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±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

Gait in hemiplegic stroke patients is generally greatly disturbed. Although gait pattern would appear to be similar for all patients, attentive examination, together with electrophysiological studies, reveals a much more complex situation.1–3 Equinovarus is commonly observed, related to premature firing of the triceps surae due to a hyperactive stretch response during limb loading and prolonged firing of the tibialis anterior during the stance phase,2 but weakness of the ankle dorsiflexors is also involved. Insufficient hip flexion and absence of knee flexion, with or without recurvatum, lead to classic circumduction. Weakness of the flexor muscles, spasticity of the extensor muscles, and a synergistic extension motor pattern may be the main causes of gait disturbance.

Spasticity is defined as a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks resulting from hyperactivity of the stretch reflex as one component of the upper motor neurone syndrome. Attention is focused on spasticity because some improvement can be achieved with treatment, but that improvement could be of little importance alongside the other problems such as loss of strength or abnormal motor patterns.4 In hemiplegic stroke patients, treating lower limb spasticity has often failed to improve gait significantly.4–7 Some good results have been obtained with neurotomy8–10 and botulinum toxin A,11–14 but only a small percentage of these patients could benefit from such treatments. In these different studies, the authors considered spasticity to produce a disabling posture of the lower limb, impairing gait by stance equinus, or to generate handicap via an unexplained mechanism. One important problem is the lack of criteria for patient selection, never accurately specified. Actually, there are no data in the literature concerning which stroke patients could benefit from spasticity treatment or the percentage who could be expected to walk better after treatment.

Patient selection before treatment is essential, but no test has been described that would fulfill this purpose. At present, locometer, video tape and force platform recordings, and electromyographic analysis are useful for analyzing gait and spasticity; however, they cannot be used routinely, and they do not differentiate the real influence of spasticity on gait from the effect of other neurological disorders any better than does clinical observation. Moreover, spasticity is commonly measured in the supine or sitting position, although it is
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; a simplified pendulum test was used for the quadriceps femoris by comparison with the other side: same (0), reduced (1), or absent (2); for the triceps surae, the start of a clonus was assessed as spontaneous (3), with slight (2) or speedy (1) stretching, or absent (0). A retraction of the triceps surae was noted if passive dorsiflexion of the ankle was not possible over the anatomic position (0°) with the knee extended. The gait was then observed. In the case of circumduction, we noted if it was related to impairment of hip, knee, or ankle flexion; the presence of overactivity of the quadriceps femoris making the knee unable to flex; or related to spasticity of the triceps surae making the foot unable to flex. In the absence of circumduction we noted whether rapid walking was possible. Presence of dystonia, defined as “a fixed or relatively fixed attitude” was noted, together with the muscle or muscles involved (tibialis anterior, extensor hallucis, toes, flexors).

Disabling Overactivity

Overactivity of the quadriceps was considered disabling when inducing inability to flex the knee during the swing phase of the gait despite a good motor test of the knee flexion in sitting and standing positions. Overactivity of the triceps surae was considered disabling when heel strike was not possible despite good control of ankle flexion in sitting position. Triceps retraction was also considered.

Role of Overactivity in the Gait Disability

Patients were then classified into the following groups depending on the role of overactivity in the gait disorders: Group 1, no spasticity or overactivity; group 2, slight but not disabling spasticity and no overactivity; group 3, disabling overactivity associated with other disabling neurological disorders (anesthesia, impaired or strongly impaired motricity, dystonia); or group 4, overactivity was the only or main disorder.

A $\chi^2$ test has been used to compare groups.

Results

Study Population

One hundred thirty-five consecutive patients were then examined: 91 men, 44 women (mean age, 55 ± 13 years; range, 24 to 85 years). Hemiplegia involved the right side for 72, and the left for 63. The cerebral lesion was an infarct for 99 (73.3%) patients, and a hemorrhage for 36 (26.7%); the lesion site was hemispheric for 121 (89.6%), and brain stem for 14 (10.4%). The mean delay after stroke was 11.5 ± 2.75 months (range, 3 to 24 months). All of them were able to walk: 79 (58.5%) without aid, 29 (21.5%) with a cane, 7 (5.2%) with a crutch, and 20 (14.8%) with a tripod cane; 5 (3.7%) walked less than 10 meters in 1 go, 18 (13.3%) walked between 10 and 50 meters, the 112 (83%) others were able to walk more than 50 meters in 1 go.

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%).

Spasticity 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.
TABLE 1. Groups According to Muscle Overactivity

<table>
<thead>
<tr>
<th>Group</th>
<th>Quadriceps</th>
<th>Triceps</th>
<th>Both</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>19</td>
<td>7</td>
<td>41</td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>18</td>
<td>27</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>48</td>
<td>87</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.

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 ability to walk. Muscle tone of the quadriceps and of the triceps surae, as well as motor testing, was assessed in sitting position and then while walking. Overactivity was considered to be disabling when inducing inability to flex the knee during the swing phase of the gait despite adequate control of knee flexion in sitting and standing positions or when heel strike was not possible despite good control of ankle flexion in sitting position. Nevertheless, quadriceps overactivity is difficult to assess clinically, and the insufficient knee flexion during swing phase can be also due to other causes such as a lack of active hip flexion in early swing. Fifty-six (41.5%) 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 contraction or hypoexsensibility, which consist of muscle shortening due to a decrease in the number of sarcomers 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. 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-
dance with the findings of Perry et al\textsuperscript{3} concerning gastrocne-
mius 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{8,9} or botulinum toxin A.\textsuperscript{12–14} Hinderer and Gupta\textsuperscript{15} 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>

**Motor testing**

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

<table>
<thead>
<tr>
<th>Ankle dorsiflexion</th>
<th>Impossible</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>With lower limb flexion pattern</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Analytic without strength</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>With strength</td>
<td>3</td>
</tr>
<tr>
<td>Ankle “eversion”</td>
<td>Impossible</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>With lower limb flexion pattern</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Analytic without strength</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>With strength</td>
<td>3</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
- Knee flexion
- Ankle dorsiflexion
- Dynamic foot equinus
- Knee recurvatum
- Circumduction because of
  - Motricity impairment
  - Spasticity
  - Both of them
- Attention required for walking
- Is speedy walking possible?
Dystonia

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Disabling Dystonia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tibialis anterior</td>
<td>□</td>
</tr>
<tr>
<td>Tibialis posterior</td>
<td>□</td>
</tr>
<tr>
<td>Extensor hallucis longus</td>
<td>□</td>
</tr>
<tr>
<td>Toes flexors</td>
<td>□</td>
</tr>
</tbody>
</table>

Conclusion

Disabling quadriceps spasticity
(unable to flex the knee during the walk despite good active knee flexion)

Disabling triceps surae spasticity
(triggers off an equinus despite good active ankle flexion)

References

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