with knee extension possible despite a nearly complete impairment.

For treatment of spasticity, drugs were used for 11 patients, had been used for 36, and were never used for 88. Alcoholization of the tibial nerve had been performed for 12 patients.

**Discussion**

The aim of this study was to assess the role of overactivity of the knee and ankle extensors in gait disorders in hemiplegics and to propose a clinical guide to help select patients who could be expected to benefit from treatment of supposed disabling spasticity. The 135 patients included in this study were followed after a first and single stroke, with a standardized physical examination. Results are reported for examinations performed at least 3 months and no more than 2 years after the stroke. All patients included had recovered the knee and ankle extensors in gait disorders in hemiplegics over several days demonstrates little change. Finally, time since stroke also has to be considered. Thilmann and colleagues showed that the increase in stretch reflex activity is at a high level between the first and third months and that the stretch reflex gain is significantly reduced when spasticity has been established for 1 year or more. Changes in passive mechanical properties could then be preeminent, in accor-

<table>
<thead>
<tr>
<th>TABLE 1. Groups According to Muscle Overactivity</th>
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<tbody>
<tr>
<td>Group</td>
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<tr>
<td>-------</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>48</td>
</tr>
</tbody>
</table>

Group 1 indicates no spasticity or overactivity; group 2, slight but not disabling spasticity and no overactivity; group 3, disabling overactivity but associated with other disabling neurological trouble; and group 4, overactivity was the only or main trouble. Each patient is represented once, in column “both” or in column “quadriceps” or “triceps.”

Actually, among the spastic patients, 41 (59.4%) had a spastic quadriceps without bad gait influence (Table 2), and 51 (57.3%) patients had a spastic triceps without bad gait influence (Table 3). Furthermore, disabling overactivity during walking was observed in 4 of the 66 (6%) patients with a sitting normal tone of the quadriceps and in 4 of the 46 (8.7%) patients with a sitting normal tone of the triceps surae. Otherwise, quadriceps overactivity was considered as useful for 3 (2.3%) patients, with knee extension possible despite a nearly complete impairment.

<table>
<thead>
<tr>
<th>TABLE 2. Disabling Overactivity of the Quadriceps During Walking Related to Sitting Spasticity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting Spasticity</td>
</tr>
<tr>
<td>--------------------</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Disabling overactivity</td>
</tr>
<tr>
<td>No disabling overactivity</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

patients were thus considered to be being handicapped by an overactivity, but taking into account the other disorders, only 16 (12%) patients were mainly handicapped by overactivity.

It is not easy to assess spasticity at a physical examination. An experimental setup to measure the excitability of the stretch reflex cannot be commonly used in clinical practice. The main clinical scale widely used is the Ashworth scale or the Modified Ashworth Scale. Nevertheless, this scale does not rate spasticity selectively but rather assesses muscle overactivity and muscle shortening globally. Apart from grade 1, it does not assess a real spastic reaction, and, in addition, it does not take into account the velocity of the stretch. Furthermore, interrater reliability does not appear to be good for Modified Ashworth Scale rating of the lower limb after stroke. Besides, it is well known that altered mechanical properties of muscle may contribute to hypertonia in spastic patients. Hypertonia can be caused by contracture or hypoextensibility, which consist of muscle shortening due to a decrease in the number of sarcomeres in series along the myofibrils. The nature of the relationship between spasticity and contracture remains unresolved. Clinical procedures to measure spasticity involve gauging the resistance of the limbs to passive movements and do not allow identification of the different causes of an increase in resistance.

Thus, a simplified pendulum test was chosen here to evaluate spasticity of the quadriceps, and triceps surae spasticity was assessed using the velocity of clonus initiation. However, a recent study involving the pendulum test, published after our work was completed, did not show any difference between intact and affected legs in stroke patients, related to changes in muscle length due to inactivity. Our scale was thus poorly discriminant.

Another difficulty is the clinical variability of spasticity, which has been emphasized. Spasticity increases with stress, weakness, or general disease and when standing and walking. Moreover, the postural influence seems to be particularly marked when the patient exhibits very little supine spasticity. Spasticity or rather overactivity as evaluated during physical examination might be different from the spasticity experienced in normal activities of life, but it is interesting to note that quantification of spastic hypertonia in chronic hemiplegics over several days demonstrates little change.
dance with the findings of Perry et al\textsuperscript{3} concerning gastrocnemius contracture.

The main difficulty is to assess the real role of spasticity in the gait disorders of hemiplegics. To evaluate overactivity is a way to approach this very difficult point. The overactivity observed in some muscles with stroke patients in standing position can be caused by spasticity or other neurophysiological mechanisms. We found in the present study that sitting spasticity does not infer disabling overactivity during walking. This is not surprising because of the enhancement of spasticity in standing position\textsuperscript{3} and in the light of a recent study showing that stretch of the gastrocnemii by knee extension aggravates plantar flexor cocontraction.\textsuperscript{28} Furthermore, this overactivity seems to be only 1 of the components of gait disorder. Actually, 2 patients were strongly disabled by total anesthesia, 76 (56.3\%) patients were principally disabled by muscle weakness, and 8 patients were disabled by dystonia. The overactivity of the quadriceps as defined here may be due to spasticity but also to a synergistic extension motor pattern. As shown by some authors,\textsuperscript{29,30} speed of gait does not seem to be affected by spasticity, in agreement with our experience. Inability to flex the knee during the swing phase is really disabling on stairs but usually does not affect the patients’ speed. It is a complaint frequently expressed by patients as unsightly or sometimes painful. In stance equinus, the causal effect of spasticity is also questionable. Spasticity of the calf muscles has been demonstrated,\textsuperscript{1,2} but its relationship to ankle flexor muscle weakness has not been examined to date. Dietz et al\textsuperscript{22} showed that during the swing phase spastic patients are unable to lift up the foot despite enhanced tibialis anterior activity but without any coactivation of the calf muscles. This is consistent with studies showing that spasticity does not induce inhibition of the antagonist muscles but rather improvement of the agonist muscles.\textsuperscript{28,31}

