A Randomized Trial Assessing the Effects of 4 Weeks of Daily Stretching on Ankle Mobility in Patients With Spinal Cord Injuries

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ABSTRACT. Harvey LA, Batty J, Crosbie J, Poulter S, Herbert RD. A randomized trial assessing the effects of 4 weeks of daily stretching on ankle mobility in patients with spinal cord injuries. Arch Phys Med Rehabil 2000;81:1340-7.

Objective: To determine the effect of 4 weeks of 30 minutes of daily stretching on ankle mobility in patients with recent spinal cord injuries (SCIs).

Design: Assessor-blinded randomized controlled trial.

Setting: Two spinal injury units in Sydney, Australia.

Patients: Consecutive sample of 14 recently injured patients with paraplegia and quadriplegia.

Intervention: Treated ankles were stretched continuously into dorsiflexion with a torque of 7.5N·m for 30 minutes each weekday for 4 weeks. Contralateral ankles received no stretches.

Main Outcome Measures: Passive torque-angle curves for both ankles were obtained at study commencement, then at weeks 2, 4, and 5 (ie, during, at the end of, and 1 week after the stretching program). Torque-angle measurements were obtained with the knee extended and flexed. Mean values for parameters (baseline angle, angle at 10N·m, slope) describing the characteristics of the torque-angle curves were derived for each knee position. Changes from pretest to each subsequent test were calculated, as well as 95% confidence intervals (CIs) for differences in these changes between stretched and controlled ankles.

Results: The stretching intervention did not significantly change any of the 3 parameters describing the torque-angle curves of the ankle in either knee position. At the beginning of the study, the mean (±SD) angles obtained with the application of a standardized torque with the knee extended for the control and stretch ankles were 105° (± 10.4°) and 106° (± 9.8°), respectively. After 4 weeks, these values were 106° (± 10.6°) and 107° (± 10.6°) (mean difference in change of angle = 0°; 95% CI, −3.3° to 3.3°).

Conclusion: Thirty minutes of daily stretching for 4 weeks does not significantly change ankle mobility in recently injured patients with SCIs.

Key words: Ankle; Flexibility; Muscles; Rehabilitation; Spinal cord injuries; Tetraplegia.

CONTRACTURES ARE A COMMON complication of spinal cord injury (SCI).[1,2] Contractures prevent some patients from attaining an optimal level of independence,[3] and can result in unsightly deformities. In addition, contractures may increase spasticity and predispose patients to pressure sores and pain.[1,2,4-6] Consequently, much attention is directed toward treating and preventing contractures in patients with SCIs.

Contracture is characterized by an increase in joint resistance to passive rotation. This may be caused by either an increase in the involuntary muscle activity of the muscles spanning a joint,[7-11] or by a decrease in the length or compliance of surrounding muscles and soft tissue structures.[10,12-16] For this reason, contractures are said to be either neurally or nonneurally mediated.[7]

In patients with an SCI with upper motoneuron (UMN) lesions, contractures are likely to be caused by both neurally mediated and nonneurally mediated mechanisms, though little attention has been paid to distinguishing between them.[17] Contractures in patients with SCIs with lower motoneuron (LMN) lesions are solely caused by non-neurally mediated mechanisms because these patients' muscles are denervated and incapable of involuntary contraction.

Animal studies suggest that the nonneurally mediated component of contracture is caused by changes in the morphology of muscles and connective tissues precipitated by abnormal mechanical stresses.[12,18,19] For example, muscles immobilized in a shortened position for prolonged periods of time are deprived of stress and become less extensible, probably as a result of a serial loss of sarcomeres and morphologic changes in connective tissue.[14,20-22] Interventions aimed at preventing and reversing the nonneural component of contractures must provide a mechanical stimulus sufficient to induce normal tissue growth and remodeling.[19]

Stretching has been advocated widely as an effective means of treating and preventing contracture.[23-25] Ample evidence from animal studies suggests that prolonged and uninterrupted stretching (ie, 24 hours daily for a week or more) will bring about long-term changes in the nonneural component of a contracture (ie, sustained stretching will induce lasting changes in the passive properties of muscles and other soft tissue structures affected by prolonged immobilization in a shortened position).[20,26] One well-designed randomized clinical trial found that 1 week of sustained stretching with serial casting increased passive ankle mobility in patients with head injuries.[27]

Prolonged uninterrupted stretching with serial casting is a time-consuming and expensive form of therapy and may be contraindicated in patients with SCIs because of their susceptibility to pressure sores. Partly for this reason, therapists have

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sought to treat and prevent the nonneural component of contractures with shorter periods of daily stretching. Commonly, stretches are provided manually by therapists for 1 to 5 minutes each day, or by some mechanical device for longer periods, such as 20 to 30 minutes.[17] The results of a small number of animal studies suggest that short periods of daily stretching may be an effective method of treating and preventing contracture.[28-30] Williams[31] showed that as little as 15 minutes of daily stretching prevented adaptive shortening in mouse soleus muscles immobilized for short lengths of time. Few high-quality, randomized clinical trials have examined the effect of intermittent stretching in humans. One study[23] found that 1 hour of sustained low-load stretching applied daily for 4 weeks was more effective in reducing chronic knee flexion contractures in bed-ridden nursing home patients than 30 minutes of daily high-load stretch and passive movements. Neither therapists or assessors were blinded in this study. The long-term effects of prolonged or intermittent stretching on the neurally mediated component of contractures is less clear, though many clinicians believe that spasticity is reduced with these types of interventions.

