The Effect of Trager Therapy on the Level of Evoked Stretch Responses in Patients With Parkinson's Disease and Rigidity

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Abstract

Objective: To quantify changes of evoked stretch responses (ESR) in the most rigid arm of patients with Parkinson's disease (PD) after Trager therapy.

Methods: Gentle rocking motion associated with this type of manual therapy was imparted to the upper limbs and body of 30 patients for 20 minutes. A pretest and 2 posttests (at 1 and 11 minutes after the treatment, respectively) were performed, consisting of electromyographic (EMG) recordings of the flexor carpi radialis and extensor digitorum communis while the patient's wrist was passively flexed and extended with an amplitude of 60° and a frequency of 1 Hz. Patients received the treatment on the most rigid side of their bodies (ipsi-group) or on the contralateral side (contra-group). Half of patients in each group received the treatment while lying supine on a massage table (ipsi- and contra-supine) or sitting in a chair (ipsi- and contra-sitting).

Results: In general, the level of ESR were reduced by 36% immediately after treatment and remained 32% lower than pretest values 11 minutes after treatment ($F = 41.45$, $P < .05$). Patients who received the treatment lying supine benefited from a 42% reduction of ESR ($F = 4.07$, $P < .05$). The side on which the treatment was performed did not significantly influence the outcome of the treatment ($F = 0.50$, $P > .05$). However, post hoc analysis of the triple interaction (test × side × position) indicated that the sitting position was much less efficient for sustained contralateral effect ($P > .05$).

Conclusions: Results from the present study strongly suggest that it is possible to modify the level of ESR by using Trager therapy. This stretch reflex inhibition may induce a reduction of the muscle rigidity seen in these patients. The present results may eventually lead to the development of a specific complementary therapy for patients with Parkinson's disease and rigidity. (J Manipulative Physiol Ther 2002;25:455-64)

Key Indexing Terms: *Massage; Neurodegenerative Disease; Vibration*

Introduction

Parkinson's disease (PD) is a progressive degenerative disorder characterized by the loss of dopaminergic neurons in the substantia nigra. Clinical signs usually include akinesia, tremor at rest, and muscle rigidity. Dietz et al[1] suggested that a change in intrinsic mechanical properties of the muscle is responsible for the increased muscle rigidity. A more accepted hypothesis stipulates that the increased muscle rigidity is caused by enhanced activity of a "long-latency" component of the stretch reflex.[2] Results from several experiments suggest that the long-latency component of the stretch reflex may originate from a transcortical pathway.[3-6] Electromyographic (EMG) recording of flexor and extensor muscles of the wrist of patients with PD indeed show distinct bursts, characterized as "evoked stretch responses" (ESR), when the hand is passively flexed

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Paper submitted June 4, 2001; in revised form July 3, 2001.

0161-4754/2002/$35.00 + 0 **76/1/126469**
doi:10.1067/mmt.2002.126469

and extended at a frequency of 1 Hz.[7] It is believed that the amount of ESR detected by using EMG correlates well with the level of clinically assessed rigidity.[8]

Several attempts have been made to reduce muscle rigidity in numerous pathologic conditions of the central nervous system by using mechanical devices. For example, high-frequency vibration is often used with success to reduce levels of spasticity.[9,10] The goal of such vibratory stimulus is to create inhibition of the antagonist muscle,[11] hence reducing its background EMG activity. Such high-frequency vibration is, however, not effective for patients who have PD with rigidity.[10] More recently, manual segmental vibration, which consists of imparting low-frequency movements (3.4-4 Hz) to a limb, was shown to produce a brief (a few seconds) but substantial reduction (95%) in the H-reflex response of normal subjects.[12] This result suggested that imparting rocking motions to body segments might alter the activity of reflex pathways. Manual segmental vibration was inspired by Dr Milton Trager's approach, called *Trager Psychophysical Integration*.[13] This approach primarily consists of imparting a series of very gentle, painless, passive rocking motions to the limbs and body.[14] Its effect on chest mobility in patients with chronic lung disease[15] and on shoulder pain of wheelchair users[13] was previously examined. To this day, however, only anecdotal observations have suggested that Trager therapy might reduce the level of muscle rigidity in patients with PD.[16-18] A recent pilot study using clinical methods of evaluation showed that muscle rigidity in patients with PD was reduced after Trager therapy.[19] Nonetheless, the impact of this therapy was never studied by using objective methods of quantification. Hence, the purpose of the present study was to quantify eventual changes in ESR in the upper limb muscles of patients with PD and rigidity after imparted rocking motions associated with the Trager approach.

Methods

Subjects

Thirty-two patients with PD from the Movement Disorders Clinic of the McGill Centre for Studies in Aging were asked to participate in the present study. Two patients were unable to attend the testing sessions. After explaining the general goal of the study, all patients signed an informed consent form. The experimental protocol followed ethical guidelines and rules of the institution in which the experiment took place (University of Quebec in Montréal). Medication regimens remained unchanged for all patients; however, they provided detailed information about their respective medications to allow for better interpretation of results.

