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Ankle Muscle Activity Before and After Botulinum Toxin Therapy for Lower Limb Extensor Spasticity in Chronic Hemiparetic Patients

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Abstract

Background and Purpose Recent studies have been made of the novel treatment of lower limb spasticity after stroke with botulinum toxin A, and the results were based mostly on the clinical assessment made before and after treatment. This study investigated the effects of the toxin on ankle muscle activity during gait in patients with severe extensor spasticity. The questions posed were whether the toxin particularly diminishes the so-called premature muscle activity as a major cause of equinovarus deformity and whether different types of altered motor control allow a prediction of the outcome of the treatment.

Methods In 12 chronic hemiparetic outpatients with pronounced lower limb spasticity, we injected 400 U botulinum toxin A into the soleus and tibialis posterior muscles and both heads of the gastrocnemius muscles. Ankle spasticity and complex gait analysis including kinematic electromyography (EMG) of the soleus and tibialis muscles were assessed before treatment and 4 weeks after the injection.

Results Nine patients profited with a reduction of spasticity, improved gait ability, and a more normal temporal pattern of muscle activity with a prominent reduction of the premature activity of the plantar flexors. Eight patients exhibited a qualitative pattern (type I) corresponding to an increased stretch-reflex excitability. Three patients did not profit: their muscle tone, gait ability, and muscle activation remained stable or even deteriorated.

Conclusions This study further supports the beneficial effects of botulinum toxin in the treatment of lower limb extensor spasticity. A correlation was observed between the clinical reduction of muscle tone, functional gait parameters, and a more normal EMG pattern with a predominant reduction of the premature activity of the plantar flexors. The qualitative type of EMG pattern corresponding to an increased stretch-reflex excitability (type I) was a positive predictor for the outcome.

Key Words: botulinum toxins • gait • hemiplegia • muscle spasticity • rehabilitation

Introduction

Botulinum toxin A (BTX) injections have been used successfully in the treatment of upper limb and lower limb spasticity in hemiparetic patients after stroke.[1] [2] [3] [4] [5] [6] Two open studies reported a reduction of muscle tone, larger passive ankle excursion, and an improved gait ability after BTX treatment for lower limb extensor spasticity.[2] [4] In one study, gait analysis (including the measurement of vertical ground reaction forces) showed a significant improvement in gait velocity, stride length, stance symmetry, and the length of the force point of action under the affected foot after the injection of 400 U BTX in four lower limb muscles (soleus, tibialis posterior, and both gastrocnemius muscles).[4]

This study investigated the effect of the toxin on the ankle muscle activity and ankle excursions during gait in patients suffering from severe lower limb extensor spasticity. In this context, the recording of the so-called kinematic electromyography (EMG) is a powerful tool. This involves measurement of the EMG activity from selected leg muscles during gait with either surface (compound activity) or wire electrodes (topical activity). The generally accepted method of evaluation involves rectifying, filtering, and averaging at least 10 strides or more and analyzing on the background of the phase of the gait cycle. Kinematic EMG of equinovarus after stroke reveals premature calf muscle activation in the terminal swing as an important cause of excessive plantar flexion and varus in stroke patients.[7] Similar observations have been made in patients with cerebral palsy.[8] An initial forefoot contact and a lowered stretch threshold of the spastic plantar flexor muscles help promote this untimely activity, which is therefore predominantly of a reflex nature. The plantar flexors normally do not become active until midstance, when they serve to stabilize the ankle, prevent excessive forward rotation as the body moves forward over the stationary foot, and help with the push off of the heel.[9]

In addition to this so-called equinus type of premature calf muscle activation (type I), Knuttson and Richards[10] described two other distinct types of altered motor control in subjects with spastic hemiparesis: decreased muscle activation (type II) and "coactivation type" (type III). The latter shows no response to stretch and is characterized by stereotyped coactivation of several or all muscles. In contrast to responses in the other two groups of patients, there were several muscle groups that were activated in a pattern similar to the normal pattern of activation.

The present study was designed to investigate to what extent BTX injected into calf muscles reduces premature activity of spastic plantar flexors during gait and by how much the functional activity of the plantar flexors diminishes during midstance. An additional question posed was whether there is a correlation between the reduction of muscle activity and improvement, if any, in functional gait parameters or abnormally increased muscle tone. Investigations were made into the possibility that different types of altered motor control would allow prediction of the benefits of the cost-intensive therapy.

Subjects and Methods

Subjects

Twelve chronic hemiparetic outpatients, 2 women and 10 men who signed an informed consent, participated in the study. Their mean age was 56 years (range, 25 to 76 years). Nine suffered from left and 3 from right hemiparesis, with a mean stroke interval of 22.3 months (range, 11.4 to 48 months). In all cases, the cause was a supratentorial ischemic lesion in the territory of the middle cerebral artery.

