Weakeness Is the Primary Contributor to Finger Impairment in Chronic Stroke

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Objective: To assess the relative contributions of several neurologic and biomechanical impairment mechanisms to overall finger and hand impairment in chronic hemiparetic stroke survivors.

Design: Repeated-measures design.

Setting: Clinical research laboratory.

Participants: Thirty stroke survivors with chronic hemiparesis. Fifteen subjects had severe hand motor impairment and 15 had moderate impairment, as measured with the Chedoke-McMaster Stroke Assessment.

Interventions: Not applicable.

Main Outcome Measures: The biomechanic factors stiffness and resting flexion torque, together with the neurologic factors spasticity, strength, and coactivation, were quantified by using a custom hand manipulator, a dynamometer, and electromyographic recordings. Both passive and active rotations of the metacarpophalangeal joints of the fingers were examined.

Results: Although subjects in the severely impaired group exhibited statistically greater passive stiffness and resting flexion torque than their moderately impaired counterparts (P<.05), the overall effect of these biomechanic changes appeared small in relation to the deficits attributable to neurologic changes such as spasticity and, especially, weakness. In fact, weakness in grip strength and isometric extension accounted for the greatest portion of the variance between the 2 groups (η² = .40 and η² = .23, respectively).

Conclusions: Thus, deficits in hand motor control after stroke seem to derive mainly from weakness, which may be attributable to the loss of descending corticospinal pathway activation of motoneurons.

Key Words: Hand; Human; Muscle spasticity; Muscle weakness; Rehabilitation; Stroke.

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CHRONIC HEMIPARESIS IS A common outcome of stroke. Roughly one third of all people who experience a stroke will have some residual impairment of the upper extremity.1-3 Hand function is routinely compromised, with impairment of finger extension being the most common contributor.4

Questions remain, however, regarding the origins of these deficits. The stroke produces an initial flaccid paresis, which is gradually replaced by hypertonicity in the muscles flexing the fingers5,6 and a stereotypically flexed resting hand posture. Certain muscle groups are more likely than others to achieve some degree of recovery, such that force production and movement in the flexion direction, for example, is relatively better than that in extension or abduction and adduction.7

These secondary changes to the initial stroke presentation have been attributed to a number of mechanisms. Although some researchers have emphasized biomechanic alterations, such as contracture,7 muscle atrophy,10 and increased muscle stiffness,11,12 other researchers have focused on neurologic changes such as spasticity,5,13,14 excessive coactivation of flexors and extensors,15,16 and reduced reciprocal inhibition.17,18

The relative contributions of the various mechanisms to the overall hand impairment have not been fully evaluated. This knowledge is important because treatment of 1 mechanism may be very different, or even in conflict with, treatment for another mechanism. As an example, questions remain as to whether perceived changes in joint stiffness after stroke have a primarily mechanical7,12 or neurologic7,19 origin. Proposed treatment of the former, with bracing or musculotendon lengthening,20 may not be beneficial if the source is neurologic. Alternatively, treatment of heightened dynamic joint stiffness with drugs reducing muscle excitation, such as botulinum toxin11 or baclofen,22 may not be efficacious if mechanical tissue changes are primarily responsible.

Thus, in our present study, we sought to determine the extent to which each potential mechanism contributes to the overall deficits in motor control. Reflex properties and voluntary muscle activation of both hands were evaluated in 30 stroke survivors with chronic hemiplegia. Based on clinical evaluations, subjects were evenly divided between those with severe and moderate impairment. We hypothesized that the group with severe impairment would show more evidence of neurologically based mechanisms (spasticity, coactivation of flexors and extensors, weakness) but not mechanically based changes (contracture, passive stiffness) than the group with moderate impairment.

METHODS

Participants

Thirty subjects with chronic hemiparesis subsequent to stroke participated in the study. Subjects were recruited through fliers and from a registry of voluntary participants, both approved by the institutional review board of Northwestern University. All subjects had to have a clinical history of a single stroke occurring at least 9 months before testing (range, 10 mo–21 y). Subjects were selected on the basis of severity of hand motor impairment, as assessed by a research occupational therapist.
using the stage of hand section of the Chedoke-McMaster Stroke Assessment (CMSA). Further inclusion criteria included passive supination of the forearm into a neutral posture and the presence of motor activation in the finger flexors. All subjects gave informed consent according to the Declaration of Helsinki.

