

Effects of Constraint-Induced Movement Therapy on Patients With Chronic Motor Deficits After Stroke

A Replication

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Background and Purpose—Constraint-induced movement therapy (CI therapy) has previously been shown to produce large improvements in actual amount of use of a more affected upper extremity in the “real-world” environment in patients with chronic stroke (ie, >1 year after the event). This work was carried out in an American laboratory. Our aim was to determine whether these results could be replicated in another laboratory located in Germany, operating within the context of a healthcare system in which administration of conventional types of physical therapy is generally more extensive than in the United States.

Methods—Fifteen chronic stroke patients were given CI therapy, involving restriction of movement of the intact upper extremity by placing it in a sling for 90% of waking hours for 12 days and training (by shaping) of the more affected extremity for 7 hours on the 8 weekdays during that period.

Results—Patients showed a significant and very large degree of improvement from before to after treatment on a laboratory motor test and on a test assessing amount of use of the affected extremity in activities of daily living in the life setting (effect sizes, 0.9 and 2.2, respectively), with no decrement in performance at 6-month follow-up. During a pretreatment control test-retest interval, there were no significant changes on these tests.

Conclusions—Results replicate in Germany the findings with CI therapy in an American laboratory, suggesting that the intervention has general applicability. (*Stroke*. 1999;30:586-592.)

Key Words: arm ■ rehabilitation ■ physical therapy ■ motor activity

Constraint-induced movement therapy (CI therapy) is a new intervention that has, to date, been used mainly for the treatment of the upper extremities of stroke patients.¹⁻⁵ Most of those treated successfully thus far have been chronic patients who experienced stroke at least 1 year before the start of CI therapy. It is estimated that patients amenable to substantial improvement as a result of CI therapy represent at least 50% of the total stroke population.⁵ Most of the work with CI therapy has involved constraining use of the unaffected upper extremity for a period of approximately 2 weeks, while giving the affected arm substantial practice in a variety of motor tasks. Training of the affected arm has frequently included a behavioral technique termed “shaping.” Research has demonstrated that CI therapy produces great improvement of motor function within a period of 2 weeks, that the treatment effect remains stable for many months after the end of therapy, and that it transfers into the everyday lives of patients. A review of treatment approaches in rehabilitation medicine⁶ concluded that CI therapy represents one of the few methods of rehabilitation that has demonstrated efficacy

in controlled experiments and whose therapeutic effects transfer into the “real-world” environment.

The principles of CI therapy are based on earlier basic research with monkeys^{1,7} in whom somatic sensation was surgically abolished from a single upper extremity by dorsal rhizotomy. The monkeys stopped using the affected extremity immediately after deafferentation and never spontaneously regained use of it. However, use of the deafferented arm could be induced either by immobilizing the intact arm for a period of consecutive days or by training the affected arm. The resulting extensive reuse of the deafferented arm was permanent, persisting for the rest of the animal’s life. Experimental evidence indicated that the loss of motor function due to deafferentation was the result of a learned behavioral suppression termed “learned nonuse.”^{1,7}

The same mechanism is thought to apply to humans who suffer mild to moderate hemiparesis after stroke. Despite the fact that patients are often capable of using their affected extremity with reasonably good quality of movement (QOM) when asked to carry out tasks in the laboratory, many of them

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exhibit relative or sometimes essentially complete nonuse of their paretic limb, beginning in the early poststroke period and continuing for the rest of their lives.^{5,8}

It was felt that the techniques that overcome learned nonuse in monkeys after unilateral deafferentation might also uncover latent motor potentials of many stroke patients and thereby constitute a potential treatment to increase upper-limb use. Multiple experiments²⁻⁵ that have applied the unaffected-arm constraint and affected-arm training techniques to stroke patients have supported this hypothesis. Somatosensory deafferentation and stroke obviously involve very different types of lesions. However, the nature of the learned-nonuse mechanism is such that it will come into operation whenever there is an injury resulting in a large initial deficit followed by a period of prolonged recovery. Learning to not try to use an extremity would appear to occur in the initial postinjury period, whatever the nature of the lesion or the consequent deficit, and having developed it tends to persist. The mechanism is viewed as being general, operating within the context of many types of insult to the central nervous system.