The experimental design was composed of 2 main groups: patients in the first main group received the manual treatment on the most rigid side of the body (ipsi-group), and the remaining patients received the treatment on the opposite side (contra-group). In the present study, the "contra" condition was used as a control. A placebo group was not used since patients could not be blinded as to whether or not they had indeed received the treatment. Half the patients in each main group received the treatment in a supine position on a massage table (ipsi- and contra-supine subgroups); the remaining patients received the treatment while sitting in a chair (ipsi- and contra-sitting subgroups). The purpose of the "sitting" subgroups was to provide information about treatment efficacy for patients who cannot lie supine on the massage table. None of the patients participating in the present experiment had such difficulty. Each patient was assigned to a subgroup in a pseudorandom fashion; the number of patients in each subgroup was increased progressively and successively, but the assignment of a patient to a particular subgroup was randomized. Consequently, it was impossible for the person performing the rigidity test to determine which side of the patient's body was treated. As results will show, the contra-sitting position specifically showed little treatment effects, thus validating this experimental design.

Trager Therapy

A licensed Trager therapist (DL) administered the treatment session for all patients. Trager is a form of manual therapy based on the assumption that the therapist is able to establish a communication with the unconscious mind (ie, central nervous system) of the subject. More specifically, the therapist's intent is to create an "imprint" of motion, when motion range is decreased, by promoting the relief of joint stiffness and muscle rigidity. The length of each session was limited to 20 minutes and consisted of gentle manipulation of the shoulder, trunk, leg, arm, and hand. The limb of interest was supported by the therapist and put into motion; gentle rhythmic rocking motion was manually imparted to the limb and surrounding soft tissues, while the patient passively lay supine on the massage table or sat passively in a chair. This type of movement is pleasant and not painful, and the therapist inquired often to ensure that the patient remained comfortable throughout the treatment session. The frequency of imparted rocking motion ranged between 3 and 4 Hz, and the amplitude was large enough to elicit a sensation of passive movement of the limb (1-4 cm). Although the momentum created by the gentle rocking motion was mainly confined to 1 or 2 articulations, low-amplitude movement might also have been felt throughout the body. The patient was expected to do nothing but relax and assimilate the increasing mobility as the muscle rigidity was reduced. It was not possible to standardize each movement imposed by the therapist because the amplitude and frequency of imparted rocking movements had to be tailored for each patient. However, the therapist respected several guidelines, such as the sequence and time spent on each limb and the side of the body to which treatment was given.

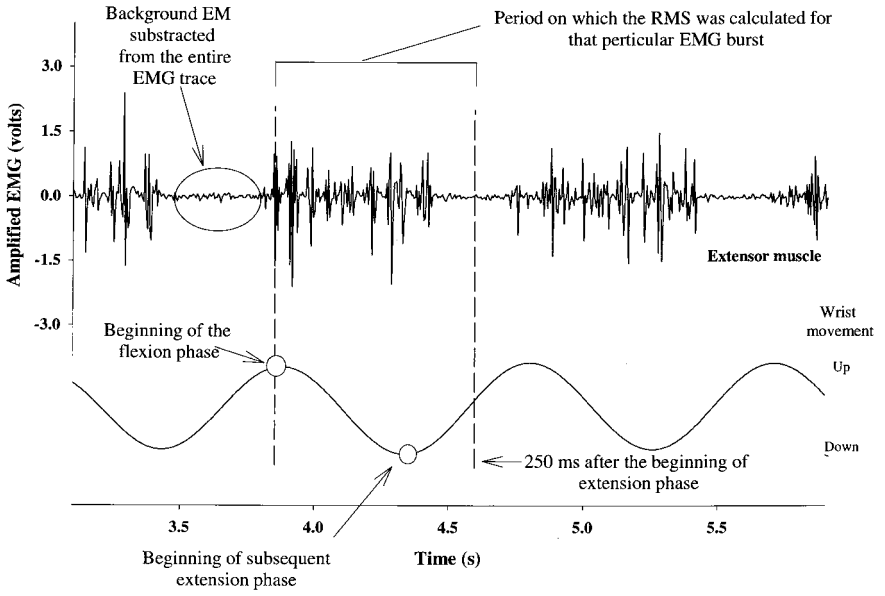

Fig 1. *Examples of EMG trace showing the period on which the RMS was calculated for each EMG burst.*