Fifteen of the subjects were classified as having severe hand impairment (CMSA stage of hand section, stage 2 or 3), whereas the other 15 subjects were classified as having moderate hand impairment (stage 4 or 5). The CMSA consists of a 7-stage rating scale corresponding to 7 stages of motor recovery, which follow a stereotypical course according to Brunnstrom’s 7-stage ratingscale corresponding to 7 stages of motor recovery, rate hand impairment (stage 4 or 5). The CMSA consists of a

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (y)</th>
<th>Time Since CVA (y)</th>
<th>FMA*</th>
<th>Right-/Left-Hand Impairment</th>
<th>Impairment of Dominant/Nondominant Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severely impaired</td>
<td>57±12</td>
<td>6.8±5.4</td>
<td>29±12</td>
<td>6/9</td>
<td>5/10</td>
</tr>
<tr>
<td>Moderately impaired</td>
<td>59±11</td>
<td>5.9±4.2</td>
<td>45±10</td>
<td>9/6</td>
<td>9/6</td>
</tr>
</tbody>
</table>

NOTE. Values are mean ± standard deviation (SD). Abbreviations: CVA, cerebrovascular accident; FMA, Fugl-Meyer Assessment. *FMA score represents the total score for the upper-extremity section (66).

Table 1: Subject Characteristics

Protocol

The less-affected (unimpaired) hand served as the control for the more-affected (impaired) hand for each subject. Although deficits in the unimpaired hand have been reported,

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The experimental trials consisted of a series of either voluntary contractions or imposed MCP rotations, intended to examine spasticity, muscle coactivation, strength, and passive stiffness. Spasticity, a velocity-dependent stretch reflex, was assessed by using the servomotor to impose constant-velocity rotation of the MCP joints from the flexion limit to the extension limit. The fingers were held at the extension limit for 2 seconds before being rotated back to the flexion limit. Subjects remained declaratively relaxed throughout the trial. Two speeds were used: a speed slow enough not to produce a stretch reflex (10°/s) and 1 fast enough to trigger a stretch reflex (300°/s). Three trials were run at each speed.

For 6 additional trials, 3 at 300°/s and 3 at 10°/s, a short cutaneous stimulus was applied to the dorsal surface of the hand immediately before the initiation of stretch through surface electrodes. The magnitude of the pulse was adjusted to a level just below pain threshold. A train of 10 pulses evenly spaced 20-ms apart began 300ms before the start of the stretch, such that the stimulation period ended 100ms before the stretch. This sequence of trials was repeated with stimulation applied to the palmar surface of the hand.

Maximum voluntary isometric MCP extension and flexion torques were then recorded. Each subject was asked to push the proximal phalanges against the vacuum pillow entrapping the fingers whereas the motor maintained a neutral (0° of finger extension) MCP posture. Three trials were performed for both extension and flexion. All trials were followed by 1-minute rest periods. Because of limitations with the servomotor and torque cell, only isometric extension could be maximally performed with the unimpaired hand.

Lastly, small perturbations were applied to the MCP joints to determine the passive characteristics of the fingers. Subjects were asked to relax while the motor imposed a pseudorandom binary sequence (PRBS) of ±2° steps over a 10-second period. The PRBS perturbations were applied at a series of operating points, spaced 10° apart, spanning the available passive MCP range of motion. The same operating points were used for both hands in a given subject.

Analysis

Grip strength was characterized by the maximum force achieved. The force values obtained were averaged across the 2 testing sessions for each hand. The grip strength for the impaired hand was then normalized by the grip strength for the unimpaired hand.

Torque, velocity, and angle data for the other trials were low-pass filtered at 150Hz with a fourth-order Butterworth filter, before sampling at 500Hz through an analog-to-digital board. The data were subsequently digitally low-pass filtered at 10Hz by using a 30-order finite impulse response filter.

Raw electromyographic data were band-pass filtered between 10 to 225Hz before sampling. The signals were then rectified and low-pass filtered at 10Hz to generate envelopes for the electromyographic activity. Envelope amplitudes were normalized by the maximum value recorded during the experimental session.