The primary aim of this study was to determine the effects of 4 weeks of 30-minute daily stretching on ankle mobility in patients with a recent SCI. Overall resistance of the ankle to passive rotation (ie, the resistance due to both neural and nonneural factors) was measured, rather than solely the resistance offered by nonneural mechanisms, because overall resistance is most amenable to measurement and is of prime clinical importance. Because stretching is used routinely both to prevent the development of contracture and to reverse existing contracture in patients with an SCI with LMN and UMN lesions, we studied patients with an SCI with and without contracture regardless of the status of their LMNs. A secondary aim was to determine whether ankle stretching primarily affects the 1- or 2-joint structures spanning the ankle. This was achieved by testing the ankle with the knee extended and flexed.[32]

METHODS

Patients

Patients from 2 SCI units in Sydney were asked to participate in the study. To be included, patients had to be participating in a rehabilitation program, have sustained an SCI within the preceding year, have no more than flickers of activity in muscles around both ankles (ie, not more than grade 1 of 5 motor strength), and be willing to cease assisted-standing and all passive exercises and stretches to their ankles for the duration of the study. Patients were not considered if they had pressure sores on their heels that prevented stretching or testing, or if they were considered unlikely to cooperate.

Fourteen patients with paraplegia ($n = 4$) or quadriplegia ($n = 10$) participated. Power calculation indicated that this sample size would provide a 90% probability of detecting a 5° effect, assuming the standard deviation (SD) of the effect of the treatment was 5° and alpha was .05. A consecutive sample of 12 patients was recruited from 1 of the 2 spinal injury units. All recently injured patients admitted to this unit over a 6-month period were eligible and chose to participate. Twelve patients did not meet the inclusion criteria because of motor sparing around the ankles ($n = 10$), pressure sores ($n = 1$), or a psychiatric condition affecting the patient's ability to cooperate ($n = 1$). Another 4 patients were eligible but declined to participate. At the other spinal injury unit, only 2 patients were recruited from a larger group. The majority of patients from this unit did not participate either because they were unwilling to stop standing for the duration of the study or because they had motor sparing around their ankles.

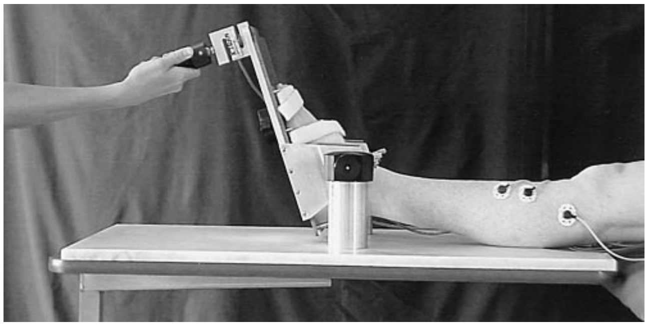

Fig 1. Device used to test the mobility of the ankle (see text for description).

The mean (± SD) age, weight, height, and time since injury were 36 years (± 16), 66kg (± 17), 176cm (± 10), and 4 months (± 2.7), respectively. All patients were men. One had a LMN lesion and the other 13 had UMN lesions with varying amounts of spasticity. Seven patients had little evidence of involuntary muscle activity (ie, spasticity) in their ankle muscles when the ankles were passively rotated. In 3 patients, involuntary muscle activity could be readily elicited with passive ankle rotation, though this muscle activity either quickly subsided or offered little resistance to passive ankle rotation. In the remaining 3 patients, involuntary muscle activity could be readily elicited with passive ankle rotation and, at times, this muscle activity offered substantial resistance to passive ankle rotation and did not readily subside.

The study received ethics approval from the appropriate institutions and informed consent was obtained from all patients.

Measurement Procedure

The overall resistance of the ankle to passive rotation was measured with a device similar to those previously described.[33-35] The properties of the device have been extensively tested.[36] It consisted of a footplate that rotated the ankle in a sagittal plane (fig 1). The axis of rotation of the footplate was aligned with the lateral malleolus of the ankle (ie, the approximate center of rotation of the ankle). The footplate had a potentiometer[a] incorporated in its axle to measure ankle angle and a force transducer[b] attached to the distal end to measure applied force. The amplified signals from the force transducer

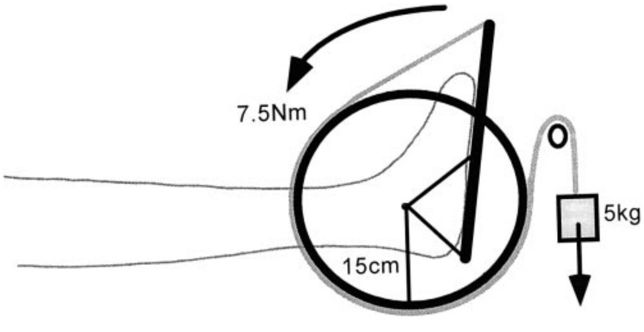

Fig 2. Schematic diagram of the device used to stretch the ankle. The device consists of a footplate attached to a wheel that rotates about the ankle in a sagittal plane. A 5-kg weight was suspended from a rope passing around the wheel rim to the end of the footplate. This ensured that a constant torque of 7.5N·m was applied, regardless of ankle angle.