Evoked Stretch Response

In the present study, the term *evoked stretch responses* (ESR) is used to identify EMG bursts present when the hand of the patient was passively flexed and stretched by the experimentalist performing the rigidity test. The ESR quantification method used here was inspired by Meara and Cody.[8] The experimentalist performing the rigidity test (CD) was blind to which side of the body was treated. Patients were asked to identify their most rigid arm. ESR were quantified in the most rigid arm of each patient before treatment (pretest) and at 1 minute and 11 minutes after treatment (posttests I and II, respectively). Each test consisted of 3 consecutive trials lasting 12 seconds each, during which EMG activity of the extensor digitorum communis and the flexor carpi radialis was recorded. The patient's most rigid arm rested comfortably on a table placed approximately 5 cm below shoulder level with the elbow bent at 90°. The hand hung freely over the edge of the table. The patient's wrist was held firmly by the individual performing the rigidity test. Imposing rhythmic passive flexion and extension of the wrist produced the desired ESR. The hand was moved "up and down" with a frequency and amplitude of 1 Hz and 60°, respectively. A metronome provided the frequency cue, and 2 lines drawn on a piece of cardboard (used as background) provided markers for amplitude. In addition, a goniometer (Biometric Inc, Gwent, UK) was attached to the last digit and wrist of the patient to insure that frequency remained constant between pretest and posttests. ESR were recorded with adhesive preamplified surface electrodes (Therapeutics Unlimited, Iowa City, IA) installed over the belly of the muscles of interest. A fishnet-like bandage was placed over the electrodes to ensure that they remained secure for the entire experiment. Electrodes and goniometer were not removed during the treatment; they were simply unplugged from the amplifier (Therapeutics Unlimited, Iowa City, IA). Plugging the electrodes and goniometer back into the amplifier and allowing for the person performing rigidity tests to reenter the room accounted for the systematic 1-minute delay between the end of treatment and the beginning of posttest I. Between posttests I and II, patients remained supine or sitting and answered questions about their respective medication regimen, impressions of the therapy, and the like.

Data Analysis

EMG signals were acquired at a sample rate of 1000 Hz. Data analysis was conducted using the S-Plus software (Mathsoft, Seattle, WA). EMG data was filtered (high-pass at 20 Hz and low-pass at 400 Hz) by using a fast Fourier transform (FFT) inverse-FFT method. Signal filtering was tapered between 399 and 400 Hz with a ramp of 1 Hz width. This type of filter prevents any signal shift resulting from filtering. Next, each peak and trough from the goniometer signal (representing the onset of the extension phase and the flexion phase, respectively) was manually identified. The root mean square (RMS) of the EMG signal was computed by an automated algorithm between the onset of extension phase and 250 ms after the onset of the subsequent flexion phase (Fig 1). All RMS from the extensor muscles in a particular trial were averaged together. For each trial, the RMS of background noise was subtracted from the RMS of each EMG burst to retain only the significant stretch activation, yielding a normalized EMG activity. RMS of flexor EMG activity was computed by using the same methodology. Since surface electrodes were not removed during the session, raw EMG activity was transformed into percentage

Table 1. *Patient characteristics*

Subjects	Age	Age at unset	H & Y	Rigidity left UPDRS	Rigidity right UPDRS	Presence of motor fluctuation	Side tested	Pharmacodynamic phase during testing†
1	67	60	3	2	2	No	R	None
2	65	60	2	2	1	No	L	None
3	48	41	4	0	2	Yes	R	Negative
4	49	44	4	2	3	Yes	R	Positive
5	74	72	2	2	1	No	L	None
6	69	64	3	3	3	No	R	Negative
7	56	51	2	1	2	No	R	None
8	52	38	3	2	3	Yes	R	Positive
9	72	64	2	3	3	No	R	None
10	66	64	2	2	1.5	No	L	None
11	57	52	3	0.5	1	No	R	None
*12	69	66	2	0	0	Yes	R	Positive
13	52	49	1	2	0	No	L	None
14	55	50	2	2	1	No	R	Negative
15	36	35	2	0	2	No	R	No medication
16	53	46	2	2	2.5	No	R	None
17	76	72	2	1	2	No	R	Negative
18	43	38	3	5	5	Yes	R	Negative
19	69	63	4	2	2	Yes	L	None
20	41	39	2	1	2.5	No	R	None
*21	59	58	1	0	2	No	R	None
22	52	47	3	2.5	2.5	Yes	R	Negative
23	53	52	2	1	2	No	R	No medication
24	64	61	2	3	2	No	L	Negative
25	44	35	2	1	2	No	R	None
26	67	59	4	2	2.5	Yes	R	Positive
*27	57	55	2	0.5	1	Yes	L	Positive
*28	54	53	2	2	2	No	L	None
29	58	54	1	2	0	No	L	None
30	76	67	2	2	1.5	No	R	Negative

*Patients set aside from analysis either because of no rigidity during EMG recording or technical difficulties (patient 28).
†Pharmacodynamic phase relates time of day testing was performed to the last drug dose. *Positive*, patient was probably benefiting from an increase of drug effectiveness during Trager treatment; *negative*, patient was subject to a decrease in drug effectiveness; *none*, patient was either stable or at peak drug effectiveness; *no medication*, patient does not take any anti-parkinsonian drugs; *H & Y*, Hoehn and Yahr; *UPDRS*, unified Parkinson's disease rating scale.

of maximal EMG. Each trial's mean raw RMS obtained from a patient was divided by the highest mean raw RMS found for that particular patient. This method allowed for direct comparisons between pretest and posttests and provided the necessary variance in pretest scores for analysis of variance statistics.