Flexor spasticity was quantified by using torque measures rather than electromyographic activity because imposed rotation of the MCP joints may trigger a reflex in a number of muscles. The peak torque, referenced from the initial resting torque, was calculated for the period from the start of rotation to the end of the hold phase. The mean peak torque for the slower (10°/s) trials was subtracted from the mean peak torque for the faster (300°/s) trials to obtain the velocity-dependent torque. Because passive tissue characteristics may increase the resistance to stretch for increasing stretch velocities, we attempted to estimate the magnitude of this effect by measuring the velocity-dependent torque for the unimpaired hand. This torque was subtracted from the velocity-dependent torque of the impaired hand to yield the reflex torque. The peak reflex torque was used as the measure of spasticity.

The delay between the start of stretch and the onset of any muscle response was quantified for each fast trial. Baseline levels of electromyographic activity were recorded for the period prior to initiation of the stretch, and the standard deviation (SD) of this activity was determined. The time between stretch initiation and the point in which muscle activity first exceeded the baseline value by 3 SDs was designated as the reflex delay for a given muscle. The mean reflex delay was obtained by averaging these values for each subject.

Isometric strengths in flexion and in extension were estimated by measuring the torque generated while the MCP joints were held in the neutral position. The maximum torque value for each trial was obtained by averaging across a 100-ms window centered at the point of maximum torque in the specified (flexion or extension) direction.

Muscle coactivation was also assessed during the isometric flexion and extension trials. For each isometric extension trial, the maximum extensor electromyographic value was found by averaging across a 100-ms window centered about the point of maximum EDC activity. FDS and FDI activity were then averaged across the same window as the EDC electromyographic activity. Electromyographic activity rather than torque was used to determine the point of maximal activity to account for trials in which subjects were unable to generate an extension torque. The maximum muscle activity for each flexion trial was obtained by averaging all 3 electromyographic signals across a 100-ms window centered at the point of maximum flexion torque. Coactivation analyses were then performed on the ratio of muscle activity during intended flexion or extension trials.

Passive stiffness was computed from the trials of PRBS perturbations. Each trial record was segmented into a series of steps from flexion to extension based on the angular velocity signal. Least-squares estimation was used to fit a parametric model to the recorded system response. One of 2 models was used, depending on the detection of an electromyographic reflex response. A reflex was determined to have occurred if the level of the FDS or FDI electromyographic activity exceeded baseline activity by more than 3 SDs after the initiation of extension. Before the reflex, or if no reflex was detected, a passive second-order inertia (I)–damping (B)–stiffness (K) model was assumed. The parameters were estimated through multiple linear regression using the measured angle, angular velocity, and torque signals and the computed angular acceleration:

$$\tau = I\ddot{\theta} + B\dot{\theta} + K(\theta - \theta_0) + \tau_s$$  \hspace{1cm} (1)

where $\tau$ is the measured torque; $\tau_s$ is the static offset torque; $I$ is the inertia coefficient; $B$ is the damping coefficient; $K$ is the stiffness coefficient; $\theta$ is the MCP angle; $\theta_0$ is the MCP angular velocity; and $\dot{\theta}$ is the MCP angular acceleration.

The full parametric model, which includes terms representing the reflex contribution to the recorded torque, was fit to the portion of the step after reflex initiation using nonlinear least-squares estimation:

$$\tau = I\ddot{\theta} + B\dot{\theta} + K(\theta - \theta_0) + B_r\dot{\theta} + K_r e^{-\frac{(t - t_{\text{reflex}})}{\sigma}} (\theta - \theta_0) + \tau_s + \tau_r t \geq t_{\text{reflex}}$$  \hspace{1cm} (2)

where $B_r$ is the reflex damping coefficient, $K_r$ is the reflex stiffness coefficient, $\alpha$ is the 30ms, $\sigma$ is the 60ms, $t_{\text{reflex}}$ is the time to reflex initiations, and $t$ is the $t - t_{\text{reflex}}$.

The gaussian reflex stiffness term, along with the values of $\alpha$ and $\sigma$, were chosen to mimic the twitch response for a mixed muscle. The optimized values for $I$, $B$, $K$, $B_r$, and $K_r$ were averaged across each perturbation within a trial. The variance accounted for in the torque signal by the model was computed for each trial.