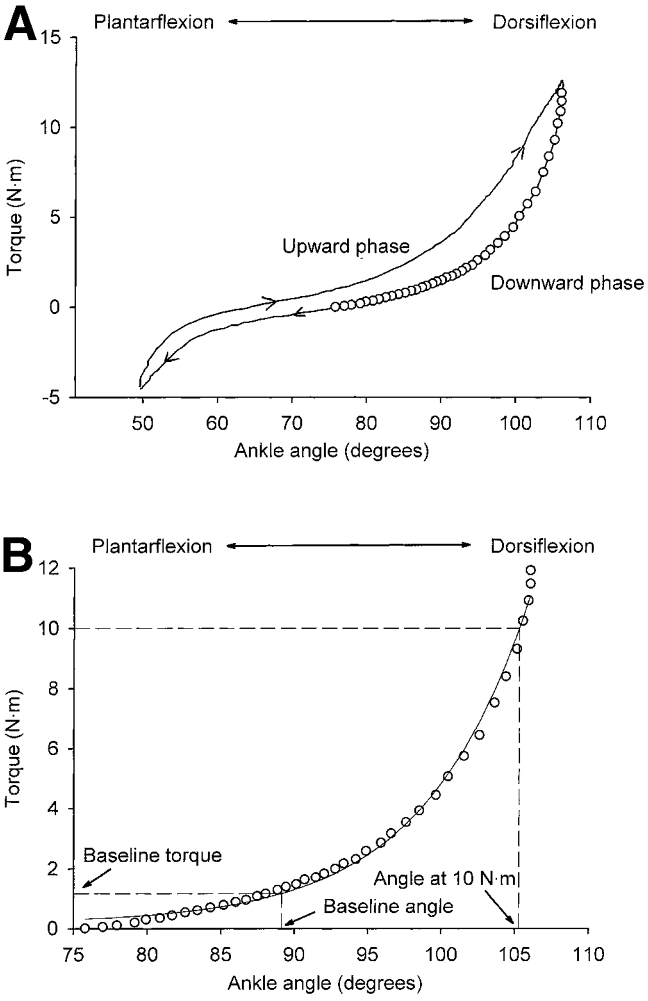

Fig 3. Ankle torque-angle properties. (A) A typical torque-angle curve. The weight of the footplate has been subtracted. The direction of the arrow indicates the application and then removal of the stretch. Open circles indicate the section of the torque-angle curves used for analysis after truncation between 0 and 12N·m. This section corresponds with the "downward" phase when the dorsiflexor torque was decreasing and the ankle was moving from a dorsiflexed to a plantarflexed position. (B) Original and fitted data obtained from the "downward" phase of a torque-angle curve. The open circles are the same data as in (A). The solid line is the fitted exponential function. The exponential function has 3 parameters that describe the characteristics of each curve (ie, baseline torque, baseline angle, slope). Baseline torque, though not used for analysis, is shown; it represents the torque from the weight of the foot. Baseline angle is the angle that corresponds with baseline torque. Slope is not shown explicitly but refers to the rate of change of torque with change in angle. An additional variable, the angle attained at 10N·m, is derived from the 3 parameters. For this ankle, baseline torque = 1.2N·m, slope = 0.14N·m/deg, baseline angle = 89° and angle at 10N·m = 105°. The plantar grade position of the ankle is represented by an ankle angle of 90°.

and potentiometer were calibrated before each testing session with a number of known forces and angles; signals were sampled at 20Hz. The measured force was multiplied by the moment arm to derive applied torque. The torque from the weight of the footplate was then subtracted. The weight torque of the footplate, which is a function of ankle angle, was determined by rotating the device and measuring the applied torque without the foot positioned in the device. The torque from the weight of the foot was ignored because the position of the foot with respect to the vertical and the weight of the foot rendered its torque negligible.

Throughout testing, electromyographic activity from the tibialis anterior and the lateral head of the gastrocnemius muscles were recorded. Adhesive silver chloride electromyographic electrodes with 11-mm discs were placed as recommended by Basmajian and Blumenstein[37] in a bipolar arrangement over each of the 2 muscles (20–30mm apart). Electromyographic signals were initially amplified by isolated preamplifiers before further amplification.[c] At this point both sets of data were band filtered between 50Hz and 500Hz using a digital eighth-order Butterworth filter and sampled by a 16-bit analog-to-digital converter at 1000Hz.

