Background: Hemiplegia, or hemiparesis, severe impairment of purposeful activation of striated musculature, is the most conspicuous and often most disabling symptom of acute cerebrovascular lesions. Spontaneous improvement of voluntary strength may extend over many months.

Objective: In this archetypical upper motor neuron syndrome we wish to ascertain the degree of functional impairment due to direct contractile impairment of the affected striated musculature.

Design: Maximal tetanic muscle contraction was elicited by electrical stimulation applied directly to the tibialis anterior of the paretic and nonparetic limbs. Maximal forces of the normal limbs were compared with the afflicted limbs both early and late after vascular lesions of the pyramidal tract. Maximal voluntary force of foot dorsiflexion in the same limbs was also determined. Similar measurements were made in healthy control participants.

Setting: Acute hospital, rehabilitation, and outpatient units of a clinical research center.

Patients: Patients with unilateral stroke were studied a few or many weeks after the ictus.

Main Outcome Measures: Comparison was made between contraction strengths induced by maximal tetanic electrical stimulation of the dysfunctional and contralateral unaffected muscles. Maximal voluntary strength of the foot dorsiflexion forces was also measured.

Results: Compared with the range of electrically evoked contractile force of tibialis anterior between the limbs of healthy participants, the directly elicited force in stroke-impaired tibialis anterior was not significantly impaired.

Conclusions: Modes of exercise therapy focused primarily on direct strengthening of striated musculature, as in resistive exercise training, are strategically questionable. Whether other approaches may be more effective remains to be proved. The central disability of the upper motor neuron syndrome is failure of rapid coordinated adjustment of graded high-frequency motoneuron firing in purposeful complex synergies.

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The purpose of this study was to assess whether the contractile capacity of hemiparetic muscle in stroke patients is significantly different from that of unaffected muscle. Because central recruitment of motoneuron activity is impaired in affected limbs, electrical stimulation was used directly to assess contractile capacity.

The loss of foot dorsiflexion at the ankle in hemiparesis is a central feature of the upper motor neuron syndrome. Deficiency of voluntary dorsiflexion and the drop-foot gait indicate that the tibialis anterior (TA) is severely impaired during motions performed both by conscious voluntary effort and by automatic central neural gait mechanisms. Accordingly, the performance of TA was selected for analysis. Similar measurements in control participants ascertained the normal range and symmetry of electrically and voluntarily evoked forces.

Participants and Methods

Informed consent was obtained from 13 healthy control participants, 17 patients with acute hemiplegia or hemiparesis (disease duration, 1-7 weeks), and 14 patients with chronic hemiplegia or hemiparesis (disease duration, 25-624 weeks) (Table). All patients had acute hemiplegia or hemiparesis resulting from cerebral hemispheric infarction or hemorrhage, with one exception (acute patient 6, Table, had infarction of the medullary pyramidal tract). All brain lesions had been confirmed by computed tomography or magnetic resonance imaging. All had increased tendon jerks in the hemiparetic limbs. Not all had clasp-knife spasticity or extensor plantar reflexes. Calf muscle atrophy (maximal circumference, at least 1.0 cm less than the normal leg) was observed in only 1 patient (chronic patient 13, Table). However, TA atrophy was not otherwise evident by clinical inspection and palpation.

Patients with acute disease were in the hospital neurorehabilitation unit (12 men, 5 women; age range, 47-99 years). The patients with chronic disease were living at home (7 men, 7 women; age range, 48-78 years). Patients with bilateral lesions or impaired capacity for cooperation and consent due to aphasia or dementia were not included. The control participants were healthy volunteers without evident neurologic impairment (3 men, 10 women; age range, 24-72 years). None had cerebral imaging procedures.

Participants were seated in a padded chair so that the hip and knee of the extremity to be tested were flexed to 90°. The sock-covered foot was strapped tightly with a 5-cm nylon strap to a flat metal footplate with the foot in 10° of plantar flexion. The isometric torque force of dorsiflexion was sensed by a strain gauge fixed to a rigid metal block that was attached in parallel to the footplate with its axis proximal to the heel. Strain gauge voltage was acquired and analyzed using the Spike 2 Cambridge University computer program (Cambridge Electronic Components, Cambridge, England).

