

From: Christian Duval, research coordinator

To: M. Georges Forest, Trager Institute, Fédération des Massothérapeutes du Québec.

Re: progress report on Trager therapy and Parkinson's disease.

August 1, 2002

Dear *M. Forest*, members of the *Trager Institute*, members of the *Fédération des Massothérapeutes du Québec*;

This letter is for objective to give you a summary of our accomplishments regarding the research on *the effect of Trager therapy on the rigidity of patients with Parkinson's disease*. As will you see, we have been able to build a solid research program and we have collected large amounts of data from more than 50 patients with Parkinson's disease. But most importantly, we communicated our results to the rest of the scientific community with abstracts in scientific meetings (see annex 1-2). We were also the subjects of a Newspaper article in "La Presse" in 1999. Most recently, we were invited to present our results and research program at the first International Symposium on Touch Therapy in Montreal (annex 3). Finally, our findings resulted in a major publication in a peer-reviewed scientific journal (soon to be available in Medline).

In resume, our results showed that the level of evoked stretch response (a measure of parkinsonian rigidity) was substantially reduced after Trager therapy. This reduction remained at least 11 minutes after the end of the therapy. These findings prompted us to propose a new research protocol, which is now looking at the effect of Trager therapy on a specific reflex component of the stretch reflex that is disrupted in Parkinson's disease. This study is currently underway and will enable us to verify if the therapy has an impact on brain functions rather than just a local effect on the muscles, since this abnormal reflex is usually under supraspinal modulatory control. A complete description of the protocol is included at the end of the document (Annex 4).

Once the aforementioned experiments are completed, we anticipate beginning the most important component of the research program: the clinical trial. This clinical trial will compare the effect of Trager therapy with other complementary therapies such as physiotherapy. In addition, it is imperative that we quantify the effect of Trager therapy on the quality of life of these patients. Indeed, we were able to detect changes in muscle rigidity using electrophysiological measurements. Although patients themselves manifested their appreciation of the therapy, these types of measurements do not provide any information on long-term clinical significance of the therapy. We must quantify such clinical impact if Trager therapy is to ever be considered by physicians as a viable complementary therapy for patients with Parkinson's disease. We are currently looking for funding for this specific project.

The aforementioned research project will once again be under the close supervision of our research group:

- Christian Duval, assistant professor at the Faculty of Applied Health Sciences (Brock University).
- Jean P. Boucher, professor at the Department of Kinanthropology (University of Quebec in Montreal).
- Alain Leroux, assistant professor at the Department of Kinesiology (Concordia University).
- Michel Panisset, neurologist and director of the movement Disorders Clinic of the Centre for Studies on Aging (McGill University).
- Denis Lafontaine, a Trager therapist with 20 years experience will coordinate the treatment sessions.

Our research group had two main objectives at the beginning of this ambitious project: first, we intended to explore changes in neural mechanisms associated with parkinsonian rigidity following Trager therapy. We are on the verge of completing this objective. Second, we intend to quantify the effect of Trager therapy on the quality of life of patients with Parkinson's disease. The ultimate goal is to propose a comprehensive model of intervention, using Trager therapy, which will be adapted specifically for patients with Parkinson's disease. This model should be easy to use by therapists, as well as by spouses or close aids of patients. Of course, this model is dependent on results obtained in the aforementioned studies, and most importantly on results obtained from the clinical trial. It is important to note that our research will also serve a springboard to examine the effect of other types of massages, as well as examine the impact of these complementary therapies on other neurological diseases such as cerebral palsy or dystonia.

This research has been an ongoing project for more than three years. Research takes time and effort. Without your support, it would have been impossible to carry out these experiments. We are grateful for your assistance and we hope to continue these exciting research avenues, and ultimately improve the quality of life of patients.

Gratefully yours,

Christian Duval

Jean P. Boucher

Alain Leroux

Michel Panisset

Denis Lafontaine

Annex 1: Abstract presented at the American College of Sports Medicine (ACSM) meeting, May 1999, Indianapolis, United States.

Title: Effects of manual therapy on muscle rigidity in Parkinson's disease.

Authors: Jacques Hébert, Christian Duval, Denis Lafontaine, Michel Panisset, Alain Leroux, Jean P. Boucher.

Purpose: The purpose of this study was to examine the effect of manual therapy on the muscle rigidity of patients with Parkinson's disease (PD).