Statistical Methods

Statistical comparisons were tested by using a Side (ipsi or contra) × Position (sitting or supine) × Test (pretest vs. posttests) 3-way factorial design, with repeated measures on the last factor.[20] The a priori significance level to declare a difference as significant was set at $\alpha = .05$, and when significant differences were found, a multiple range Newman-Keuls post hoc test was used.[21] Post hoc statistical power was ascertained by using Tang's method as described by Kirk[21] and is expressed as the probability of type II error, $\beta = 1 -$ power. In addition, repeated measure analysis of variance was used to evaluate pretest and posttest differences for 2 specific comparisons: the first for patients showing ESR in their flexor muscles, and the second for patients who were not on medication or near their respective end-dose period for anti-parkinsonian drugs.

Results

Characteristics of Patients

Table 1 shows the individual characteristics of patients. In brief, 18 men and 12 women with a mean age of 58.5 ± 11 years and a diagnosis of PD were selected for the study. Mean duration of the disease was 4.7 ± 3 years. Patients had mild to moderate rigidity, and their global Hoehn and Yahr stage of illness scores ranged between 1 and 4 (mean: 2.3 ± 0.85). In addition to the 2 patients who did not attend the experimental session, data from 3 patients in which no ESR could be detected on EMG recordings were subsequently rejected from analyses. In addition, data from 1 patient was rejected because of technical problems. As a result, data from 26 patients were analyzed.

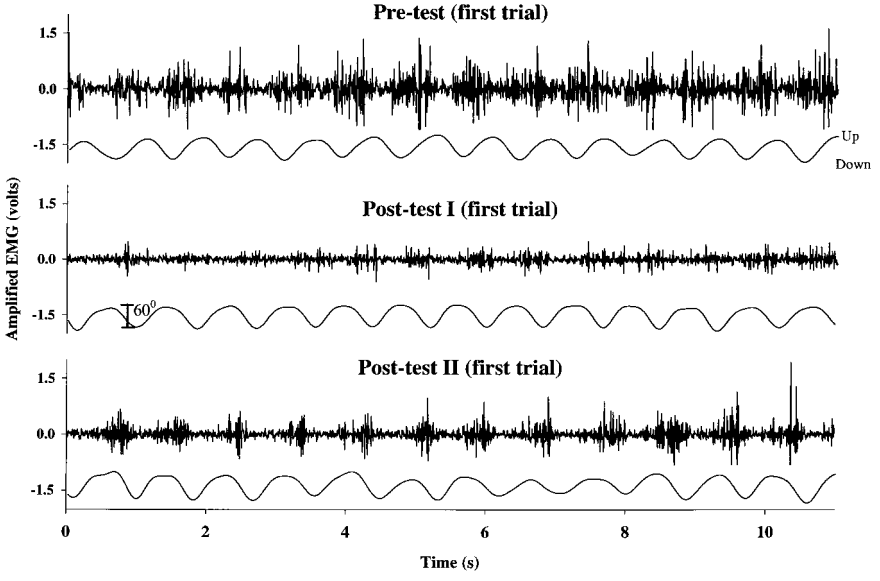

Fig 2. *Evoked stretch responses from extensor digitorum communis, and hand displacement signal in patient no. 15. Notice the important reduction of stretch response in posttest I. Stretch response increased slightly in posttest II, without regaining pretest values. Patient no. 15 received the therapy on the most rigid side of his body, while in sitting position (ipsi-sitting). Patient no. 15 does not take any antiparkinsonian medication.*

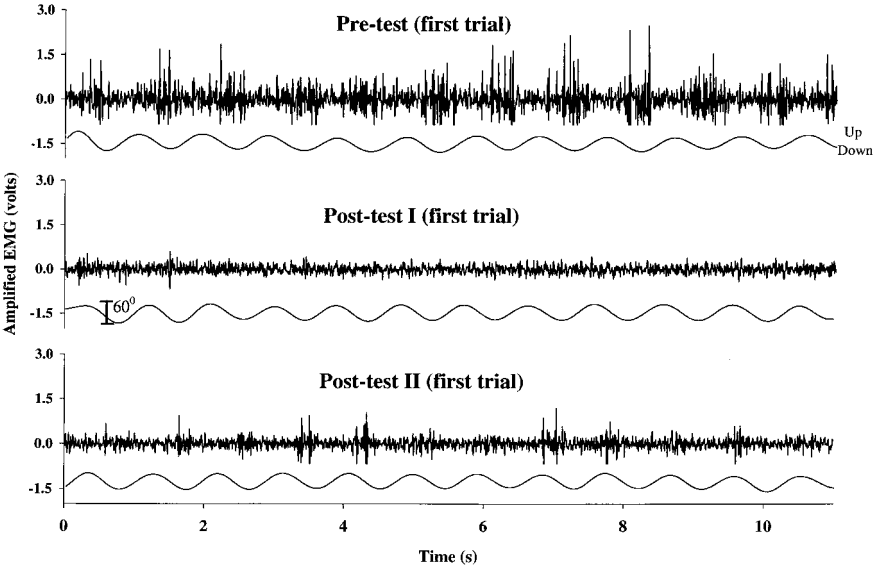

Fig 3. *Evoked stretch responses from extensor digitorum communis and hand displacement signal in patient no. 9. Notice the near complete elimination of stretch response during posttest I. In fact, EMG activity present in this particular trial is similar to what one would expect of the background EMG recorded while the hand is not moving. Stretch response increased in posttest II, without regaining pretest values. Patient no. 9 had taken his antiparkinsonian drugs 6 hours prior to testing (Levodopa-Benzerazide, 1/2 * 100/25 mg) and did not present drug-induced motor fluctuations.*