Studying the effect of treating spasticity could help answer the question of its role in gait disorders. However, such studies are scarce and have used variable methods; results are of debatable value. For some, clinical satisfaction after tibial nerve neurotomy is related to the decrease of the H reflex.\textsuperscript{9} Others report a lack of improved function after local\textsuperscript{5} or general\textsuperscript{6} treatment of lower limb spasticity, whereas others report good results,\textsuperscript{32} especially with neurotomy\textsuperscript{6,9} or botulinum toxin A.\textsuperscript{12–14} Hinderer and Gupta\textsuperscript{13} emphasized that very few of the functional scales are operational for monitoring changes related to spasticity. Although patients were selected for the studies, the selection criteria were not given. The usually small number of included patients could be an indirect argument for the limited role of spasticity in gait disorders. To the best of our knowledge, no study has reported treatment in a large population before selection. In practice, a peripheral motor block can be useful in evaluating the disability induced by the overactivity of some muscles,\textsuperscript{5,8,10,34} but it cannot help determine the precise role of spasticity in this overactivity. Motor block is easily carried out for the tibial nerve but is unusual for the femoral nerve because of the difficulty of selecting among its branches and the risk of loosing strength.

In conclusion, extensor muscle overactivity is 1, but rarely the main, component underlying gait disorders in stroke hemiplegics. The real role of spasticity as a cause of this symptom is clinically difficult to assess. A guide is proposed here to conduct discussion before treatment of a supposed disabling lower limb spasticity.

### Appendix

**Role of Spasticity in Hemiplegic’s Gait Disorders: Clinical Guide**

<table>
<thead>
<tr>
<th>Sensitivity</th>
<th>Orthopedic trouble</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Equinus: ankle dorsiflexion in supine position</td>
</tr>
<tr>
<td>Impaired</td>
<td>With knee extension: _°</td>
</tr>
<tr>
<td>Anesthesia</td>
<td>With knee flexion: _°</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Motor testing</th>
<th>Sitting</th>
<th>Standing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip flexion</td>
<td>Impossible</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>With trunk extension</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Without trunk extension, but incomplete</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Complete, without trunk extension</td>
<td>3</td>
</tr>
<tr>
<td>Knee extension</td>
<td>Impossible</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>With trunk extension</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Without trunk extension, but incomplete</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Complete, without trunk extension</td>
<td>3</td>
</tr>
<tr>
<td>Knee flexion</td>
<td>Impossible</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Over 90°</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>With strength</td>
<td>2</td>
</tr>
</tbody>
</table>
## Appendix

### Ankle dorsiflexion

<table>
<thead>
<tr>
<th></th>
<th>Impossible</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td>With lower limb flexion pattern</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Analytic without strength</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>With strength</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

### Ankle “eversion”

<table>
<thead>
<tr>
<th></th>
<th>Impossible</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td>With lower limb flexion pattern</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Analytic without strength</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>With strength</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

### Spasticity

#### Quadriceps

In supine position: Modified Ashworth Scale

In sitting position

- Pendulum test
  - Similar to other side
  - Slower
  - Impossible

In standing position, alternated movement of knee flexion and knee extension

- Fast
- Slow
- Impossible

#### Triceps surae

In supine position: Modified Ashworth Scale

In sitting position

- Ankle clonus: none
  - With fast stretch
  - With slow stretch
  - Spontaneous

- Alternated movements of ankle flexion and ankle extension
  - Fast
  - Slow
  - Impossible

### Walk

<table>
<thead>
<tr>
<th>Aid</th>
<th>Inside</th>
<th>Outside</th>
<th>Distance in 1 Go</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without</td>
<td>□</td>
<td>□</td>
<td>&lt;10 meters</td>
</tr>
<tr>
<td>Cane</td>
<td>□</td>
<td>□</td>
<td>10 to 50 m</td>
</tr>
<tr>
<td>Crutch cane</td>
<td>□</td>
<td>□</td>
<td>50 to 500 m</td>
</tr>
<tr>
<td>Tripod cane</td>
<td>□</td>
<td>□</td>
<td>&gt;500 m</td>
</tr>
</tbody>
</table>

Hip flexion Adequate/impossible
Knee flexion Adequate/impossible
Ankle dorsiflexion Adequate/impossible
Dynamic foot equinus □
Knee recurvatum □
Circumduction because of Motricity impairment □
Spasticity □
Both of them □
Attention required for walking □
Is speedy walking possible? □
Dystonia
- Tibialis anterior
- Tibialis posterior
- Extensor hallucis longus
- Toes flexors

Disabling Dystonia
- (unable to flex the knee during the walk despite good active knee flexion)
- (triggers off an equinus despite good active ankle flexion)

References
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