CI therapy constitutes a family of treatments. The most frequently used variant involves motor restriction of the unaffected upper extremity by a resting hand splint and sling and training of the affected extremity. However, there are other related variants that are also effective.^{4,5} The effective common factor in all forms of CI therapy appears to be inducing patients to repeatedly practice use of the paretic arm for many hours a day for a period of consecutive days. This massed practice of skills is likely to be responsible for the occurrence of use-dependent increase in cortical reorganization demonstrated with transcranial magnetic stimulation in the patients in this study.⁹ This CI therapy-induced cortical plasticity is presumed to be the basis for the long-term increase in the amount of use (AOU) of the affected extremity. The repetitive training model of CI therapy has received support from recent seminal studies from the laboratory of Mauritz,¹⁰⁻¹¹ in which substantial therapeutic effects were obtained in stroke patients with repetitive concentrated interventions.

Though the results from the application of CI therapy to date are suggestive and encouraging, they must be replicated in other laboratories before therapeutic efficacy can be considered to have been demonstrated conclusively. The question addressed in the current study was whether the results of CI therapy would be the same as in earlier work if it were carried out (1) in a different setting, (2) by different personnel, and (3) in a country where the healthcare system is dissimilar from that in the United States and where patients receive more conventional therapy after stroke. A further difference from earlier work is that patients were accepted for CI therapy as early as one-half year after stroke, rather than 1 year as previously done. The therapy was administered and testing carried out by investigators who received training at the CI Therapy Laboratory (University of Alabama at Birmingham) of one of the coauthors. However, he was not involved in the supervision or collection of data for this experiment. To enable comparison across laboratories, the measurement instruments used here were the same as in

previous research. The experimental protocol was approved by this institution's ethics committee.

Subjects and Methods

Subjects

The subjects were 15 adults (6 female; mean age, 54 years; range, 33 to 73 years) (see Table 1). Twelve patients were married, and 3 lived alone. The patients had experienced an average of 1.2 CVAs (range, 1 to 2 CVAs); mean chronicity since (last) CVA was 5.1 years (range, 0.5 to 17 years). Nine patients exhibited hemiparesis on the right and 6 on the left side; all reported being right-arm dominant before stroke. All patients had received extensive physical therapy, beginning with a mean of 8.4 weeks of inpatient medical rehabilitation in the acute/subacute period and followed by a range of 20 to 300 half-hour sessions per year over the entire period since the time of stroke. Each patient either met or exceeded a minimum motor criterion of at least 20° extension of the affected wrist and 10° of each finger. Other inclusion criteria were the following: no balance problems sufficient to compromise safety when wearing the project's constraint device (3 subjects used a straight cane and no sling while walking in the street but not indoors; the other patients did not use assist devices); no serious cognitive problems (ie, aphasia, attention deficits, visual neglect, disorders of reasoning, and memory); patients were not allowed to score below the sixtieth percentile on tests for these functions [see below]; no serious uncontrolled medical problems; limited spasticity and pain; and a maximum score of 3.0 on the Motor Activity Log (see below), to exclude patients whose good motor functioning was too close to a ceiling for them to be benefited by the therapy. Subjects were recruited by referral from neurologists and personal physicians and by advertisements and an article in a local newspaper. They were screened for suitability by telephone. All patients received separate examinations by a neurologist and a physical therapist to determine that all inclusion criteria were met.

Intervention

Treatment consisted of 2 main elements: (1) restriction of movement of the unaffected upper extremity by placing it in a resting hand splint/sling ensemble for 90% of the hours spent awake for a period of 12 days and (2) training of the affected arm by a procedure termed "shaping" for approximately 7 h/d on the 8 weekdays during that period.

Movement Restriction

The ventrum of the affected lower arm and hand was placed in a resting hand splint that was fastened across its dorsal surface by Velcro straps; it does not permit wrist flexion and grasp and thus prevents the manipulation of objects. The arm in the resting hand splint was then placed inside a sling. Learning to put on and remove the splint/sling ensemble usually required no more than one-half hour of instruction before subjects could accomplish these tasks by themselves without difficulty. A formal behavioral contract with the subject was set up detailing the agreed-upon activities the patient would carry out while not wearing the constraint ensemble (eg, bathing, washing, some aspects of dressing, and any activity in which safety would be compromised) and the activities that the patient would carry out while wearing the resting hand splint and sling (eg, grooming, household tasks, eating).