EMG Stretch Responses

Figure 2 shows the extensor EMG activity associated with ESR in patient no. 15. In this example, the stretch response was considerably reduced in posttest I. In posttest II, the stretch response increased, remaining below pretest amplitude. At the time of the experiment, no anti-parkinsonian medications had ever been prescribed to this patient (*de novo*). In Figure 3, one can observe that the stretch response was absent in the extensor muscles of patient no. 9 during posttest I. Once again, the stretch response increased in posttest II, remaining below pretest amplitude. Surprisingly, patient no. 9 was part of the contra-supine subgroup, a group that was not necessarily expected to benefit from the treatment since the latter was not delivered directly to the measured limb.

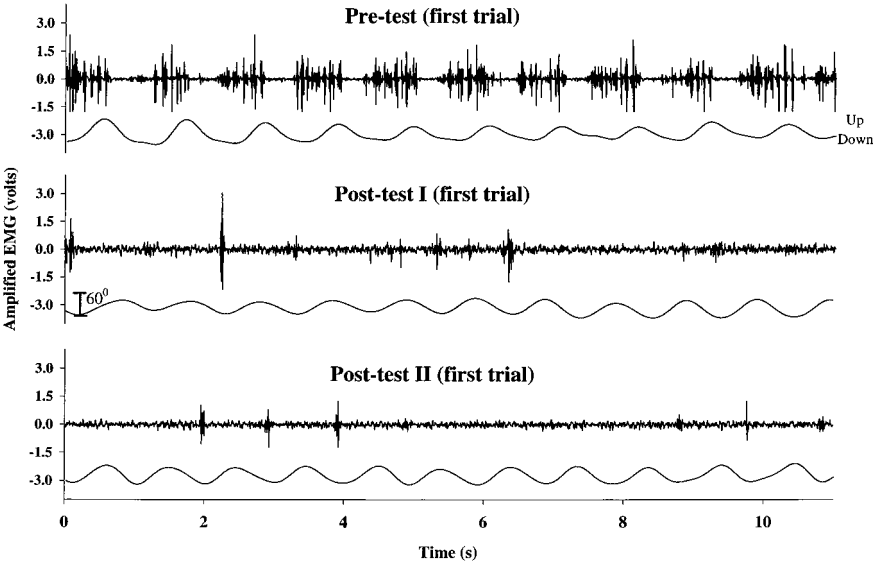

Fig 4. *Evoked stretch responses from flexor carpi radialis, and hand displacement signal in patient no. 22. Stretch responses were markedly reduced during posttest I. Interestingly, stretch response remained low in posttest II. Patient no. 22 was part of the ipsi-supine subgroup. Patient no. 22 had taken his antiparkinsonian drugs 3 hours prior to testing (Levodopa-Carbidopa, 1/2*100/25 mg; Ropinorole, 1 mg) and did not present drug-induced motor fluctuations.*

Table 2. *Summary of statistical comparisons using a 3-way factorial design*

	SS	*df*	MS	F	*P*	Power
Between subjects	1.741	27				
S	0.072	1	0.072	1.155	0.293	
P	0.005	1	0.005	0.082	0.777	
SP	0.168	1	0.168	2.704	0.113	
Error 1	1.495	24	0.062			
Within subjects	3.922	56				
T	2.194	2	1.097	41.450	0.000	0.99
TS	0.026	2	0.013	0.496	0.612	
TP	0.216	2	0.108	4.074	0.023	0.99
TSP	0.216	2	0.108	4.079	0.023	0.99
Error 2	1.270	48	0.026			
Total	5.663	83				

The following significant differences were obtained: test main effect ($F_{2,48} = 41.45$, $P < .05$, $\beta < 0.01$); test position 2-way interaction ($F_{2,48} = 4.07$, $P < .05$, $\beta < 0.01$); and test side position 3-way interaction ($F_{2,48} = 4.08$, $P < .05$, $\beta < 0.01$). *SS*, Sums of square; *MS*, means of square; *S*, sides; *P*, positions; *T*, tests.

Figure 4 shows the same pattern of reduction in stretch response, this time in the flexor muscles of patient no. 22. In this particular patient, even after a period of 11 minutes posttreatment, most of the stretch response was absent.

Group Effect (Extensor Muscles)

Results indicate that ESR were significantly reduced after manual therapy (pretest: 75% ± 18.8 SD; posttest I: 38.6% ± 19.0 SD; posttest II: 42.8% ± 23.8 SD; $F_{2,48} = 41.45$, $P < .05$, $\beta < 0.01$). Analysis of double interactions (test × side) and (test × position) revealed that only the position of patients (sitting or supine) influenced the test results (supine: $F_{2,48} = 4.07$, $P < .05$, $\beta < 0.01$). Table 2 shows the summary of statistical results from the 3-way factorial design used to determine when significant reduction of ESR occurred.