The ankle resistance to passive rotation was measured with patients lying supine. The measuring device was secured to a table at the foot of the bed. Measurements were taken of the right and then left ankle with the knee extended. These measurements were repeated with the hip and knee positioned at 90° and the long axis of the tibia horizontal. The angle of the hip and knee and the inclination of the tibia were checked with a goniometer and a digital inclinometer. The ankle was slowly rotated backward and forward between a dorsiflexed and plantarflexed position with a dorsiflexor torque between 0 and 15N·m. Angular velocity was monitored in real time during the test and was kept below 5°/sec to minimize reflex contraction of the muscles around the ankle.[38] A small number of trials were discarded by the blinded assessor at the time of testing because of clonus or obvious electromyographic activity in the tibialis or gastrocnemius muscle groups. At least 6 trials were collected from each ankle from each patient for each condition (ie, with knee extended and flexed).

Experimental Protocol

On completion of all initial measurements, 1 ankle of each patient was randomly allocated to the stretch group whereas the other ankle was allocated to the control group. A computer-generated random allocation schedule was determined before the study by an investigator who was not involved in patient recruitment or group allocation. To ensure concealment, allocations were placed in sealed, opaque, sequentially numbered envelopes by an investigator who was not involved in determining eligibility for the trial. The envelopes were not opened until after the initial tests had been performed. Patients were considered to have entered the trial at this time.

Each patient's experimental ankle was stretched 5 to 7 times each week for 4 weeks (mean total number of treatments per

Table 1: Initial Values of Parameters of Stretched and Nonstretched Ankles With the Knee Extended and Flexed

	Knee Extended		Knee Flexed	
	Nonstretched	Stretched	Nonstretched	Stretched
Baseline angle (deg)	87 (±10.3)	89 (±9.9)	104 (±11.1)	104 (±10.1)
Angle at 10 N·m (deg)	105 (±10.4)	106 (±9.8)	120 (± 9.7)	121 (±10.2)
Slope* (N·m/deg)	.14 (.11 to .16)	.13 (.12 to .15)	.13 (.12 to .16)	.14 (.12 to .17)

Values given as mean ± SD.
* Slope presented as median and interquartile range.

patient ± SD = 21 ± 0.6). The control ankle received no stretches during this time; thus, this ankle acted as a control for the stretched ankle. After 4 weeks, stretches to the treated ankle were ceased, though the study continued for another week. Three patients briefly (ie, for 4–6 days) ceased stretching after 5 to 10 days because of medical complications not associated with the study. These 3 patients recommenced stretching once their medical conditions had resolved; they received the same total number of treatments, though over a slightly longer period of time.

Patients did not receive any type of manual therapy (ie, passive movements or other stretches) to either ankle for the 5 weeks of the study, nor did they stand or walk during this time. One patient used a passive leg cycle for both legs twice a week, but the leg cycle was modified to fix the ankles at 90°, preventing cyclic stretch at the ankles. All patients' feet were moved as required for activities of daily living (ADL) (ie, washing and transferring) and patients were positioned in their wheelchairs with their ankles at 90°. Some patients slept with both feet positioned at 90° ($n = 6$), but this was not possible for those who primarily slept on their sides. Most nursing staff and therapists were unaware that their patients were participating in the study and those that were aware did not know which foot was being treated.

Patients' medications were not recorded or controlled, though some patients were on antispasticity medication. The risk of these and other cointerventions biasing results is minimized by random allocation. In addition, the use of a within-patient design further minimized the effects of cointerventions that act bilaterally, such as antispasticity medication.

Patients were tested at weeks 2, 4 (ie, at the end of the stretch period), and 5 (ie, 1 week after the cessation of stretching). Measurements at weeks 2 and 4 were taken 24 hours after the last stretch, except in 1 patient in whom the measurements were taken 48 hours later. A blinded therapist was responsible for all measurements, and all measurements were taken at the same time each day (ie, immediately after breakfast, before patients sat out of bed).

The experimental ankle was placed on a constant stretch each day for 30 minutes by rotating the ankle into dorsiflexion with the knee extended. Patients were either positioned supine on their beds or seated in their wheelchairs for these stretches. A device designed specifically for the purpose was used to stretch the ankle (fig 2). The device consisted of a footplate that rotated the ankle in a sagittal plane. A rope attached to the end of the footplate was looped around the rim of a wheel of 15-cm radius and passed through a pulley. A 5-kg weight suspended vertically from the end of the rope created a torque that rotated the footplate and foot into dorsiflexion. This ensured that a constant torque of 7.5N·m was applied, regardless of ankle angle. The small torques associated with the weight of the stretching device and foot were ignored.