All patients suffered from at least a marked increase in muscle tone of the lower limb. Patients were tested during passive ankle dorsiflexion in a supine position with the modified Ashworth Scale.[11] One patient scored grade 3 (marked increase in muscle tone), 8 scored grade 4 (passive movements difficult), and 3 scored grade 5 (rigid). Walking barefoot, initial contact was made with the forefoot, and gait was impaired by stance equinus in all cases. Patients could walk independently for at least 10 m with the help of assistive devices: all except 2 walked with a cane and 7 wore a semirigid ankle-foot orthosis (AFO).

Injection Technique

BTX (Botox, Allergan) was injected through 21-gauge needles that were coated with polytetrafluoroethylene (Teflon) except for the tip. These could also be used as EMG electrodes. BTX, which was diluted with saline to a concentration of 10 U/0.1 mL, was injected at two sites close to the motor point that were identified by standard neurophysiological techniques. The toxin was injected only when either a continuous or stretch-induced EMG activity was recorded; otherwise, other injection sites in the vicinity were investigated until a suitable activity was identified. In all patients, the soleus, tibialis posterior, and medial and lateral heads of the gastrocnemius muscles of the affected side were treated with a total dose of 400 U BTX (100 U each).

Measurements

The patients were examined by two independent persons before the treatment and 4 weeks after. Muscle tone was rated with the modified Ashworth Scale.[11] The ankle range of motion was tested with patients in a supine position for dorsiflexion. It was graded from 0 for no increase in muscle tone to 5 for a rigid plantar flexion. The Rivermead Motor Assessment score for the leg and trunk was used to assess motor functions after a stroke.[12]

Gait Analysis

For the measurement of basic gait-cycle parameters, the patients walked 10 m unrestrained at their maximum speed. The time needed for this was measured with a stopwatch, and the numbers of steps were counted. This enabled the calculation of velocity and cadence, and mean stride length was then determined by dividing the gait velocity by cadence and multiplying it by 2.[9]

Further goniograms of both ankle joints and kinematic EMG of the soleus and tibialis anterior muscles of both sides were measured. The patients walked at their own comfortable speed over a conductive walkway (10x2 m) while wearing ballet slippers incorporating conductive areas in the region of the heel and forefoot, enabling the detection of initial and terminal contact.

Biaxial Penny & Giles goniometers (type M 180, output signal sampled at 100 Hz, angular detection accuracy approximately 2.5° according to specifications of the manufacturer) were used for measurement of plantar-dorsal flexion and inversion-eversion of both ankle joints.

For the bilateral recording of the EMG of the soleus and tibialis anterior muscles, Ag-AgCl surface electrodes (diameter, 8 mm) were used; these were attached 1.5 to 2 cm apart on the muscle bellies (output signal sampled 800 Hz). The impedance was checked and kept below 20 k{Omega}. Electrode sites were marked with a water-resistant pen and kept constant during subsequent measurements. Signals were preamplified with standard electroencephalographic Oxford Metric preamplifiers (type 8515) attached to the limb. All signals (ie, contacts, angles, and EMG measurements) were gathered and amplified to the range of 0 to 5 V by a data logger worn by the patient, and these signals were transmitted via a cable to a personal computer. The data were sampled using a Keithley 64-channel card (type 1801) and displayed on-line to ensure that the sampled data were of acceptable quality. Self-developed Asyst software was used to achieve data acquisition.

The EMG data were digitally filtered (band-pass, 10 to 300 Hz), rectified, averaged over at least 10 strides, and time-normalized to the mean cycle duration set to 100%. To quantify the premature and functional muscle activities of the soleus muscle, mean values of the non–low-passed signals were calculated in the interval from 90% to 110% (premature activity) and from 20% to 50% (functional activity) of the cycle duration. It should be noted that clonogenic activity might have contributed to the so-called functional activity, predominantly at the beginning of the selected interval. In accordance with previous findings that the premature activity of the plantar flexors might occur during the terminal swing, we stipulated an interval of measurement ranging from 90% to 110% of the gait cycle.[7]

For visual pattern detection, a low-pass filter was used with an upper limit of 6 Hz to obtain linear envelopes. Ground-contact periods and goniograms were also averaged and normalized with respect to time.