Shaping

This is a commonly used operant conditioning method in which a behavioral objective (in this case movement) is approached in small steps of progressively increasing difficulty.¹²⁻¹⁴ The subject is rewarded with enthusiastic approval for improvement but is never blamed (punished) for failure. A basic principle is to keep extending motor capacity a small increment beyond the performance level already achieved. A battery of approximately 50 tasks was used for shaping, from which a subset of 15 to 20 was selected for individual subjects. Task objects were frequently used household objects (eg, jars, eating utensils, spring-loaded clothespins), children's toys (eg,

TABLE 1. Subject Characteristics

Subject	Age	Gender	Handedness	Side of Paresis	Territory	Pathomechanism	Time Since Stroke, y	Barthel ADL Score	Copenhagen Stroke Scale Score	Initial Value of WMFT-Time
1	65	F	R	R	Probably A. cerebri media	Ischemic	9	100	43	NA
2	66	M	R	R	A. cerebri media (frontomedial lesions), A. cerebri anterior, probably premotor area (only cortical lesions)	Unknown	2	85	35	NA
3	73	F	R	R	Basal ganglia	Hemorrhagic	7	95	36	37.5
4	41	M	R	L	Probably A. cerebri media	Ischemic	2	95	41	14.6
5	55	M	R	L	Thalamus	Ischemic	2	85	37	29.1
6	42	M	R	L	Unknown	Ischemic	2.5	95	42	14.6
7	66	M	R	R	A. cerebri media (cortical and subcortical areas)	Ischemic	1.5	75	42	5.4
8	52	M	R	R	A. cerebri inferior posterior, bilateral subcortical (brainstem, thalamus, medulla)	Ischemic	0.5	100	42	4.3
9	33	F	R	R	Parietal lobe, motor cortex, subcortical areas (up to dorsal horn of lateral ventricle)	Hemorrhagic	9	100	38	3.6
10	53	M	R	R	Capsula interna	Ischemic	17	100	43	4.3
11	56	M	R	L	A. cerebri media (parietal and subcortical lesions)	Ischemic	5.5	80	39	5.2
12	64	F	R	L	A. cerebri media	Ischemic	14	95	34	18.5
13	59	M	R	L	Capsula interna	Ischemic	0.5	90	42	6.1
14	47	F	R	R	A. cerebri media (cortical and subcortical areas)	Ischemic	3	90	31	32.0
15	44	F	R	R	Thalamus (mainly), basal ganglia	Hemorrhagic	1	90	37	31.9

A. indicates arteria; NA, not available.

building blocks, marbles), and standard devices used in physical and occupational therapy.

Testing

Before pretreatment testing and after posttreatment testing, EMG, EEG, MRI, and transcranial magnetic stimulation studies were carried out; their results will be reported elsewhere. Each subject received a neuropsychological battery, which consisted of the following tests: Aachen Aphasia Test,¹⁵ Attentional Deficit Test Battery,¹⁶ BIT-Sternchen Test,¹⁷ Leistungs-Prüf-Serie (LPS),¹⁸ and Word Association Test (WAT).¹⁹

A distinction was drawn in this study between motor performance carried out at the experimenter's request in the laboratory and use of the affected extremity in the real-world setting. Laboratory motor function was determined by means of the Wolf Motor Function Test (WMFT),^{2,3,20} a 16-item instrument consisting of 13 timed, 2 maximum force exertion, and 1 signature task. The timed tasks were also rated according to 6-step scales (0 to 5) for functional ability (FA) and QOM. The rating scales were developed for use with the Arm Motor Ability Test,²¹ in which they have been found to have high reliability and validity.^{21,22} Real-world outcome was assessed by the Motor Activity Log (MAL), which is a semistructured interview that obtains information about 20 important activities of daily living (ADL) carried out outside the laboratory, from such areas as feeding, dressing, and grooming.^{3,20} Depending on the time of administration, information was obtained about the previous day, weekend, or week. For the last 6 subjects tested, a device (Miltner Constraint Compliance Device) became available for measuring subject compliance with the instruction to wear the movement constraint ensemble for 90% of the waking time spent outside the laboratory (W.H.R. Miltner, S. Franz, E. Taub, unpublished data, 1998).