Procedures: 20 patients with PD were tested for rigidity (Unified Parkinson's Disease Rating Scale) on the side of their body most affected (pre-test). Subsequently, 10 patients received 20 minutes of manual therapy on the tested side (ipsilateral), the others on the contralateral side. Then, rigidity was reassessed twice on the pre-tested arm, immediately after (post-test 1) and 10 minutes following (post-test 2) the manual therapy. The nurse performing the rigidity test was blind to which side manual therapy was applied. Manual therapy involved 12 minutes of gentle tractions, oscillations, vibrations, and rocking motions while subjects lay supine on a massage table. For the remaining 8 minutes, subjects received oscillations, vibrations, and rocking motions more specifically on the arm while sitting in an armless chair. Subjects were also tested for speed and number of steps during a predetermined walking distance.

Results: Rigidity was significantly reduced from the pre-test (score of 1.70 ± 0.71) to post-test 1 (score of 1.10 ± 0.91), independently of the side receiving the manual therapy. No improvements were detected on the walking task. In conclusion, the present results indicate that even one manual therapy session of 20 minutes can significantly reduce muscular rigidity normally associated with PD. The positive short-term effect of manual therapy on muscular rigidity could play a motivating role in the field of functional therapy. Furthermore, future projects will shed more light on long-term effects and the mechanisms underlying the reduction of muscle rigidity.

Annex 2: Abstract presented at the Neuroscience meeting, November 2001, San Diego, United States.

The effect of Trager therapy on the level of evoked stretch response in upper limb muscles of patients with Parkinson's disease. Jean P. Boucher, Michel Panisset, Marc Bélanger*, Denis Lafontaine, Jacques Hébert, Alain Leroux and Christian Duval. Université du Québec à Montréal, Canada, H3C 3P8.

Trager therapy (TT), which uses low frequency, large amplitude oscillation to massage body segments, was shown to decrease the H reflex in normal individuals (Hébert & Boucher, 1999). The goal of the present study was to determine if TT could alter the evoked stretch response (ESR) in patients with Parkinson's disease (PD). Patients (N=30) received the treatment for 20 minutes, either on the most rigid side of their body or on the contralateral side. Half of the patients in each group received the treatment either while laying supine or while sitting in a chair. The ESR was quantified using EMG recording of extensor digitorum communis: the patient's wrist was passively flexed and extended with an amplitude of 60° and a frequency of 1 Hz. ESR evaluation was done before, 1 and 11 minutes after treatment. ANOVA showed that the level of ESR was significantly reduced (36%) following treatment and remained significantly lower (32%) at post-test II. Patients who received the treatment in the supine position benefited from a 42% reduction of ESR. The side on which the treatment was performed did not significantly influence the outcome of the treatment. However, post hoc analysis of the triple interaction (test*side*position) indicated that the sitting position was less efficient for sustained contralateral effect. In conclusions, these results suggest that TT can modify the level of ESR in PD patients. These findings may lead to the development of a complementary therapy for PD. Supported by Mr. Georges Forest, the Trager Institute and the Fédération des Massothérapeutes du Québec.

Trager therapy and Parkinson's disease: potential benefits and future research directions

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and Jean P. Boucher⁴

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2- Exercise Science, Concordia University, Montreal, PQ.

3- McGill Centre for Studies in Aging, McGill University, Montreal, PQ.

4- Dept. of Kinanthropology, Université du Québec à Montréal, Montréal PQ.

Abstract

Introduction: a previous study by Hébert et al. (1998), showed that gentle rocking motion imparted to the leg could modify the reflex response of healthy subjects. However, whether this reflex modulation is simply due modification of local spinal reflex circuitry or the results altered brain modulation on these reflexes remains to be determined. The difference is important in that a change in brain modulation may imply the sensation associated with the therapy modified brain activity itself. One way to verify if Trager therapy modifies brain activity is to study its effect on the rigidity of patients with Parkinson's disease (PD). In PD, reflex circuits that involve the brain itself are modified due to a progressive loss of the neurotransmitter dopamine.

