Facilitation of triceps brachii muscle contraction by tendon vibration after chronic cervical spinal cord injury

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Ribot-Ciscar, Edith, Jane E. Butler, and Christine K. Thomas. Facilitation of triceps brachii muscle contraction by tendon vibration after chronic cervical spinal cord injury. J Appl Physiol 94: 2358–2367, 2003. First published February 14, 2003; 10.1152/japplphysiol.00894.2002.—One way to improve the weak triceps brachii voluntary forces of people with chronic cervical spinal cord injury may be to excite the paralyzed or submaximally activated fraction of muscle. Here we examined whether elbow extensor force was enhanced by vibration (80 Hz) of the triceps or biceps brachii tendons at rest and during maximum isometric voluntary contractions (MVCs) of the elbow extendors performed by spinal cord-injured subjects. The mean ± SE elbow extensor MVC force was 22 ± 17.5 N (range: 0–23% control force, n = 11 muscles). Supramaximal radial nerve stimuli delivered during elbow extensor MVCs evoked force in six muscles that could be stimulated selectively, suggesting potential for force improvement. Biceps vibration at rest always evoked a tonic vibration reflex in biceps, but extension force did not improve with biceps vibration during triceps MVCs. Triceps vibration induced a tonic vibration reflex at rest in one-half of the triceps muscles tested. Elbow extensor MVC force (when >1% of control force) was enhanced by vibration of the triceps tendon in one-half of the muscles. Thus triceps, but not biceps, brachii tendon vibration increases the contraction strength of some partially paralyzed triceps brachii muscles.

MANY PEOPLE WITH CERVICAL spinal cord injury (SCI) have weak triceps brachii muscles (31, 33). This weakness limits their ability to push a wheelchair, to transfer from one place to another, and to reach objects in the environment. The various factors that contribute to this weakness of triceps brachii during voluntary contractions may include disruption of descending inputs from higher centers to the spinal cord, death of motoneurons near the injury site, poor central drive, co-activation of biceps and triceps brachii, and disuse atrophy (30, 31, 33). It is also typical for triceps brachii selectively. The associated partial deactivation and atrophy of triceps brachii often mean that the subject cannot drive voluntarily. However, after chronic cervical SCI, it is not always easy to stimulate triceps brachii selectively. The associated partial denervation and atrophy of triceps brachii may result from an illusory sensation of forearm extension due to vibration of the biceps brachii tendon.

Earlier studies in people with SCI have shown that tendon vibration generally induces either no TVR or a weak response in the parent muscle (2, 8, 9, 13, 16). The AVR was not explored. Sometimes only electro-
myographic (EMG) signals and/or joint angles were recorded. When both the agonist and antagonist muscles were excited simultaneously by vibration, it was unclear how contraction strength was changed. Other studies have focused only on leg muscles or have provided case reports.

Given the evidence that the maximal triceps brachii voluntary force-generating capacity is significantly less in subjects with chronic cervical SCI than in controls (mean ± SD: 18 ± 22% of control force), but the force-generating capacity of the entire triceps brachii muscle was predicted to be greater (i.e., the strength of the voluntary contraction; the fraction activated poorly by voluntary effort and/or the paralyzed muscle fraction was 30 ± 26% of control force; Ref. 33), we hypothesized that the maximal voluntary elbow extensor force of individuals with chronic cervical SCI could be increased by exciting the triceps brachii motor pool with tendon vibration. Thus the primary aim of the present study was to determine whether vibration of either the triceps or biceps brachii tendons during a maximum voluntary contraction (MVC) of triceps brachii was a way to enhance the elbow extension force. To address this aim, it was necessary to answer two preliminary questions. First, we analyzed whether there was the potential to improve the elbow extensor force in the present subject population with chronic SCI (>1 yr). The maximal voluntary force of each triceps brachii muscle tested was compared with the predicted force-generating capacity of the entire muscle by using the twitch interpolation technique (1, 17, 23, 33). Second, for tendon vibration to improve maximum voluntary elbow extensor force, it was necessary that the vibration-induced inputs excited the triceps brachii motoneurons. The effectiveness of the vibration was tested by analysis of whether a tonic vibration reflex and/or AVR was induced in each of the triceps brachii muscles at rest. If a reflex response was induced by vibration before or during the voluntary contractions, vibration may facilitate the initiation, maintenance, and/or strength of the voluntary contraction.