Data Reduction and Analysis

Torque and angle data were used to construct torque-angle curves. The first 2 stretch cycles were not analyzed.[39] The ''downward'' phase of the next 4 torque-angle curves, when the dorsiflexor torque was decreasing and the ankle was moving from a dorsiflexed to a plantarflexed position, were then truncated between 0 and 12N·m and used for further analysis (fig 3). The ''downward'' phases of the torque-angle curves were used because these parts of the curves showed less between-session variability than corresponding ''upward'' phases. The lower variability obtained from the ''downward'' phase of the torque-angle curves may have been caused by less involuntary muscle activity associated with rotation into plantarflexion than dorsiflexion,[17] though this hypothesis was not formally tested.

The following exponential function was fitted to each torque-angle curve:

$$\text{torque} = \text{baseline torque} + e^{(\text{slope} \times (\text{angle} - \text{baseline angle}))} - 1$$

where, baseline torque is the asymptotic minimal torque, slope determines the slope of the curve, and baseline angle is the angle corresponding with baseline torque.

This equation was used because (1) it provided a good fit for each curve (median $R^2 = .997$; interquartile range = .995–.999); (2), its parameters have a simple physical interpretation; and (3), it is similar to equations used by others in studies on isolated muscle.[40,41] The fitted data were used to derive another parameter, the angle of the ankle at 10N·m torque. The slope, baseline angle, and the angle at 10N·m were used for analysis. Baseline torque was not used for analysis because it primarily reflects the torque from the weight of the foot and consequently does not change over time.

Data from each patient's 4 trials attained at each testing session were pooled to obtain a median value for each patient. For each of the 3 variables (ie, slope, baseline angle, angle at 10N·m), changes from pretreatment to weeks 2, 4, and 5 measurements were calculated for the 2 knee positions. Differences in these changes between stretched and controlled ankles for each of the 2 knee positions were determined. A positive change in any of the parameters indicated an increase in the mobility of the stretched ankle compared with the controlled ankle. Paired *t* tests were used for the baseline angle and angle at 10N·m to generate 95% confidence intervals (CIs) of the differences between means.[42] The slope data were not normally distributed and could not be easily transformed, so median differences were calculated and Wilcoxon signed rank tests were used to determine *p* values. All data were analyzed by intention-to-treat analyses.[43]

Data obtained from the controlled ankle at weeks 4 and 5 were used to determine test-retest reliability. These data were used because they were least likely to be affected by real changes in ankle mobility. Most parameters were highly reproducible. For example, the intraclass correlation coefficients (ICC) of baseline angle and angle at 10N·m were .84 and .90, respectively. The corresponding value for the slope data was .31. This low value was caused by 1 highly irregular data point, and when the correlation was calculated without this value, the ICC was .81. The high ICCs indicate that, regardless of any reflex muscle activity that may have been present during testing, measurements derived from the ''downward'' phase of the torque-angle curves were reliable.

RESULTS

No patient withdrew from the study and all patients' ankles were treated according to their initial allocation. Outcome measures were obtained from all patients except in 1 patient in whom knee flexion data were not collected at week 2 because of an acute medical complication. At the commencement of the study, differences between stretched and controlled ankles for all 3 parameters were small and insignificant (table 1). Parameters varied considerably between patients, reflecting differences in initial ankle mobility. Some patients appeared to have normal or near-normal ankle mobility whereas others had obvious clinical signs of contracture.

Figure 4 shows the change in the 3 parameters of stretched and controlled ankles over the course of the study. There was little change in the mobility of either ankle over the course of the 5 weeks and little difference between them. Mean or median