Figure 5 shows the individual and collective results from patients in each subgroup for the extensor muscles. Post hoc analysis on triple interaction, (test × side × position), revealed that the contra-sitting group had the lowest decrease of stretch response in posttest I (Fig 5, *D*), albeit statistically significant ($P < .05$). Contrary to the posttest II of other subgroups who remained statistically lower than in the pretest, results from the posttest II of the contra-sitting subgroup were statistically similar to that of their pretest results ($P > .05$), indicating that the sitting position was much less efficient for sustained contralateral effect.

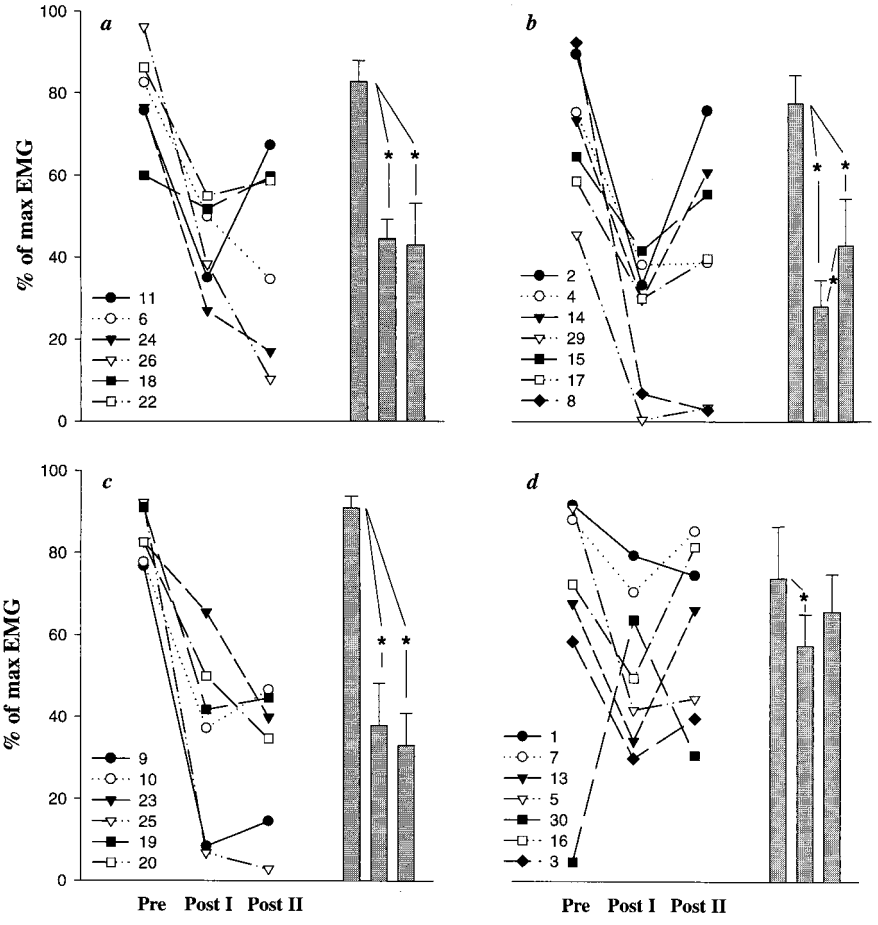

Fig 5. *Individual (points) and collective results (bars) from patients in each subgroup for the extensor digitorum communis muscles. Stars indicate a level of significance at 0.05 resulting from post hoc analysis. a: ipsi-supine; b: ipsi-sitting; c: contra-supine; d: contra-sitting. Vertical bars are presented in the same order as individual results (pre: left bar; post I: middle bar; post II: right bar). Error bars represent the standard error. Note that no statistical differences were found between groups for age (F 0.24, P = 0.87), duration of disease-age at onset (F 0.16, P = 0.92), stage of disease (F 1.27, P = 0.31) and rigidity (left arm; F 2.21, P = 0.11, right arm: F 1.88, P = 0.15).*

Group Effect (Flexor Muscles)

Not all patients showed ESR in their flexor muscles. This fact may be explained by the interpatient variability in location of rigidity. Fig 6 shows individual and collective results from patients who did present some stretch responses in the flexor muscles during their respective pretests (n = 9). A significant reduction of stretch response was detected once again for the test main effect ($F_{2,20} = 7.14$, $P < .05$, $\beta < 0.01$).

Group Effect (Extensor Muscles, Without Positive Drug Pharmacodynamic)

Finally, Figure 7 shows individual and collective results from patients who were either not taking any anti-Parkinson drugs (*de novo*) or near their respective end-dose period (n = 11). A significant reduction of stretch response was detected for the test main effect ($F_{2,20} = 6.22$, $P < .05$, $\beta < 0.01$), meaning that a reduction of stretch response could be achieved without positive drug pharmacodynamic.