Procedure

After telephone screening or referral, subjects were brought into the laboratory, usually several months before project intake, for a physical therapy examination to determine their eligibility for the experiment. If subjects were deemed appropriate, they received an explanation of project procedures and signed an informed consent. A structured MAL interview was then carried out to determine the AOU and QOM of the affected arm for the 2 weeks before the "first contact." Fifteen days before the beginning of treatment, subjects received a second MAL interview ("baseline"), and they were also given the WMFT. The MAL and WMFT were administered again on the day before treatment initiation ("pretreatment"). The repetition of these tests after a 2-week interval without treatment was designed as a control procedure to determine whether the simple lapse of time after an intensive testing contact with the project and for the same 2-week period required by the treatment protocol was sufficient to produce a treatment effect. On the day before treatment, the cognitive test battery was presented; in addition, an MAL was administered in a separate room to a subject's significant other. A medical/neurological examination was carried out, usually before the baseline testing day or a few days afterward (in 2 cases). On the day after the completion of treatment ("posttreatment"), the WMFT and MAL were repeated, as was an independently administered MAL for the subject's significant other. The patients returned to the laboratory 4 weeks after treatment ("post-4") and 6 months after treatment ("follow-up") for further testing with the MAL and WMFT and, in addition, were given the MAL by phone for the first 3 weeks after treatment ("post-1" to "post-3"). At the time of this writing, all subjects had completed 1-month follow-up testing, and 12 had completed their 6-month follow-up testing (there was 1 death before the 6-month time point). The MAL was divided into 2 roughly equivalent halves. Before and after treatment, both parts of the MAL

TABLE 2. MAL Scores for Individual Subjects

Subject	AOU						QOM					
	First Contact	Baseline	Pre	Post	Post-4	Follow-Up	First Contact	Baseline	Pre	Post	Post-4	Follow-Up
1	1.8	2.2	2.5	3.7	4.2	3.9	2.1	2.3	2.4	3.4	3.6	3.5
2	1.3	1.7	2.0	3.1	3.4	2.8	1.3	1.7	2.1	2.6	3.3	3.2
3	1.1	1.1	1.1	2.8	3.3	2.8	0.7	0.7	0.7	1.9	2.1	2.1
4	0.5	0.7	0.9	2.2	3.6	3.3	0.5	0.8	0.6	1.5	2.2	2.4
5	0.0	0.3	0.4	2.1	2.4	2.6	0.0	0.4	0.5	1.4	2.0	2.0
6	3.3	3.6	3.8	4.3	4.6	4.2	2.8	3.0	3.1	3.9	4.1	3.7
7	1.7	2.4	2.5	4.0	4.3	NA	1.0	1.7	1.9	3.0	3.4	NA
8	2.3	3.5	3.6	4.4	3.8	4.3	2.3	2.7	3.0	4.0	3.9	4.0
9	2.2	2.3	2.3	4.1	3.3	3.0	2.1	1.8	1.8	3.8	3.3	2.9
10	2.1	1.5	1.4	3.4	4.4	4.4	1.9	1.3	1.4	3.0	4.1	4.4
11	2.3	2.7	2.7	4.4	4.6	4.3	2.0	2.8	2.8	4.5	4.4	4.1
12	0.2	0.3	1.0	3.3	3.6	3.0	0.2	0.3	0.8	2.7	2.8	2.5
13	2.3	2.4	2.4	4.2	4.2	4.5	1.5	1.8	1.9	3.6	4.1	3.8
14	0.8	0.6	1.1	2.4	2.1	NA	0.5	0.3	0.4	0.8	1.3	NA
15	0.7	1.0	1.3	1.9	3.7	NA	0.3	0.8	0.8	1.6	1.3	NA

Pre indicates pretreatment; Post, posttreatment; and NA, not available.

were administered on each testing occasion and both the QOM and AOU scales were used. During treatment, only 1 part of the MAL was used on individual days (to avoid patient boredom due to repetition). In addition, just the QOM scale was given during treatment; when use of the unaffected arm is prevented, the AOU scale for the affected arm yields an artificially inflated score.