Statistics

To test the impact of the different mechanisms on functional impairment, a doubly multivariate analysis of variance (ANOVA) was performed by using SPSS software. The following dependent variables were used in the analysis: the normalized grip strength, the peak isometric extension (ISO_EXT), the peak spastic reflex torque (SPAS), the flexor/extensor activity ratio during voluntary extension, the extensor/flexor activity ratio during voluntary flexion, dynamic stiffness (K), and static stiffness (OFF_TRQ). The independent variable was subject impairment group. Where the multivariate analysis of variance results proved significant according to the Wilks $\lambda$ value, subsequent univariate ANOVA testing of each dependent variable was performed. The $F^*$ values were computed to estimate the variance explained by each dependent variable.

The impact of cutaneous stimulation on the magnitude of the spastic reflex response was assessed independently. Reflex torques obtained from trials with palmar and dorsal stimulation and without stimulation were compared through a repeated-measures ANOVA.

Finally, to test for an association with global arm impairment, the outcome measures listed above were included in a multiple linear regression versus the upper-extremity FMA score. Model variables were included in a stepwise fashion, with an entry criterion of $P$ less than .05 and a deletion criterion of $P$ greater than .10.

RESULTS

We found that there was an orderly relation between overall impairment severity, evaluated with the CMSA, and several quantitative measures of impairment sources, as revealed by the multivariate analysis (Wilks $\lambda < .001$). Subsequent univariate testing of the dependent variables showed most of the
potential impairment mechanisms tested were significantly different for the 2 subject groups (table 2).

For example, the peak spastic reflex torque was more than twice as great for the severely impaired subjects as for the moderately impaired subjects (fig 1). The threshold delay until the initiation of reflex electromyographic activity was shorter for the severely impaired group, although the difference was marginally insignificant (61 ± 23ms vs 79 ± 27ms, \( P = .06 \)). Pre-conditioning of the stretch reflex with cutaneous stimulation to either the palmar or dorsal surface of the hand had no effect on the magnitude of the spastic stretch reflex (\( P = .53 \)).

Abnormal muscle activation patterns were also seen during voluntary isometric extension of the MCP joints. During voluntary extension, the more severely impaired subjects exhibited a much greater ratio of normalized flexor to extensor activity for the impaired hand (\( 3.17 \pm .17 \) vs \( .33 \pm .29 \), respectively) (fig 2). The higher ratio resulted primarily from smaller extensor activation rather than greater flexor activation. There was no significant difference in extensor coactivation during voluntary flexion between the 2 groups (\( P = .32 \)).

Inability to properly activate the extensors was especially prominent in the severely impaired subjects. The difference in extensor activity during voluntary extension and flexion was much smaller in the severely impaired subjects for the impaired hand (\( .05 \pm .26 \) vs \( .20 \pm .23 \) for the moderately impaired group, \( P < .01 \)) (fig 3).

There were indications that passive resistance to stretch was increased as well. The peak difference in static stiffness between the impaired and unimpaired hand was significant for both groups of subjects (fig 4 provides a representative example). Static stiffness was marginally greater for the severely impaired subjects than for the moderately impaired subjects (\( P = .053 \)). The difference in dynamic stiffness was significantly greater in the severely impaired subjects (\( P = .016 \)) (fig 5 provides a representative example). There was no significant difference between the hands in passive damping for either

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Severe</th>
<th>Moderate</th>
<th>( P )</th>
<th>( \eta^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRIP (% unimp)</td>
<td>22 ± 12</td>
<td>45 ± 16</td>
<td>&lt;.001</td>
<td>.399</td>
</tr>
<tr>
<td>ISO_EXT (% unimp)</td>
<td>10 ± 16</td>
<td>33 ± 26</td>
<td>.007</td>
<td>.232</td>
</tr>
<tr>
<td>SPAS (Nm)</td>
<td>0.88 ± 0.61</td>
<td>0.36 ± 0.37</td>
<td>.008</td>
<td>.226</td>
</tr>
<tr>
<td>( K ) (Nm(^{-2}))</td>
<td>0.011 ± 0.016</td>
<td>(-0.003\pm 0.013)</td>
<td>.016</td>
<td>.191</td>
</tr>
<tr>
<td>CO_FLX</td>
<td>1.06 ± 0.78</td>
<td>0.53 ± 0.46</td>
<td>.032</td>
<td>.153</td>
</tr>
<tr>
<td>OFF_TRQ (Nm)</td>
<td>0.28 ± 0.25</td>
<td>0.12 ± 0.17</td>
<td>.053</td>
<td>.128</td>
</tr>
<tr>
<td>CO_EXT</td>
<td>0.83 ± 0.35</td>
<td>0.65 ± 0.61</td>
<td>.325</td>
<td>.035</td>
</tr>
</tbody>
</table>