Abnormalities of motoneuron activation thus compromise the voluntary tension developed by muscle that could in turn affect the properties of the contractile proteins. As is well established in muscle biology, the tension demand on the muscle is the stimulus for adding or losing sarcomeres.27 The key question is whether the disturbed central drive produces enough secondary effect on muscle to decrease contractile capacity. If diminished muscular force is an important factor in hemiparesis, then methods of improving muscle strength should be included in the rehabilitation program. If not, other strategies need to be emphasized.

The skin over the tested anterior upper part of the leg was wiped dry with alcohol sponges; the lateral tibial margin was then palpated and marked with ink so that the conductive adhesive pads (Versa-Stim; Electromed, Miami, Fla) could be uniformly situated 0.5 cm from the bony edge. These electrodes were 7 cm long, 4.5 cm wide, and 1 cm apart. The upper pad was trimmed to fit the curve of the upper tibial origin of the TA (Figure 1). The combined electrode resistance ranged from 47 to 60 Ω. The Versa-Stim 380 (Electromed) constant current stimulator delivered 10-millisecond duration, 2.5-kHz sine wave bursts at the rate of 50 per second. This instrument was used because it was specifically designed to deliver a high-intensity stimulus that was comfortable for use in rehabilitation. As anticipated, we found good subjective tolerance of a...
1.5-second duration stimulus, including a 0.4-second ramp rise to steady current. The routine was to deliver three 1.5-second stimulus bursts at 2-second intervals followed by a minimal 2-minute rest, which provided good recovery of force between stimulus epochs. As the stimulus intensity was gradually increased, the subject first reported a buzzing tactile cutaneous sensation, then a painless muscle shortening. The strongest intensities were perceived as brief painful muscle cramps.

Stimulus intensity was increased stepwise until the generated force reached a plateau for several increments or began to decrease. After a rest, the subject was instructed to dorsiflex the foot maximally at the ankle 3 times at 2-second intervals. These contractions ranged from 1½ to 3 seconds. The patient’s unaffected limb was always tested first. The largest force generated by electrical stimulation (stimulus maximal force [StMF]) or voluntary effort (voluntary maximal force [VoMF]) was used for data analysis.

**RESULTS**

Muscular size and strength along with sex and age varied widely among both the patients and the control participants (Table). Among the healthy participants, the StMF elicited from the TA ranged from 32 to 235 newtons (N). To compare the data statistically and graphically, the original force numbers for each subject were normalized (Figures 2, 3, and 4) and the data are presented as mean (SD). For the control participants, the maximal response to electrical stimulation of each left TA was arbitrarily set at 100% as the reference standard for the other direct force measurements in the same subject (Figure 2). Thus, the mean StMF in the right TA of the healthy participants was 96% (SD, 29%) of the left.

The VoMF of foot dorsiflexion was much larger than the StMF because TA was reinforced by extensor digitorum longus, extensor hallucis longus, and peroneus tertius. For the control subjects (Figure 2), with reference to each participant’s left StMF, the average left VoMF was 259% (range, 141%-398%) and on the right the mean VoMF was 249% (range 134%-408%). The mean difference between normal left and right VoMFs was 29% (SD, 22%) of the reference left StMF. With the premise that both electrically induced and voluntary forces should be
symmetrical in healthy participants, these data are the measure of experimental range of our methods.

The patients in our early stroke group (Table; Figure 3) were examined 1 to 5 weeks after the stroke. The StMF of the unaffected limb (NPST) served as the 100% reference standard; among 17 patients, these forces ranged from 21 to 209 N. To our surprise, the mean StMF of the affected limbs (PST) was larger, 126% (SD, 48%). The paired t test value between limbs was just significant (2.16; \( P = .045 \)).