To differentiate between a toxin-induced general reduction of activity (irrespective of the gait phase) and a specific diminution of the premature activity, a ratio of the premature activity and the functional activity of the soleus muscle was calculated. In this way, the problem of varying recording conditions before and after treatment was mitigated. In addition, the EMG patterns of the lower-limb muscles were qualified according to the criteria of Knuttson and Richards[10] (type I, premature calf muscle activation, equinus type; type II, extremely low activity of the soleus muscle; and type III, pathological coactivation of the soleus and tibialis anterior muscles).

Goniograms of the affected ankle were assessed for maximal dorsiflexion at midstance and during both maximal plantar flexion and inversion during swing. A clinically relevant change was assumed when the ankle excursions changed at least 5° after the injection. This limit was derived from experience with videographic measurements of ankle joints in patients with equinovarus deformity (S.H., unpublished results, 1994). An improved initial contact and a better pivoting over the foot required changes at the ankle joint of at least 5°.

Results

Nine of the 12 patients (referred to in the following as subgroup A) reported an improvement 4 weeks after the injection: 7 could walk better, Achilles tendon clonus was diminished in 5, and toe clawing within the extensor synergy was reduced in 4 (Table 1). Their Ashworth scores showed a reduction of 1 point in 6 of the patients and a reduction of 2 points in 3 others. The Rivermead score improved by 1 point in 5 of these patients. Two subjects could actively dorsiflex the ankle with the knee partly flexed and another subject could with an extended knee. Two patients could additionally tap with the nonaffected foot five times while standing on the affected one. Their gait velocity, stride length, and cadence improved on average 33.3%, 16.9%, and 15.7%, respectively.

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Table 1. Variables of Group A Hemiparetic Patients Who Profited From Injection Before and 4 Weeks After Treatment With Botulinum Toxin A

The kinematic EMG of the 9 patients of group A, who profited from the injection, showed a significant reduction of the premature activity of the soleus muscle after the injection, at an average of 35.3% (Figs 1 through 3). At the same time, functional activity of the soleus muscle (ie, during midstance) decreased in 8 of these patients, with an average of 38.6%.

Figure 1. Raw electromyogram of the soleus muscle and basogram of the affected side in a left hemiparetic patient (patient 3 in Table 1) before (top) and after (bottom) botulinum toxin treatment. The basogram symbolizes the foot contact phases of the left lower limb. Note the reduction of the clonogenic activity after treatment. T indicates time.

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Figure 2. Averaged electromyography (EMG) envelopes of the soleus and tibialis anterior muscles and corresponding basograms of the affected (left) and nonaffected (right) side before (dashed lines) and after (solid lines) treatment in the same patient as in Fig 1. Note the reduction of the premature activity of the soleus muscle of the affected side. EMG data were digitally filtered, rectified, averaged over at least 10 strides, and time-normalized to the mean cycle duration (=100%). The present EMG envelopes were obtained after additional low-pass filtering with an upper limit of 6 Hz, which enables visual pattern detection. T indicates time.

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Figure 3. Averaged electromyography envelopes of the soleus and tibialis anterior muscles and corresponding basograms of the affected (left) and nonaffected (right) side before (dashed lines) and after (solid lines) treatment of a left hemiparetic patient (patient 4 in Table 1). T indicates time.

The ratio between the premature and functional activities of the soleus muscle was 0.76 before and 0.66 after the injection. A marked (>25%) to moderate (>15%) decline was observed in 4 and 2 patients, respectively, and an increase was seen in the remaining 3 patients.

The activity of the tibialis anterior muscle markedly increased in 1 of these 9 patients. Clinically, this patient could actively dorsiflex his ankle with the knee flexed in a prostrate position after the injection. In the rest of the group, no obvious changes were detected.

The EMG patterns of 8 patients from group A were classified as type I (ie, premature clonogenic activity of the soleus muscle) and in 1 patient as type II (ie, rather tonic low-amplitude activity throughout the whole stance phase of the soleus muscle).

Clinically relevant changes of ankle excursions were observed in 6 of the 9 patients: greater dorsiflexion during midstance in 3 cases, and reduced plantar flexion in 4 and inversion in 3 cases during swing. The 9 patients of group A who had responded to treatment reported that the beneficial effects waned after 8 to 12 weeks after the injection.

Of the remaining 3 patients (subgroup B; Table 2), 1 reported no benefit and 2 experienced a deterioration after the injection, both complaining about a more unsecured gait after the injection. Their Ashworth and Rivermead scores remained constant, and gait velocity was unchanged in 1 and deteriorated in the other 2 patients (8.3% and 5.6%, respectively). The mean changes of cadence and stride length were -10.5 res. +5.1% in group B.