NOTE. Values are mean ± SD.

Abbreviations: CO_EXT, extensor:.flexor activity ratio during voluntary flexion; CO_FLX, flexor:extensor activity ratio during voluntary extension; GRIP, grip strength; ISO_EXT, peak isometric extension; \( K \), dynamic stiffness; OFF_TRQ, static stiffness; SPAS, peak spastic reflex torque; % unimp, percentage of value for unimpaired hand.

Fig 1. The difference in the reflex torque response to stretch between the 2 hands for the severely impaired and moderately impaired subjects. Error bars represent 1 SD. The response was larger for the severely impaired subjects (\( P < .01 \)).

Fig 2. The ratio of normalized finger flexor to finger extensor electromyographic (EMG) activity during voluntary isometric extension for the impaired and unimpaired hands.

Fig 3. The difference in the amount of extensor activity during voluntary extension and voluntary flexion for the severely impaired and moderately impaired subjects. Error bars represent 1 SD.
group of subjects \( (P = .371) \). The parametric models fit the data reasonably well; variance accounted for was 79\% \pm 6\% across all hands.

Deficits in strength accounted for the greatest percentage of the variance between the groups, as quantified by the \( \eta^2 \) values. Grip strength of the impaired side relative to the unimpaired side was twice as great for the moderately impaired subjects in comparison with the severely impaired subjects. No significant difference existed in absolute grip strength in the unimpaired hand for the 2 groups (361 \pm 104N vs 352 \pm 105N, \( P = .78 \)).

The multiple-regression results revealed that a model for overall clinical impairment of the upper extremity could be created from the hand impairment variables. The stepwise regression yielded 2 significant variables (ISO_EXT, \( P = .05 \); SPAS, \( P = .05 \)). With these 2 variables in the model, the adjusted \( R^2 \) value was .36.

**DISCUSSION**

We observed quantitative differences in measures of a number of potential mechanisms contributing to impairment between the severely and moderately impaired groups. As hypothesized, neurologically based mechanisms, such as spasticity, coactivation, and weakness, were more evident in the severely impaired group. However, we did also see significant differences in mechanically based mechanisms, namely passive stiffness and offset torque, which are suggestive of contracture.

The relative contributions of these mechanisms, however, suggest that hand impairment after stroke is predominantly a neurologic rather than biomechanic phenomenon. Although passive mechanical tissue characteristics were altered in the impaired hand in stroke survivors, the relative impact on voluntary finger extension was small in comparison with deficits derived from neural mechanisms.

For example, it has been proposed that the increased resistance to imposed stretch in stroke survivors results from changes in intrinsic muscle properties rather than altered activation in response to stretch.\(^{11,12}\) We found no difference in passive damping properties between the impaired and unimpaired hands and only a small, but statistically significant, difference in dynamic stiffness for the severely impaired subjects (\( \Delta K = .01 \text{Nm}/\text{deg} \)).
Similarly, muscle contracture has been described as a major impediment after stroke. Indeed, we did measure greater flexion resting torque in the impaired hand across static MCP positions. This suggests that contracture is present, although low-level activation may actually be contributing to some of the supposed resting torque. Yet, the observed 0.3-Nm increase in resting torque for the severely impaired stroke survivors represents less than 20% of the peak isometric extension that they could generate with the nominally unimpaired hand.