Treatment days began with administration of the MAL, followed by discussion of the amount of compliance since the last treatment day and how compliance could be improved. Shaping of performance on a variety of tasks was then carried out for the remainder of the morning and for a 4-hour period in the afternoon. Eating lunch in the university cafeteria was carried out with the more-affected hand; the subject was helped to the extent required to enable eating (eg, carrying a tray, cutting meat).

Results

Motor Activity Log

Table 2 displays individual test scores of the 2 MAL measures obtained at first contact, baseline, pretreatment and posttreatment, and at post-4 and follow-up. Figure 1 presents the group means and SEs of the 2 MAL scores at each of the main time points of the experimental procedure for all 15 subjects up to the 1-month follow-up point and for the 12 subjects who had reached the 6-month follow-up at the time of this writing. Figure 1 also contains the data from the patients' significant others. Separate repeated-measures ANOVAs for AOU and QOM scales with side of hemiparesis as a between-subjects factor revealed significant differences for both scales from first contact to 6-month follow-up for the 12 subjects who reached that time point [$F_{\text{AOU}}(5,55)=62.07$, $P<0.0001$, $\epsilon=.397$; $F_{\text{QOM}}(5,55)=75.66$, $P<0.0001$, $\epsilon=.519$]. Additional separate analyses of contrasts indicated that there was a significant treatment effect from first contact to follow-up [$F_{\text{AOU}}(1,11)=121.9$, $P<0.0001$; $F_{\text{QOM}}(1,11)=154.74$, $P<0.0001$], and from pretreatment to the fourth week post-treatment [$F_{\text{AOU}}(1,11)=98.46$, $P<0.0001$; $F_{\text{QOM}}(1,11)=121.69$, $P>0.0001$]. However, the scores did not change

significantly between first contact and baseline, between baseline and pretreatment, or between post-4 and follow-up. No main effect was found for side of hemiparesis, nor was there a significant interaction between side of hemiparesis and treatment in this analysis or any of those reported below.

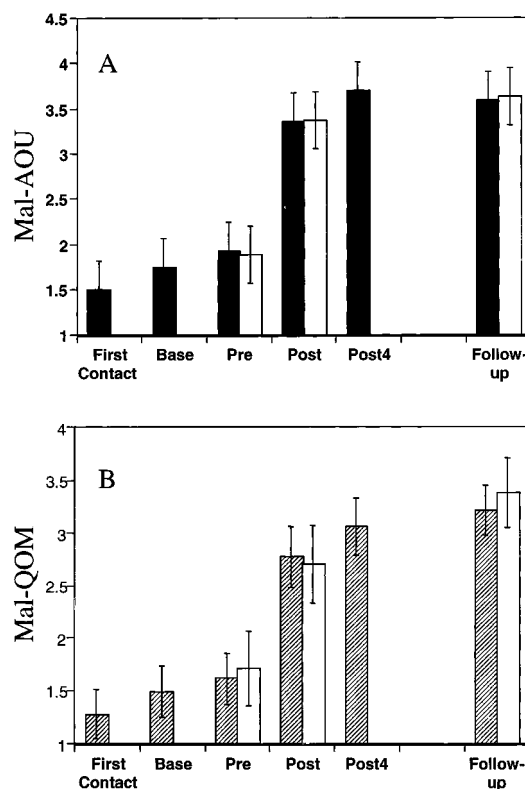


Figure 1. MAL scores for AOU (panel A) and QOM (panel B) for subjects (filled bars) and significant others (open bars) at first contact, baseline, pretreatment, posttreatment, post-4, and follow-up (month 6).