MATERIALS AND METHODS

Subjects. Experiments were performed on eight volunteers (mean ± SE age: 40 ± 4 yr) who had sustained a SCI at C7 or higher, as assessed by the criteria of the American Spinal Injury Association (21). Table 1 gives some characteristics of each person, in particular his or her injury level and American Spinal Injury Association classification, the time since injury, and the cause of injury. These subjects were chosen because they had weak or completely paralyzed triceps brachii muscles when evaluated by a manual examination. Only one arm was studied in five people due to time constraints. Both arms (usually paralyzed to different extents) were studied in the other three subjects (n = 11 muscles in total). All but one subject (subject 8, Table 1) had normal biceps function in the arm tested, as assessed by manual muscle examination (i.e., a score of 5). All subjects gave their informed, written consent to participate in this study, which was approved by the University of Miami Institutional Review Board and conducted according to the Declaration of Helsinki.

Experimental setup. Each subject sat in his or her wheelchair. The arm rest of the wheelchair was removed on the test side. The test arm lay palm up on an inclined support, such that the elbow was flexed to ~90°, an optimal angle for generating isometric force in triceps brachii (Del Valle A, Bigland-Ritchie B, and Thomas CK, unpublished observations) (Fig. 1A). The subject’s forearm and hand were held against the support with Velcro. The test shoulder was always strapped with a car seatbelt attached to the subject’s wheelchair at a point just behind the shoulder of the contracting arm. The strap was bound firmly across the chest to a point on the other side of the wheelchair at waist level. This setup gave stability to the trunk during the contractions and immobilized the test shoulder. Thus most of the force that we measured was attributed to elbow extensor muscles (triceps brachii, anconeus). A transducer (FT10, Astro-Med, West Warwick, RI) was used to record the forces applied in both the extension and flexion directions. The transducer lay beneath the incline of the support, just proximal to the wrist, as during the previous experiments involving able-bodied control subjects (31, 33).

Pairs of electrodes were used to record surface EMG from triceps and biceps brachii. One pair was placed over the lateral head of the triceps brachii muscle, ~3 cm apart. The other pair was positioned near the middle of the biceps brachii muscle belly. A ground electrode was strapped on the upper arm.

A bipolar electrode was strapped over the radial nerve at the point at which single electrical stimuli (50-μs duration; model DS7H stimulator, Digitimer, Hertfordshire, UK) evoked the largest triceps brachii muscle compound action potential (M wave). The intensity of the stimuli was gradually increased up to the level that evoked a maximum triceps

Table 1. Participant history

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Gender</th>
<th>Age, yr</th>
<th>Arm Studied</th>
<th>Time Since Injury</th>
<th>Level of Injury</th>
<th>ASIA Classification</th>
<th>Cause of Injury</th>
<th>Medication for Spasticity</th>
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<td>51</td>
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<td>A</td>
<td>Diving</td>
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<td>L/R</td>
<td>17 yr</td>
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<td>M</td>
<td>50</td>
<td>R</td>
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<td>L</td>
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<td>M</td>
<td>31</td>
<td>R</td>
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<td>12 yr</td>
<td>C4</td>
<td>A</td>
<td>Gunshot</td>
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</table>

*ASIA, American Spinal Injury Association; F, female; M, male; L, left; R, right; C, cervical.
brachii M wave. Stimuli that were 25% above maximal M-wave intensity were used during the test trials.

The vibrator (Zeniter model TMT-18, Heiwa Electronic Industrial) was positioned on either the triceps or the biceps brachii tendons near the elbow by using an elastic Velcro strap. The firmness of the strap was adjusted for each subject so as to maintain the vibrator in place throughout the experiment without reducing the amplitude of the vibration.