In contrast, the magnitude of the peak spastic reflex response to imposed extension of the MCP joints was 0.9Nm, a substantial value, for the severely impaired subjects. This value is 3 times as great as the resistance to extension from passive tissue changes. (We have previously shown that the spastic reflex torque is neurally mediated.) Surprisingly, however, conditioning with cutaneous stimulation to either the palm or dorsum of the hand did not increase the magnitude of the spastic stretch reflex. The lack of increase did not appear to arise from saturation of the motoneurons because voluntary flexion torques were still greater than spastic responses. Thus, adverse actions of cutaneous input may not be as large a concern as previously suggested by some therapeutic techniques. It should be noted, however, that the magnitude of the cutaneous stimuli remained below the pain threshold. Excitation of the pain fibers via a larger stimulus may lead to an increase in spasticity.

Still, the largest impediment to finger extension was voluntary weakness. For the severely impaired subjects, the deficit in isometric extension strength between the 2 hands was greater than the sum of the increases in the factors resisting finger extension (dynamic and static stiffness and spasticity). Thus, with normal extension strength, finger extension would still be possible despite the impedance due to these other mechanisms. In agreement with other studies, clinical categorization, based on the regression analysis, was closely related to strength, even for the entire arm. Weakness explained the greatest amount of variance between the severely and moderately impaired groups (it should be noted that the CMSA does not test for weakness per se but only the performance of specific finger movements). Flexion strength, as well as extension strength, was diminished, but extension was preferentially impaired, as reported previously.

Of course, it should be noted that flexion strength had to be characterized by using grip force rather than MCP flexion torque because of limitations in the peak torque permissible with our system. The deficits in grip force measured in this study were consistent with the deficits we had measured previously for flexion force at the tip of the index finger. Additionally, we only examined properties at the MCP joint. Although we speculate that findings would be similar for the proximal interphalangeal and distal interphalangeal joints, mechanical properties may prove to have greater impact at these joints.

Certainly, alterations in muscle tissue, such as atrophy or conversion in fiber type, may have contributed to the strength deficits. The magnitude of the strength deficits, however (80% reduction in extension torque, 70% reduction in grip strength), make it unlikely that these changes were primarily responsible. Typical levels of atrophy and fiber conversion in chronic hemiparesis are around 10% to 20%. Indeed, Landau and Sahrmann previously showed that electric stimulation of weak muscles in stroke survivors produced near normal levels of force, even in subjects with chronic hemiparesis.

Thus, the observed weakness most likely results from neurologic mechanisms. Some of the weakness can be attributed to excessive coactivation between finger flexor and extensor muscles. As reported in earlier studies, excessive coactivation between finger flexor and extensor muscles was present in stroke survivors during voluntary extension, and it was greater in the severely impaired than in the moderately impaired subjects. In the past, we found that anesthetization alone of the finger flexor muscles did lead to immediately improved voluntary finger extension in select subjects, although not for those with major impairment.

Deficits in neural activation of the muscle are likely also present and are probably the predominant source of weakness. Losses in strength may result from a reduction in corticospinal activation of spinal segmental neurons, although we currently have no direct evidence supporting this idea. We did, however, routinely record equal or greater EDC activity during voluntary MCP flexion than during voluntary extension, thereby suggesting that the motoneurons could be activated but that it was difficult to do so voluntarily. Indeed, I study reported a reduction in the percentage of the cross-sectional area of the muscle that was active in the paretic arm during maximum voluntary contraction. Muscle activation may also be reduced because of a reduced firing rate of the motoneurons.

Further studies are necessary to elucidate the primary source of weakness. In particular, it would seemingly be beneficial to examine motor unit–firing characteristics, quantify atrophy in the finger muscles, and determine which motor units are active after stroke.

CONCLUSIONS

Weakness of voluntary activation of finger muscles is potentially a prime target for therapy. Studies focusing explicitly on improving voluntary strength through resistance training after stroke have observed positive changes, as summarized elsewhere, although there is a question as to whether the increased strength leads to improved capabilities. At the very least, strength training may serve to enlarge those muscle fibers still under voluntary control. Alternative therapies are also being explored in an effort to increase cortical excitability to increase cortical activation of muscle.

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c. Model HA625-2500 incremental encoder; DynaTech, 1275 Brummel Ave, Elk Grove Village, IL 60007.
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g. DS7A; Digitimer Ltd, 37 Hydeway, Welwyn Garden City, Hertfordshire, AL7 3BE, UK.
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i. SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.