TABLE 3. WMFT Scores for Individual Subjects

Subject	FA				QOM				Time, s			
	Baseline	Pre	Post	Follow-Up	Baseline	Pre	Post	Follow-Up	Baseline	Pre	Post	Follow-Up
3	2.99	3.3	3.4	NA	2.93	2.8	3.2	NA	14.6	16.5	9	NA
4	1.9	2.6	2.8	3.8	1.9	2.4	2.5	2.4	37.5	42.9	24	27.2
5	2.4	2.1	3.6	4.5	1.9	1.6	3.7	3.8	29.1	30.8	10.5	7.2
6	2.99	3.7	4.0	2.4	2.93	3.9	4.1	4.5	14.6	6.3	5.3	5.0
7	4.1	3.9	4.5	NA	4.2	3.9	4.5	NA	5.4	4.1	3.5	NA
8	4.2	4.4	4.7	5.0	4.1	4.4	4.6	5.0	4.3	3.4	3.4	2.7
9	4.2	4.1	4.9	4.8	4.2	4.1	4.9	4.8	3.6	3.1	4.0	4.3
10	3.4	3.6	4.5	4.5	3.5	3.6	4.5	4.5	4.3	4.6	3.6	4.1
11	3.3	3.1	3.8	4.3	3.3	3.1	3.9	4.3	5.2	4.9	4.6	4.5
12	1.7	2.1	2.6	2.9	1.6	1.9	2.6	2.9	18.5	16.5	15.4	18.1
13	3.3	3.6	4.5	4.6	3.4	3.7	4.5	4.6	6.1	5.1	4.0	4.0
14	2.5	2.9	3.9	NA	2.5	2.8	3.8	NA	32	17.3	15	NA
15	1.9	1.8	2.7	NA	1.7	1.6	2.7	NA	32	39	27.6	NA

Pre indicates pretreatment; Post, posttreatment; and NA, not available.

All 15 subjects had reached the fourth week of posttreatment. ANOVAs indicated that there was a significant difference in both AOU and QOM scores between first contact and post-4 [$F_{\text{AOU}}(4,56)=88.49$, $P<0.0001$, $\epsilon=.568$; $F_{\text{QOM}}(4,56)=82.35$, $P<0.0001$, $\epsilon=.597$]. However, again, AOU and QOM scores did not change significantly between first contact and baseline and between baseline and pretreatment.

Paired t tests were carried out for the 11 significant others for whom complete data were available. They confirmed a treatment effect from pretreatment to posttreatment for both AOU [$t_{\text{AOU}}(10)=-5.64$, $P<0.0002$] and QOM [$t_{\text{QOM}}(10)=-7.69$, $P<0.0001$]. The scores for the significant others are presented in Figure 1 as unfilled bars at pretreatment, posttreatment, and follow-up, the times at which these data were collected. The closeness of the agreement between the data of the significant others and the subjects may be noted.

Wolf Motor Function Test

Individual data for the WMFT scores obtained at baseline, pretreatment, posttreatment, and follow-up are displayed in Table 3, and group data for all 3 WMFT measures are presented in Figure 2. ANOVAs on the WMFT scores for the 9 subjects for whom data were available for all observation periods indicated that there was a significant difference for FA and QOM and a trend for performance time from baseline to follow-up [$F_{\text{FA}}(3,24)=11.28$, $P<0.004$, $\epsilon=.46$; $F_{\text{QOM}}(3,24)=25.00$, $P<0.0001$, $\epsilon=.541$; $F_{\text{time}}(3,24)=3.29$, $P=0.095$, $\epsilon=.409$]. Additional separate analyses of contrasts showed that the overall improvement in motor function was due to the change in scores from pretreatment to posttreatment [$F_{\text{FA}}(1,8)=10.00$, $P<0.02$; $F_{\text{QOM}}(1,8)=22.84$, $P<0.0015$; $F_{\text{time}}(1,1)=4.54$, $P=0.076$]. No change was observed from baseline to pretreatment or from posttreatment to follow-up for any of the 3 measures.

ANOVAs for each of the 3 WMFT measures for the 13 patients for whom data were available from baseline, pretreatment, and posttreatment indicated a significant overall

effect for these time points [$F_{\text{FA}}(2,24)=41.63$, $P<0.0001$, $\epsilon=.95$; $F_{\text{QOM}}(2,24)=29.46$, $P<0.0001$, $\epsilon=.87$; $F_{\text{time}}(2,24)=6.24$, $P<0.009$, $\epsilon=.91$]. Separate analyses of contrasts showed that each measure improved significantly from pretreatment to post-4 [$F_{\text{FA}}(1,12)=74.89$, $P<0.0001$; $F_{\text{QOM}}(1,12)=36.89$, $P<0.0001$; $F_{\text{time}}(1,12)=7.58$, $P<0.013$], but the differences in scores between baseline and pretreatment were not significant.