Discussion

Significant reduction of ESR was observed in the majority of patients with PD who participated in the present study. The amount of reduction ranged from slight to almost complete. Only patient no. 30 showed increased ESR in his extensor muscles (Fig 5, *D*). In addition, patients no. 1 and 19 showed an increased stretch response in their flexor muscles (Fig 6). Unexpectedly, the same 2 patients showed a reduction in their respective extensor muscles (Figs 5, *C* and *D*). These rare contradictions remain unexplained. Neither patient no. 31, 1, or 19 complained of increased muscle rigidity. In fact, the great majority of patients verbally expressed a sense of well-being after the treatment.

We reject the possibility that the supine position was solely responsible for the reduction of ESR seen in that particular condition; the ipsi-sitting group also showed a significant reduction, which was as powerful as the one seen

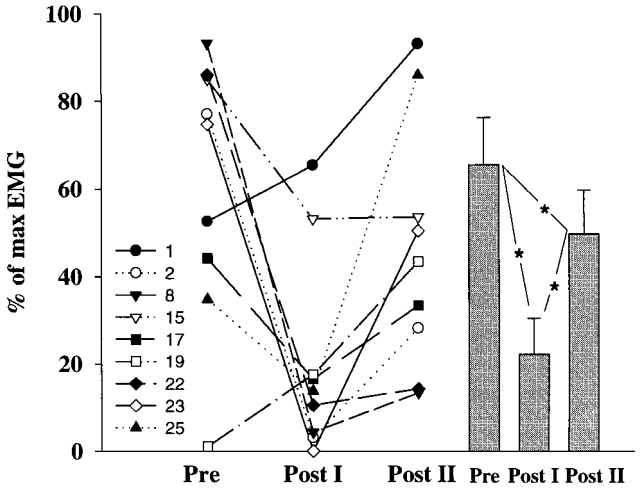

Fig 6. *Individual and collective results from patients showing evoked stretch responses in their flexor carpi radialis during their pretest (n = 9). ANOVA with repeated measures: $F_{2,20} = 7.14$, $P < 0.05$, $\beta < 0.01$. Patients no. 1 and 19 showed an increased stretch response; they belonged to the contra-sitting and contra-supine subgroups, respectively. Stars indicate a level of significance at 0.05 resulting from post hoc analysis. Error bars represent the standard error*

in the supine position. Results seem to indicate that the contra-sitting position is much less significant than other conditions. In fact, scores from the second posttest were not statistically different from pretest scores.

The present results were obtained despite a strict experimental environment that could potentially have increased the level of anxiety of patients. Also of note, the positive effects of the therapy were obtained from a 20-minute session, as opposed to the usual 45 minutes in a clinical setting, indicating that a reduction of ESR can be achieved within a short time frame. The present study showed that patients with PD and rigidity benefited from a significant reduction of ESR after Trager therapy. The significant reduction in ESR was still present 11 minutes after the treatment. If one assumes that muscle rigidity primarily decreased during the 20-minute treatment, patients may in fact have benefited from a 31-minute period in which their muscle rigidity was reduced. Consequently, this reduction must be considered as clinically significant.

Possible Drug Interaction with Trager Therapy

Patients who were not taking anti-parkinsonian drugs, as well as patients near their respective end-dose period, benefited from a significant reduction of ESR (Figs 2 and 7). Consequently, the results of the present study suggest that beneficial outcome can be achieved without positive drug pharmacodynamic. Nonetheless, an interaction between medication and Trager therapy is probable; one possibility is that the medication may render the patients more responsive to imparted rocking motion associated with this type of manual therapy by facilitating the relaxation effect.

In a previous study in normal subjects, the positive effect of manual segmental vibration, which is similar to Trager therapy, was shown to last only a few seconds.[12] In consequence, the reduction of stretch response seen in the present study was not expected to exceed seconds or a minute. It is for this reason that a third posttest was not performed some time after posttest II. A third posttest may have provided a better recovery curve of the level of ESR over time, and is recommended, should this research be extended.

Possible Correlation Between Reductions of Clinically Assessed Rigidity and Evoked Stretch Response