Goal: accordingly, we quantified changes of evoked stretch responses (ESR) in the most rigid arm of Parkinson's disease (PD) patients following 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 pre-test and two post-tests (one 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 body (ipsi-group) or on the contralateral side (contra-group). Half of patients in each group received the treatment laying 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 following treatment and remained 32% lower than pre-test values eleven minutes after treatment ($F = 41.45$, $P < 0.05$). Patients who received the treatment lying supine benefited from a 42 % reduction of ESR ($F = 4.07$, $P < 0.05$). The side on which the treatment was performed did not significantly influence the outcome of the treatment ($F = 0.50$, $P > 0.05$). However, *post hoc* analysis of the triple interaction (test * side * position) indicated that the sitting position was much less efficient for sustained contralateral effect ($P > 0.05$).

Conclusions: results from the present study strongly suggest that it is possible to modify the level of ESR using Trager therapy. This stretch reflex inhibition might induce a reduction of the muscle rigidity seen in these patients. We are now conducting experiments to quantify directly the reflex modulation following Trager therapy. We hope to show that Trager therapy indeed modifies selectively the altered reflexes in PD.

Annex 4: *Research protocol on long-latency reflex in after Trager therapy in Parkinson's disease*

CONCORDIA UNIVERSITY

Part One: Basic Information

1. Names of Researchers:

Names and details on all other researchers involved in this research:

- Christian Duval PhD (c), Dept. Neurology & Neurosurgery, McGill University (514-524-3958)
- Alain Leroux, Ph.D, Dept. Kinesiology, Concordia University(843-8382-3958)
- Jean P. Boucher PhD, Chair, Dept. Knanthropology, UQAM, (514-987-3000, ext.: 4072)
- Michel Panisset MD, Director of the movement disorders clinic, Centre for Studies in Aging, McGill University, (514-766-2010)

2. Title of Research Project:

The effect of Trager therapy on long-latency reflex in Parkinson's disease

3. Granting Agency or Contractor (if any):

Trager institute (USA)

4. Brief Description of Research:

Preliminary results have shown that Trager therapy can modify the amount of evoked stretch response (ESR) in the most rigid arm of patients with Parkinson's disease (PD) (1). In that particular experiment, the objective was to measure, using objective means (i.e. electromyography), the effectiveness of Trager manipulations in reducing the muscle stiffness of patients with PD. Trager therapy consists on manually imparting gentle rhythmic rocking motion to the limbs and surrounding soft tissues, while the patient passively lay supine on the massage table. 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 is usually large enough to elicit a sensation of passive movement of the limb (1-4 cm). Although the momentum created by the gentle rocking motion is mainly confined to one or two articulations, low amplitude movement might also be felt throughout the body. The patient is expected to do nothing but to relax and assimilate the increasing mobility as the muscle rigidity is reduced. In summary, results showed that in general, the levels of ESR were significantly reduced by 36 % immediately following treatment and remained 32% lower than pre-test values eleven minutes after treatment. Patients who received the treatment lying supine benefited from a 42 % reduction of ESR. Results from the present study strongly suggested that it is possible to modify the level of ESR using Trager therapy.

Current evidence suggests that the amount of ESR detected in patients is correlated with clinically detectable rigidity (7). In addition, ESR may originate from a transcortical pathway (2, 3, 4, 5, 6). From the aforementioned observations, we hypothesized that Trager therapy impacted upon the long-loop reflex, which is abnormal in PD (2). Accordingly, we intend to quantify changes in long-latency reflex following Trager therapy. These results may provide valuable information on the impact of Trager therapy on transcortical reflex and provide the bases for the elaboration of a specific complementary therapy for patients with PD.

References:

1. Duval C, Lafontaine D, Hébert J, Leroux A, Panisset M, Boucher JP. The effect of Trager therapy on the level of evoked stretch response in Parkinson's disease patients with rigidity. *Journal of Manipulative and Physiological Therapeutics*. Accepted July 2001, In press.
2. Tatton WG, Lee RG. Evidence for abnormal long-loop reflexes in rigid parkinsonian patients. *Brain research* 1975; 100: 671-676.
3. Marsden CD, Rothwell JC, Day BL. Long-latency automatic responses to muscle stretch in man: origin and function. *Adv Neurol* 1983; 39: 509-39.
4. Cody FWJ, Macdermott N, Matthews PBC, Richardson HC. Observation on the genesis of the stretch reflex in Parkinson's disease. *Brain* 1986; 109: 229-249.
5. Matthews PB, Farmer SF, Ingram DA. On the localization of the stretch reflex of intrinsic hand muscles in a patient with mirror movements. *J Physiol (Lond)* 1990; 428: 561-577.
6. Matthews PB. The human stretch reflex and the motor cortex. *Trends in Neuroscience* 1991; 41(3): 87-91.
7. Meara RJ, Cody FWJ. Relationship between electromyographic activity and clinically assessed rigidity studied at the wrist joint in Parkinson's disease. *Brain* 1992; 115: 1167-1180.