Other Parameters: Chronicity, Amount of Prior Treatment, Compliance

Separate ANCOVAs using the variables chronicity and amount of prior treatment as covariates failed to show a significant impact of either variable on any of the outcome scores for any period of observation. In addition, there were no significant correlations between either of these variables and any of the outcome measures. It is of interest that the 2 subjects who had suffered a stroke less than 1 year before to project intake (6 months in both cases) did approximately as well as the more-chronic subjects. For example, on MAL-

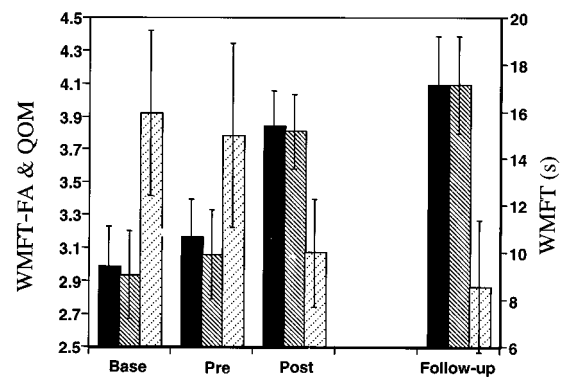


Figure 2. WMFT scores for FA (solid bars), QOM (hatched bars), and performance time (dotted bars) at baseline, pretreatment, posttreatment, and follow-up (month 6). The scale on the left ordinate refers to the FA and QOM scales and that on the right ordinate refers to performance time (in seconds).

TABLE 4. Effect Sizes for QOM and AOU Scores on the MAL and for Performance Time, AOU, and FA on the WMFT at Posttreatment, Post-4, and Follow-Up, all Compared With the Scores at First Contact

	Post vs FC	Post-4 vs FC	FU vs FC	Mean
MAL				
QOM	1.33	1.70	2.14	1.72
AOU	2.07	2.98	2.68	2.58
Mean				2.15
	Post vs Baseline	FU vs Baseline		
WMFT				
Time	0.72	0.82		0.77
FA	1.06	1.25		1.16
AOU	1.08	1.18		1.13
Mean				1.02
Study-wide effect size				1.59

Data are available for 15 subjects at first contact, posttreatment, and post-4 and for 12 subjects at follow-up. Post indicates posttreatment; FC, first contact; and FU, follow-up.

QOM, 1 of these individuals tied for the top score while the other subject scored at approximately the group mean. Both subjects scored below the group mean on the MAL-AOU measure, but there were 3 more-chronic subjects who scored lower.

Effect Size

Table 4 contains the effect sizes for the MAL and WMFT at different time points. The effect sizes for the MAL measures are extremely large, ranging from 1.33 to 2.98 at different time points, with a mean of 2.15. The effect sizes for the WMFT measures are also substantial; however, they are smaller than those for the MAL scores, ranging from 0.72 to 1.25, with a mean of 1.02. The study-wide effect size was 1.59.

Discussion

The results indicate that CI therapy is a powerful treatment for improving the rehabilitation of movement of the affected upper extremity in chronic stroke patients. The mean effect size for the 2 MAL measures was 2.15, while the mean effect size for the 3 WMFT measures was 1.02. In the meta-analysis literature, effect sizes of 0.2 are considered small, effect sizes of 0.4 to 0.6 are deemed moderate, and effect sizes of ≥ 0.8 are judged to be large.²³ Thus, the magnitude of the effect sizes here must be considered extremely large by the standards of the field. For example, the mean MAL-AOU scores went from 1.7 at first contact to 3.7 at post-4. A score of 1.7 lies between "very rarely" (a score of 1 indicates virtually not used) and "rarely" (2, used the affected extremity), while a score of 3.7 is two thirds to three quarters of the way to "nearly normal" AOU. These are group values; 6 of the 15 subjects scored above 4 ("nearly normal" AOU) at post-4. These data replicate in almost all respects the data obtained previously with CI therapy.^{3,5} The effect sizes for the different parameters are very similar. Thus, CI therapy may be seen

to produce similar therapeutic gains in 2 different laboratories located in countries with different healthcare systems.