The present study did not directly address whether the reduction of ESR correlated with clinical reduction of rigidity. However, the experimenter conducting the flexion-extension tests often did feel a reduction of rigidity in posttests, suggesting clinical improvement. Furthermore, patients themselves noticed changes in their muscle rigidity. In fact, an earlier pilot study involving 20 patients and a similar experimental protocol as the present study[19] supports clinical improvement after Trager therapy. In this pilot study, an experienced nurse clinically assessed patients by using the rigidity-related test included in the Unified Parkinson's Disease Rating Scale (UPDRS). The nurse was blind to the side on which the Trager therapy was applied. Results showed that UPDRS scores were lower in the posttests when both groups were collapsed (pretest: 1.7 $\pm$ 0.70 SD; posttest I: 1.23 SD $\pm$ 0.91; posttest II: 1.10 $\pm$ 0.97 SD; $F = 8.10$, $P < .05$). These observations support the notion that a reduction of stretch response may be accompanied by a reduction in clinically assessed rigidity after the treatment. As in the present study, EMG measurements of stretch response and clinical evaluation were not performed simultaneously. Consequently, we cannot confirm a direct relationship between the two. Nevertheless, the evidence is compelling and is in agreement with previous findings from Meara and Cody,[8] who showed that the amount of ESR detected by EMG recording correlates well with clinically assessed rigidity when imposed movements to the wrist are repetitive (low frequency of 1-2 Hz) and high in amplitude (60°). Thus, the presence of exaggerated stretch response in patients with PD seen in the present study can be interpreted as indicative of pathologically induced changes in reflex activity, which are probably associated with increased muscle rigidity. In turn, reduction in the stretch response may correlate with a reduction of parkinsonian rigidity.

Possible Neural Circuits Affected by Trager Therapy

Investigating the possible neurophysiologic mechanisms responsible for the reduction in muscle rigidity observed here was beyond the scope of the present study. Nevertheless, the goal and technique of Trager therapy may provide some clues as to why such treatment is effective in reducing the level of ESR in patients with PD and rigidity. Contrary

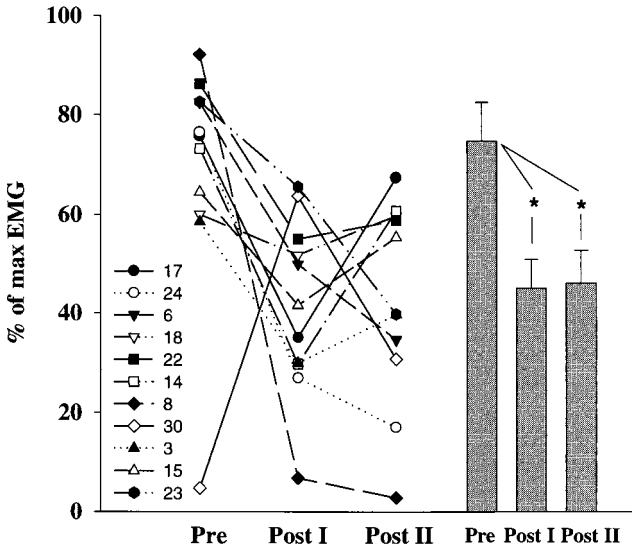

Fig 7. *Individual and collective results from patients either off-medication (subjects 15 and 23) or close to their respective end-dose period for antiparkinsonian drugs. ANOVA with repeated measures:* $F_{2,20} = 6.22$, $P < 0.05$, $\beta < 0.01$. *The patient showing increased stretch response belonged to the contra-sitting subgroup. Stars indicate a level of significance at 0.05 resulting from post hoc analysis. Error bars represent the standard error.*

to most massage therapy in which the muscle is specifically targeted for palpation, Trager therapy focuses mainly on imparting rocking motion to the limb, thereby sending not only cutaneous but also proprioceptive and kinesthetic sensory inputs to the central nervous system. In fact, in the present experimental protocol, the muscle on which EMG was recorded was never directly targeted by the therapist. It is possible that sensory information resulting from this rocking motion may impact on mechanisms responsible for the generation of the stretch response in PD. Imparted rocking motion may help regulate the activity of the long-latency reflex response, which is suspected to be responsible for the increased muscle rigidity.[3-6] EMG traces in Figures 3 and 4 clearly show that it is possible to practically eliminate stretch response in patients with PD for a relatively long period of time by passively imparting rocking motion to the upper limb and body. The suspected modulation of the long-latency stretch response after Trager therapy will be the focus of a subsequent investigation. Understanding the effect of Trager therapy on reflex mechanisms may eventually help increase or optimize the period in which the patient benefits from a reduction of rigidity.

Future Directions

The results of the present study must be interpreted with caution. First, the Trager approach uses motion of the limbs and body, not direct palpation of the muscle. Hence, the present results do not suggest that classical massage will produce similar reduction in muscle rigidity. Second, to reduce inter-patient variability in the treatment technique, one therapist with 20 years experience provided the treatment for all patients. Inter-therapist variability must naturally be anticipated. Most importantly, the present study was not designed as a clinical trial; the number of subjects was relatively low. Hence, a long-term study with a higher number of subjects and suitable control groups receiving either no treatment or other forms of conventional manual therapy (ie, physiotherapy) must be undertaken to evaluate the real impact of the Trager approach on patients' quality of life.

Conclusion

In conclusion, results from the present study strongly suggest that it is possible to modify the level of ESR by using Trager therapy. This stretch reflex inhibition may induce a reduction of the muscle rigidity seen in these patients. The present results may eventually lead to the development of a specific complementary therapy for patients with PD patients and rigidity.

Acknowledgments

This study was supported by donations from Mr Georges Forest, the Trager Institute, and the Fédération des Massothérapeutes du Québec. The authors wish to thank Marie-Josée Fortin for her help in recruiting the subjects, and Katherine Rother for reviewing the manuscript.

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