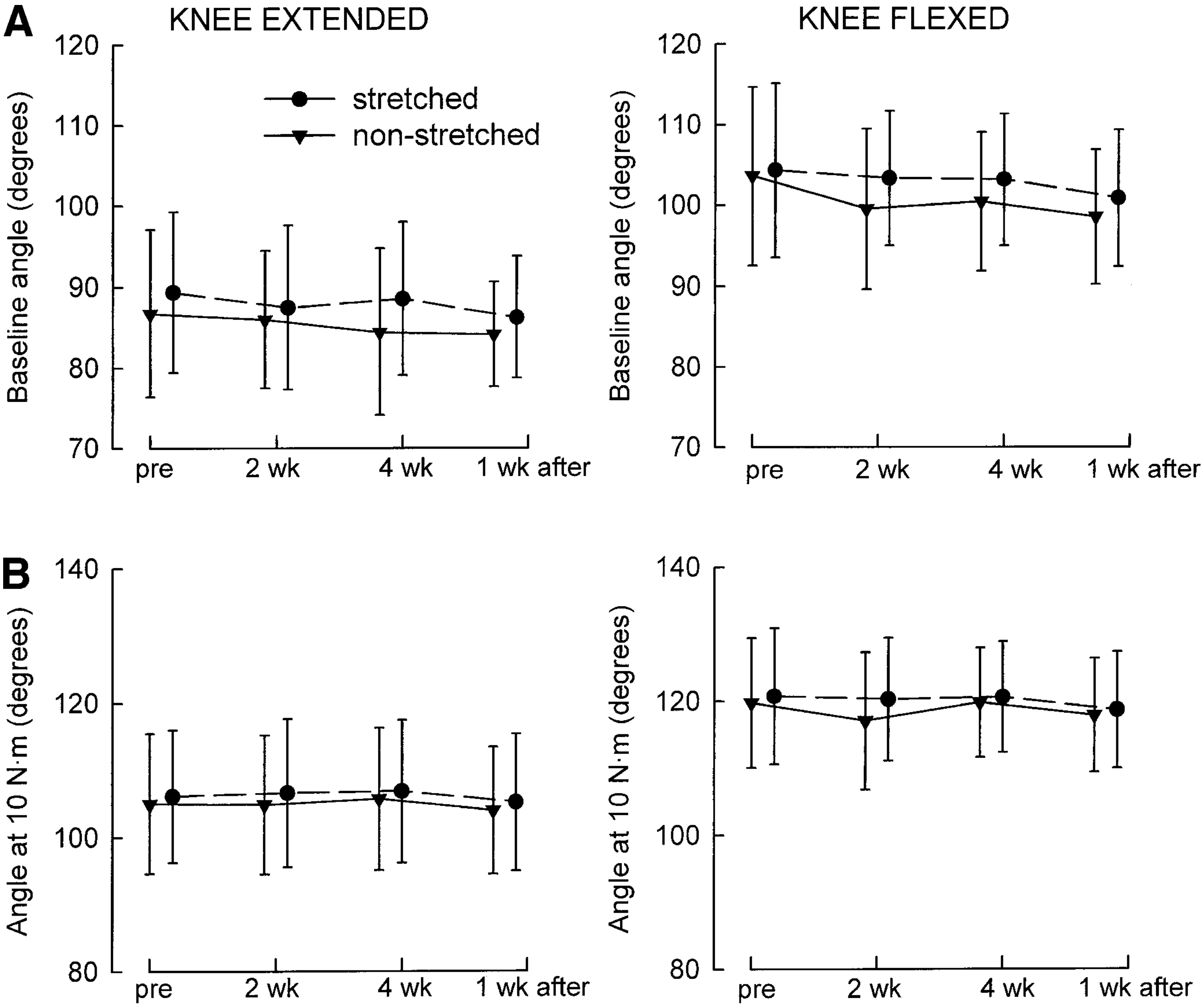

Fig 4. Effects of stretching on torque-angle properties. (A) mean (± SD) baseline angle, (B) mean (± SD) angle at 10N·m, and (C) median (interquartile range) slope at the beginning (pre), 2 weeks (2 wk), 4 weeks (4 wk; at the end of the stretch intervention) and 1 week after cessation of the stretch intervention (1 wk after). In each pair of graphs, the left graph represents the data obtained with the knee extended and the right graph represents the data obtained with the knee flexed. The closed circles and dashed lines represent the stretched ankle, and the triangles and solid lines represent the controlled ankle. Data from both ankles have been slightly offset on the *x* axis for clarity, but the measurements were taken at comparable times. The plantar grade position of the ankle is represented by an ankle angle of 90°.

differences between stretch and controlled groups and their 95% CIs are given in table 2. The results clearly indicate that stretching did not have a clinically meaningful effect on ankle mobility.

We have considered the possibility that stretching might have been more effective in those patients with preexisting ankle contracture. Fig 5 indicates the range of the treatment effect for each patient in relation to initial ankle mobility. There was no apparent relationship between these 2 variables. There was no apparent relationship between the range of the treatment effect and other variables such as age, time since injury, or level of spasticity.

DISCUSSION

The aim of this study was to determine the effectiveness of stretching that is routinely provided to patients with SCIs for the treatment and prevention of ankle contractures. To ensure that sufficient stretching was applied, stretches were conducted for 30 minutes each day, which is a session that is considerably longer than those frequently applied in many clinical settings. In addition, stretching was administered with a mechanical device capable of providing more intense stretches than a therapist can manually administer for a sustained period. Despite the application of longer and larger stretches than those routinely used in the clinical setting, the study clearly indicates that 4 weeks of stretching does not affect ankle mobility in patients with recent SCIs. Contrary to expectations, the patients with existing contractures did not lose ankle mobility with or without 4 weeks of stretching.

Not surprisingly, initial ankle mobility varied between patients. Mean angle of the ankle with the application of 10N·m of torque was 105° ± 10.4° (ie, 15° greater than plantar grade). Although comparisons with other studies[32,36,44,45] are difficult because of different methods of data analysis, it appears that the