Procedure. Before the recorded trials, the vibrator was manually applied to the biceps brachii tendon of the test arm, with the arm extended slightly and supported by an experimenter. This permitted each subject to become familiar with tendon vibration and the associated sensation of movement illusions. Then, with the test arm placed in the support, the subject was asked to perform a series of MVCs of the elbow extensors. Visual feedback of the force was provided on an oscilloscope, and verbal encouragement was given to help the subject maintain a maximal force for 6 s. When necessary, auditory feedback of triceps brachii EMG was also provided to facilitate muscle relaxation before the vibration or the voluntary contraction. Auditory feedback was turned off during the test MVCs and vibration.

The test protocol consisted of five different series presented in random order (see Fig. 1B). Each series was composed of five trials. There was a 2-min rest between trials. In one series, the subject was asked to perform a MVC of the elbow extensor muscles for 6 s, with continuous visual feedback of the force and verbal encouragement from the experimenters. Two different auditory cues from the computer were used to signal the beginning and the end of each contraction. To assess the extent to which triceps brachii was under voluntary control, four single supramaximal stimuli were delivered to the radial nerve (2 and 4 s after the beginning of the contraction and 2 and 4 s after the contraction).

In two other series ("vibration alone"), vibration was applied perpendicularly to the tendons of either the triceps or the biceps brachii muscles for 9 s. Vibration was delivered at 80 Hz, because this frequency is known to activate muscle spindle primary endings preferentially (27). During the vibration, the subjects were asked to relax as much as possible.
They were instructed to watch the vibrated arm during triceps vibration so as to facilitate the TVR. During biceps vibration, they were asked to close their eyes to preferentially evoke an AVR. At the end of each trial, the subject reported whether he or she felt any sensation of movement during the vibration.

In the other two series, the subject was asked to perform a MVC of the elbow extensor muscles for 6 s, together with vibration of either the triceps (CVT) or biceps brachii tendons (CBV). The vibration was turned on 3 s before the voluntary contraction so as to initiate a vibration reflex. Vibration was stopped at the end of the voluntary contraction. Both the onset and offset of the vibration were marked by auditory cues from the computer.

**Data recording and analysis.** Surface EMG from the triceps and biceps brachii muscles and force were amplified, filtered (30–300 Hz, direct current 100 Hz, respectively), and sampled on-line (3,200 and 400 Hz, respectively) by using an SC/Zoom data-acquisition and analysis system (Department of Physiology, University of Umeå, Sweden).

Data were analyzed off-line by using Zoom software. EMG signals were rectified and integrated between two target times (see below) and normalized for time to provide average EMG values. Force and integrated triceps and biceps brachii EMG were measured at the following target times during each trial. For the vibration-alone trials, measurements were made for two time windows (each of these and the other time windows were ~2 s) before vibration, three time windows during vibration, and two time windows after vibration. For the trials involving a MVC with or without vibration (see Fig. 3), measures were made for two time windows at rest (Pre1 and Pre2), one time window before the MVC with or without vibration (0), three time windows during the MVC (1, 2, 3), and two time windows after the MVC (Post1 and Post2).

The degree of voluntary activation of the elbow extensors was estimated by using the twitch interpolation technique (1, 17, 23). The increment in force produced when the radial nerve stimulus was superimposed on the ongoing voluntary contraction was compared with the twitch forces evoked when the muscles were stimulated at rest

Voluntary activation

\[
= \left[1 - \left(\frac{\text{Superimposed twitch}}{\text{Rest twitch}}\right)\right] \times 100\%
\]

These data were also used to infer the magnitude of the whole muscle force-generating capacity (that which can be produced by voluntary effort and by muscle that is either paralyzed or poorly activated by voluntary drive) and thus the potential for force improvement by vibration. For example, in Fig. 1C, the superimposed and rest twitches were 12.6 and 17.7 N, respectively, an indication that only 29% of the innervated muscle could be activated voluntarily. Thus 71% of this muscle was paralyzed and/or poorly activated by voluntary effort. The MVC force for this subject was 36.5 N. With 29% voluntary activation, these data suggest that 125.8 N could be produced by the entire muscle (36.5/0.29), providing the potential to produce 89.3 N by vibration or some other external means.