Part Two: Research Participants

1. Sample of Persons to be Studied:

14 patients diagnosed for early stages of Parkinson's disease, having mild to moderate rigidity, will be asked to participate in the present study. In addition, 14 age-matched control subjects in good health will be tested. Exclusion criteria for patients include: the presence of motor fluctuation, tremor-dominant disease and drug-induced dyskinesias. Patients will be asked to refrain from taking their anti-parkinsonian drugs at least 8 hours prior to testing.

2. Method of Recruitment of Participants:

Patients will be recruited at the Centre For Studies in Aging (McGill University). After explaining the general goal and requirements associated with the present study, research nurses will ask patients who qualify for the experimental protocol, as they are scheduled for appointments at the Centre, if they would be interested in taking part to the study. As well, spouses or friends of patients will be asked if they would participate also as control subjects.

3. Treatment of Participants in the Course of the Research:

Experimental design

Patients and subjects will be separated in two subgroups. Accordingly, there will be a patient-treatment subgroup and a patient-non-treatment subgroup. As well, there will be a control-treatment subgroup and a control-non-treatment subgroup (see below).

	Patients		Controls	
	Treatment	Non-treatment	Treatment	Non-treatment
Pre-test	\bar{X}			
Post-test I				
.				
.				
Post-test X				

The experimental protocol will consist of a pre-test of reflex measure (see below for details on reflex quantification methodology), followed by 20-minute Trager treatment for the treatment subgroups. Subsequently, there will be a post-test I immediately after the treatment and subsequent post-tests at 1 minute intervals for 10 minutes. Subjects from the non-treatment group will not receive the Trager treatment, but will be asked to relax as much as possible (self induced relaxation).

Reflex quantification method

The reflex quantification method is inspired by Chen et al. (1998). The median nerve will be stimulated percutaneously by a cathode positioned proximal to the wrist. The electrical stimulus will consist a square wave pulses with duration of 200 μ s, given at a rate of one Hz for 10 s. The intensity of the stimulation will be adjusted to the motor threshold. Electromyographic (EMG) signals from the abductor pollicis brevis will be recorded by bipolar, pre-amplified surface

electrodes. The recording electrodes will be placed over the belly of the muscle. Subjects will be instructed to hold on to a custom-made handgrip and perform sustained isometric thumb abduction (25-30 % of maximal voluntary effort) against a force transducer. Audio feedback will allow subjects to reproduce the expected force. The handgrip will insure that the position of subjects is identical between pre- and post-tests.

EMG signals will be filtered (1-300 Hz) and full-wave rectified. Absolute latencies of reflex components (short and long-latency) will be measured at the onset of EMG spikes. Absolute amplitudes of reflex components will be assessed from the EMG baseline to the peak of the EMG spike. In order to perform inter-subject comparisons, raw amplitudes from each reflex component will be transformed into percentage of baseline EMG activity, calculated 20 ms prior to electrical stimulation. Results from each stimulation will then be averaged over each test.

Statistical comparisons

Statistical comparisons will be performed using a group (patients versus controls) * (treatment or non-treatment) * Test (pre- versus post-tests) 3-way factorial design, with repeated measures on the last factor (20). The *a priori* significance level to declare a difference as significant will be set at $\alpha = 0.05$, and when significant differences will be found, a multiple range Newman-Keuls *post hoc* test will be used (21). *Post hoc* statistical power will be ascertained using Tang's method as described by Kirk (21) and is expressed as the probability of type II error, $\beta = 1 - \text{power}$.

References:

1. Chen CC, Chen JT, Wu ZA, Kao KP, Liao KK. Long latency responses in pure sensory stroke due to thalamic infarction. *Acta Neurol Scand* 1998 Jul;98(1):41-8
2. Kirk RE. *Experimental design: Procedures for the behavioral sciences*. Brooks/Cole Publishing Co.: Pacific Grove (1982).