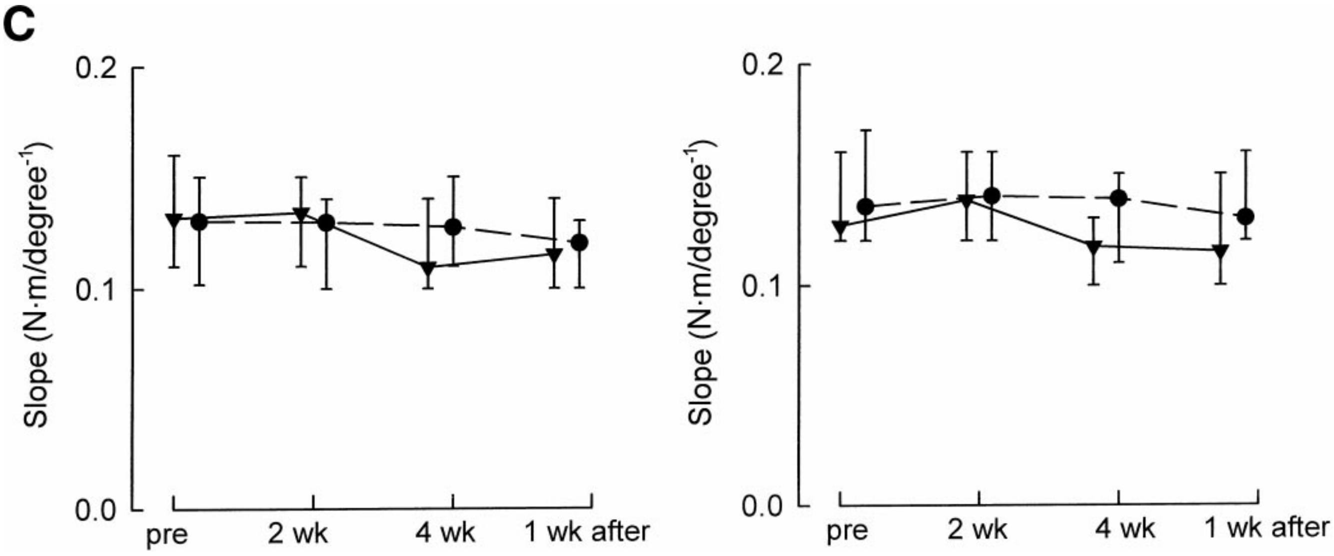

Fig 4. (*continued*)

majority of patients had stiffer ankles than healthy able-bodied individuals, and several patients (ie, those with less than 100° ankle dorsiflexion with the application of 10N·m) had clinically obvious contracture.

Stretching may not be necessary to prevent loss of ankle mobility because patients benefit from other daily activities and therapies. In particular, all patients sat in their wheelchairs with their feet supported at 90° and some slept with their feet in this position. These and other routine interventions alone may have provided adequate stimuli to muscles and soft tissue structures to prevent loss of mobility and may have rendered additional stretches redundant. However, if so, these activities were not sufficient to reverse contracture in those patient who had them before the 4-week stretch intervention.

It is unlikely that the controlled ankles received additional therapy or interventions that masked the effect of the stretch on the treated ankles. No patient had more than grade 1 of 5 strength in the muscles spanning the ankle nor did any partake in weight-bearing sports or exercises. Moreover, treating physiotherapists were blinded to group allocation, and most therapists and nursing staff were unaware that patients were participating in the study. All therapies specific to the ankle were withdrawn and any other factors likely to change the stiffness of the ankle, such as medication, practice of ADL, or presence of edema were unlikely to have unilateral effects.

Patients with UMN and LMN lesions were included in this study because these types of patients routinely receive ankle stretches and because there is little quality evidence to indicate that these 2 groups of patients respond differently to stretching. In those patients with UMN lesions (ie, all but 1), ankle mobility was measured without distinguishing between the resistance from neural and nonneural factors. Therefore, it is possible that beneficial effects of stretching on the passive mechanical properties of muscles and other soft tissue were obscured by concurrent increases in spasticity. However, this seems unlikely for 2 reasons. First, more than half the patients had either minimal or no spasticity in the muscles spanning the ankle. Second, our measurement and analysis method minimized the influence of spasticity. At the time of testing electromyography was monitored and the ankle was rotated slowly to minimize reflex muscle contractions. In addition, only the "downward" part of the torque-angle curves was used for

Table 2: Effect of Stretching on Parameters of Torque-Angle Curves Obtained With the Knee Extended and Flexed

	2 Weeks	4 Weeks	5 Weeks
Knee Extended			
Baseline angle (deg)	−1 (−5.4 to 3.1)	2 (−2.7 to 5.7)	−1 (−4.7 to 3.7)
	$p = .57$	$p = .45$	$p = .80$
Angle at 10N·m (deg)	1 (−2.5 to 3.7)	0 (−3.3 to 3.3)	0 (−3.0 to 3.1)
	$p = .68$	$p = .99$	$p = .95$
Slope (N·m/deg)	−.01	.01	.00
	$p = .31$	$p = .5$	$p = .24$
Knee Flexed			
Baseline angle (deg)	2 (−1.2 to 5.2)	2 (0 to 4.4)	1 (−2.3 to 5.1)
	$p = .20$	$p = .05$	$p = .43$
Angle at 10N·m (deg)	2 (−0.7 to 4.8)	0 (−2.7 to 2.4)	0 (−3.2 to 2.4)
	$p = .13$	$p = .92$	$p = .77$
Slope (N·m/deg)	.00	.01	.01
	$p = .85$	$p = .17$	$p = .19$

Data are differences between stretched and controlled ankles of change from baseline to weeks 2, 4, and 5 (week 1 after the cessation of the stretching intervention). Means and 95% CI are given for baseline angle and angle at 10N·m. Median difference in slope is provided without CI. (A positive value indicates an increase in ankle mobility of the stretched ankle with respect to the controlled ankle.)