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Table 2. Variables of Three Patients Who Did Not Profit From Injection Before and 4 Weeks After Treatment With Botulinum Toxin A

Their EMG patterns revealed an increase of the premature activity of the soleus muscle (+69.7% on an average), and the functional activity of the soleus was not changed. In 2 patients, the classification of the EMG pattern was slightly ambiguous, but the pattern corresponded most closely to type III (ie, coactivation of soleus and tibialis anterior muscles during the transition period between stance and swing phases). The other patient (whose poststroke interval of 48 months was the longest in the whole study group) was classified as type II.

Changes of the ankle excursions were not observed in these 3 patients of group B, except for a reduced plantar flexion in 1.

The statistical analysis over the entire group revealed significant changes (nonparametric Wilcoxon test, $P<.05$) of walking speed, stride length, and premature activity of the soleus muscle after the injection. The remaining variables (cadence, functional activity, and angle at the ankle joint of the affected leg) showed no significant change after therapy (Table 3).

View this table: [in this window] [in a new window]	**Table 3.** Variables of All Patients (n=12) Before and After Treatment With Botulinum Toxin A

There were no systemic or local side effects other than a slight weakness of the plantar flexion in 2 patients after the injection.

Discussion

In accordance with the literature, the majority of hemiparetic stroke patients (9 of 12) profited from the injection of BTX into the calf muscles.[1] [2] [3] [4] [5] [6] Eight of these patients showed the EMG pattern type I and one showed type II.[10] Clinically, Achilles tendon clonus and painful toe clawing were reduced, and these patients reported better gait.

BTX reduced their muscle tone as indicated by the Ashworth grades of ankle dorsiflexion. With the reduction, these 9 patients (group A) walked faster at an average of 34%, exceeding a reported 25% threshold of clinical significance.[13] The improvement resulted from both an increase of cadence and stride length, indicating a physiological mechanism.[9]

The reduction of muscle tone and improvement of gait ability both had a correlation with a reduction of the premature activity of the soleus muscle during gait in this subgroup of patients. In equinovarus after stroke, the premature activity of the plantar flexors with its onset already in the terminal swing is an important cause of plantar flexion and varus.[7] With a reduction of the untimely activity of the calf muscles, some patients were able to walk with less plantar flexion and inversion, thereby presumably improving the mode of initial contact as previously shown by the authors.[4]

The neurolytic toxin also reduced the functionally relevant activity of the plantar flexors at midstance, but it was reduced to a lesser extent than the so-called premature activity, which is mainly of reflex origin. This can be seen in the decrease of the ratio between the premature and functional activities of the soleus muscle as defined in "Methods."

There are two possible explanations for this dissociation: (1) the mode of initial contact (plantigrade versus forefoot) is rendered more normal in most patients, thus giving less opportunity for rapid stretch of the calf muscles during the loading phase; and (2) findings in experimental animals suggest action of BTX on the intramuscular fibers of muscle spindles, thus decreasing stretch sensitivity of Ia and II afferent fibers.[14] [15]

The assumption of a lowered reflex activity is supported by the clinical observation of less Achilles tendon clonus and the raw EMG signals of some patients showing fewer clonogenic 5- to 8-Hz bursts after initial contact.

Four patients achieved voluntary dorsiflexion of their affected ankle while lying down, and in 1 patient the activity of the tibialis anterior muscle during gait increased. Again, a possible explanation based on the aforementioned animal studies might be a reduction of reciprocal inhibition due to reduced spindle sensitivity in the extensor muscles.[14] [15] Furthermore, several articles discuss direct action of BTX at the spinal level mediating recurrent inhibitory synaptic processes.[16] [17]

Three patients (group B) did not profit, with 1 exhibiting EMG pattern type II. The generally low EMG activity despite the markedly increased muscle tone (Ashworth score 5) of this patient is probably due to the longest poststroke interval of 48 months, which could lead to structural changes in muscle.[18]

The responses of the other 2 patients of group B most closely resembled EMG pattern type III. As mentioned, type III EMG is characterized by a completely disrupted activation pattern of several or all limb muscles, with virtually absent stretch reaction; therefore, no marked beneficial effect could have been expected from the treatment of only a single muscle group. Nevertheless, general reduction of the increased synergistic muscle tone might have been an advantage, and the additional treatment of the tibialis anterior muscle would have been worth a trial.

In conclusion, the present study adds further support to the beneficial effect of BTX in the treatment of lower limb extensor spasticity in hemiparetic stroke patients. There was a correlation between the clinical reduction of muscle tone (tested with patients lying down), functional gait parameters, and a more normal EMG pattern, with a predominant reduction of the so-called premature activity of the plantar flexors. The qualitative type of EMG pattern corresponding to an increased stretch reflex excitability (type I) can consequently be regarded as a positive predictor for the outcome of the treatment.

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