The patients with chronic stroke (Table; Figure 4) were studied between 25 and 437 weeks after the ictus. The 100% reference StMF forces of the unaffected extremity (NPST) ranged from 31 to 308 N. There was still a trend for the PST to be larger in the affected TA, mean 118% (SD, 60%; paired t test, 1.1; \( P = .30 \), not significant). The PST of chronic patient 13 (Table), the only patient with definite calf muscle atrophy, was 135% of the reference NPST. This patient was also the only one with chronic disease whose voluntary paralysis of TA remained complete.

For the early patient group (Figure 3), the mean VoMF of the unaffected limbs (NPV) was 311% (SD, 115%). The mean VoMF of the affected limbs (PV) was less than a third of this magnitude, 90% (SD, 81%). The mean difference between unaffected and affected limb VoMFs was 221% (SD, 149%). For the patients with chronic disease (Figure 4), the mean VoMF of the unaffected limb (NPV) was 278% (SD, 143%) and for the paretic limb (PV) 171% (SD, 166%). The mean difference between individual unaffected and affected limb VoMFs was 107% (SD, 102%). Of course, these were not the same patients, but the mean degree of voluntary paresis in the chronic group had recovered to about half that of the acute group.

With electrical stimulation, the active musculature is the artificial and relatively restricted product of our best practical effort to produce equivalent electrical fields in the regions just lateral to the fibiae and accurately to duplicate the force measurement setups in both limbs. We presume that with an increasing stimulus current the StMF reaches a plateau when the electrical field becomes sufficiently broad and intense to activate the entire TA. We suspect that decreasing dorsiflexor force beyond the maximum signals that current has spread to the peroneus longus, whose action is to plantar flex and evert the foot. In many participants, this eversion could be readily seen.

High-frequency motor unit firing and consequent tetanic muscle contraction is the necessary pattern for strong purposeful movement. Theoretically, tetanic stimulation of the isolated TA motor nerve would be a more precise technique, but preliminary experiments showed that pain makes the procedure intolerable for unanesthetized human participants. The degree of discomfort produced by direct muscle stimulation was readily accepted by all of our participants. The range of asymmetry between the 2 limbs of our healthy participants represents both physiologic and instrumental factors (eg, imperfect symmetry of electrode placements).

We have no obvious explanation for the slightly larger StMF in the paretic TA muscles of our patients, a finding that was statistically significant in the early stages. For those patients whose paretic TA StMF was smaller than the nonparetic, we cannot exclude the possibility of a small element of decreased contractility of the muscle tissue.

Nevertheless, we believe that these data support the conclusion that true failure of contractile muscle force competence following stroke is not a major factor in the common gait and other functional deficits of hemiparesis. Both early and late after the lesion, and even with fairly good recovery of purposeful foot dorsiflexion, the primary manifestation of hemiparesis is impaired initiation and coordination. Although slight atrophy may occur, this contrasts with the gross weakness and atrophy that characterizes motoneuron or muscle disease. With higher-level lesions (Jackson's middle level and Gowen's upper motor neuron), movement dexterity and repertoire are conspicuously impaired relative to force.

Of course, both purposeful voluntary force and improved gait coordination almost always increase following stroke, with or without formal physical therapy. Evidently this improvement is accomplished by recruitment of more motoneurons and by increased motor unit firing rates that produce tetanic muscle contraction. Whether this central nervous system recovery is improved or best improved by instructed voluntary effort focused upon strength is empirically uncertain.

Our extended suggestion from these observations in stroke is that the symptom usually labeled as weakness by patients with primary brain disease does not represent a major pathophysologic disturbance of muscle tissue. Also likely, but unproved, is the hypothesis that impaired persistence and pattern of activation of common path motoneurons account for the operational "weakness" in cerebral palsy, multiple sclerosis, parkinsonism, and other encephalopathies that affect motor performance. Of course, primary voluntary effort may be decreased by associated pain and is decreased in somatization syndromes.


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In stroke, the conceptual basis of therapeutic approaches should be directed toward correcting the retarded, diminished, and interfering patterns of central nervous system malfunction rather than toward the striated musculature, which serves only as the peripheral end organ that generates force. Whether particular adaptive instructional programs can improve upon spontaneous recovery from acute upper motor neuron impairment remains to be proved.50-43

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