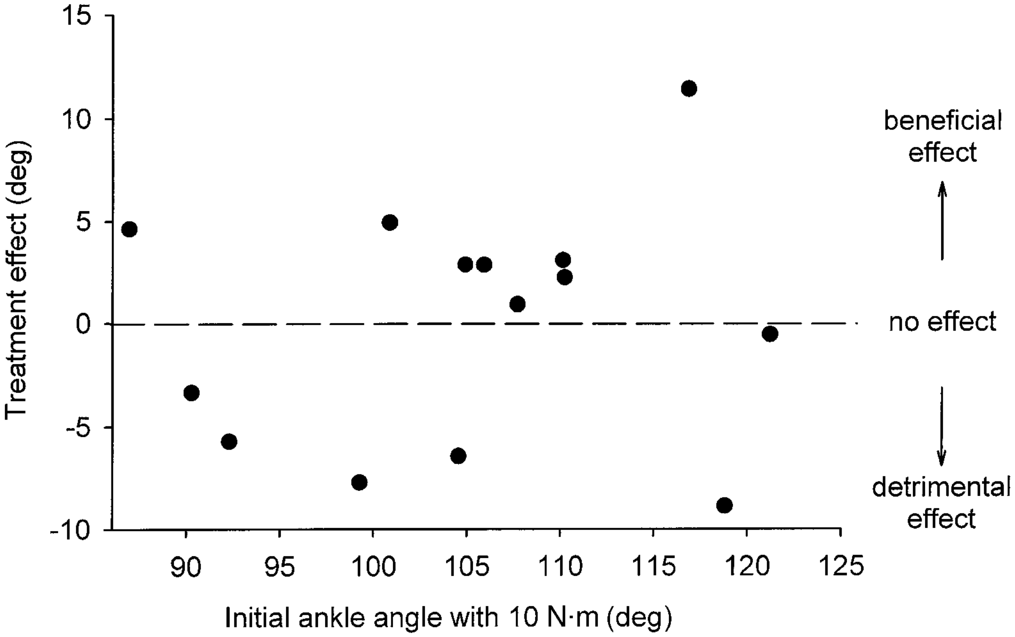

Fig 5. The size of the treatment effect as a function of initial mobility of the ankle. The size of the treatment effect (vertical axis) for each patient is represented by the difference between stretched and controlled ankle of change in angle at 10N·m from pretreatment to the end of the stretching intervention (ie, 4 weeks). Initial ankle mobility is the angle at 10N·m of the controlled ankle at the commencement of the study (horizontal axis). Each data point represents 1 patient. All measurements are with the knee extended.

analysis. This phase of the curve corresponds with the release rather than the application of stretch to the plantarflexor muscles, a period when reflex muscle activity is least likely to be elicited in the plantarflexor muscles. The high reliability of measurements taken from the controlled ankle 1 week apart also indicates that the method of data collection we used was unlikely to have been distorted by reflex muscle activity. Even if the measurements did reflect reflex muscle activity to some degree, the results indicate that overall resistance to passive rotation (ie, the resistance caused by neural and nonneural factors) is not affected by stretch. The beneficial effects of stretching on the passive mechanical properties of muscles and other soft-tissue structures are of little clinical relevance if they are not accompanied by an overall decrease in the resistance of the ankle to passive rotation.

Our secondary aim was to determine whether a 4-week program of stretching primarily affected the 1- or 2-joint structures spanning the ankle. Therefore, the ankle was tested with the knee flexed and extended. The gastrocnemius muscle in particular is stretched less with the knee flexed. For this reason, tension in this muscle will restrict overall ankle mobility more when the knee is extended as opposed to flexed. The results of this aspect of the study indicate that 4 weeks of stretching does not affect the 1- or 2-joint structures of the ankle. In addition, the results provide some information about the contribution of the gastrocnemius muscle to ankle mobility in patients with SCIs.[32] They indicate that 15° more dorsiflexion is obtained with a 10N·m torque when the ankle is tested with the knee flexed rather than extended. Similar findings have been reported in a study of able-bodied healthy patients.[32]

CONCLUSION

The results of this study indicate that patients with recent SCIs do not benefit from 4 weeks of 30-minute daily stretches. Those with poor initial ankle mobility did not respond more readily to regular stretching than others. Although our results do not suggest that patient subgroups will benefit from stretching interventions of this kind (fig 5), further studies are needed to clarify this issue. For example, it may be interesting to determine if the degree of spasticity influences the effect of stretching. Further research also is needed to ascertain whether stretching is effective if administered daily over longer time periods (ie, months and years rather than weeks), or with greater torques, or for longer periods each day (ie, for several hours rather than 30 minutes). Until further studies are performed, therapists cannot be confident that stretching is effective for the treatment and prevention of contracture in patients with SCIs.

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