Two aspects of the data from different periods of the experiment time line should be noted. The MAL and WMFT values did not differ significantly between baseline and pretreatment. These time points were separated by 2 weeks in which no therapy was administered. It is the same interval separating pretreatment and posttreatment testing, between which large differences were observed. The baseline testing thus serves as a control procedure indicating that the simple lapse of time, test practice effects, and expectancy of treatment do not alter test scores and therefore cannot account for the experimental results. These data (baseline versus pretreatment) also indicate that the MAL and WMFT have a good intertest reliability. There was also no diminution in test scores between posttreatment, post-4, and follow-up. This supports previous findings that CI therapy produces a long-term improvement in motor function.⁵

It might intuitively seem that side of hemiparesis could be an important factor in determining the effect of the therapy, with subjects having greater motivation to regain use of a premorbid dominant upper extremity than a premorbid nondominant extremity. This, however, did not prove to be the case. All subjects were right-arm dominant before stroke, and subjects with paresis of the left, nondominant limb exhibited as large a treatment effect as subjects with right hemiparesis. This result suggests that the motivation to use a nondominant limb is sufficiently great to yield a full treatment outcome. The effective factor in producing a rehabilitative improvement would seem to be simply the AOU that the affected extremity engages in during the intervention period.

It was not anticipated that amount of prior conventional therapy would have an impact on the effect of CI therapy.⁶ This expectation was borne out. In addition, the time since stroke was found to make no difference in the motor improvement produced by CI therapy for the patient population defined by the inclusion criteria of this study. Two aspects of this lack of relationship are particularly noteworthy, relating to the 2 ends of the dimension of chronicity studied in the present work. First, mean chronicity was 5.1 years, with 4 subjects being at 9 or more years after the event. The traditional view in the rehabilitation field, supported by numerous studies, is that patients reach a plateau in their motor recovery at 6 months to 1 year after stroke from which there will be little or no further improvement for the rest of their lives.²⁴ The subjects in this study, however, all showed a very substantial improvement in motor function compared with the motor plateau that each of them had presumably reached before the beginning of CI therapy. Moreover, the 4 subjects with the longest postevent times (9, 9, 14, and 17 years) exhibited a mean improvement on both the AOU and QOM scales of the MAL that was greater than the mean score for the group. Thus, even very chronic stroke survivors are amenable to CI therapy and do as well as individuals who are much closer in time to the focal event. Chronic stroke patients are rarely given therapy to improve motor function because the evidence to date indicates that it is of little value.⁶ However, the present data demonstrate that CI therapy can

benefit very chronic stroke survivors who have had a stroke as many as 17 years earlier.

At the other end of the chronicity spectrum, it was found that the 2 subacute patients who suffered a stroke just 6 months before the initiation of CI therapy received as much benefit from the therapy as more-chronic patients. In the past, the effect of CI therapy has been studied with patients who are >1 year poststroke. However, the present results strongly suggest that CI therapy is also effective for subacute patients. This has practical importance, because subacute patients are much more likely to already be in the treatment system than more-chronic patients and are therefore more accessible to clinicians; moreover, they would often still be eligible for reimbursement for therapy and for the provision of services by medical treatment payers in the United States.

Finally, it should be noted that within the range of patients accepted for treatment in this study (ie, those who met or exceeded the minimum motor criterion but did not exceed a score of 3.0 on the QOM scale of the MAL), initial level of motor ability did not correlate with treatment outcome. The 2 subjects at project intake who scored ≤ 80 on the Barthel ADL Scale showed treatment gains on the AOU and QOM scales of the MAL that were slightly greater than the group means, as did the 4 subjects whose median pretreatment performance time on the WMFT was greatest (≥ 10 seconds); for the 3 subjects who scored ≤ 35 on the Copenhagen Stroke Scale, the mean improvement was either as great (AOU) or a little greater (QOM) than the mean group improvement. Thus, for the subgroup of stroke survivors defined by the inclusion criteria of this study, neither level of initial motor ability, amount of chronicity, amount of prior therapy, nor side of hemiparesis affected the very substantial amount of improvement in motor ability produced by CI therapy.

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Effects of Constraint-Induced Movement Therapy on Patients With Chronic Motor Deficits After Stroke: A Replication

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