Both the MVC and predicted whole muscle forces were expressed relative to the average triceps brachii force produced by control women or men in (Fig. 1D) to give an indication of the extent of muscle weakness in the SCI subjects and the magnitude of the muscle atrophy. The mean maximal voluntary forces produced during elbow extension performed by control women and men using the same experimental setup were 157 and 268 N, respectively (Ref. 31; 18 subjects, 9 women, 29 ± 8 yr; 9 men, 24 ± 6 yr). Thus the MVC force for the female SCI subject shown in Fig. 1C was 23.2% of control maximal force [(36.5 N/157 N) × 100%], whereas the estimated force that could be evoked from the entire muscle was 80.1% of control maximal force [(125.8 N/157 N) × 100%], leaving the potential to produce 56.8% of control force by vibration. These data also indicate that 19.9% of control force was attributable to muscle atrophy (157 N − [(125.8 N/157 N) × 100]); see Ref. 33), a strength deficit that will not be ameliorated by vibration or other external stimuli.

Mean ± SE values are given. Data were collected from a total of 11 different arms (8 subjects). Because cervical SCI usually induces asymmetrical effects on triceps brachii, the function of one or the other triceps brachii muscles in the same subject is often very different (n = 52 cases; Ref. 25). Differential impairment by the injury was also observed in the present group of subjects, as attested by the different MVC amplitudes (see Fig. 1D, solid bars), and thus allowed us to consider data from each arm independently. Improvement in the strength of either triceps brachii muscle is also of functional importance to an individual with chronic cervical SCI.

**RESULTS**

Maximum voluntary elbow extensor force and total elbow extensor force. All of the SCI subjects had difficulty in contracting triceps brachii voluntarily. This was reflected both as muscle weakness and as an inability to contract triceps brachii selectively. Figure 3A shows an example of the force from five trials performed by one subject (subject 4). Her MVCs were weak (~12% of the average control female MVC force), reproducible (18.5 ± 2.6 N), and always involved co-contraction of biceps and triceps brachii. Figure 1D shows the mean forces developed during elbow extensor MVCs for 9 of the 11 muscles tested (solid bars; 2 muscles were totally paralyzed, subjects 7 and 8, Table 1), expressed relative to the mean force exerted by healthy women and men tested with the same apparatus (31). The mean forces developed by the SCI subjects ranged from 23 to 0.2% of control values.

Supramaximal stimulation of the radial nerve gave rise to three different responses: selective activation of triceps brachii (n = 6), contraction of triceps brachii together with other radial-innervated muscles in the forearm (n = 4), and muscle spasms that influenced the elbow extensor force (n = 1). Further stimulation was only delivered to those muscles that showed a selective triceps brachii response. In all six of these muscles, triceps force increments (mean ± SE, 5.4 ± 2.2 N) were produced by radial nerve stimulation during isometric MVCs of the elbow extensor muscles. In five cases, the paralyzed and/or submaximally activated part of the elbow extensor muscles was stronger than the fraction that was under voluntary control (Fig. 1D). The voluntary force exceeded the force from the paralyzed and/or submaximally activated fraction in another muscle. The mean forces from paralyzed and/or submaximally activated muscle ranged from 0.2 to 56.8% of control values, an indication of the extent to which tendon vibration may be able to improve the triceps brachii contraction strength.
Response to tendon vibration alone. With triceps and biceps brachii at rest, vibration was applied either to the triceps tendon while the subject looked at that arm, or to the biceps tendon while the subject had his or her eyes closed. Vibration of the triceps brachii tendon was expected to induce contraction of the triceps brachii muscle and a clear extension response, as shown for one SCI subject in Fig. 2A.

Figure 2C shows the mean (±SE) force (n = 5 trials) developed by each muscle during the whole period of vibration, with the data reported in order of the strongest triceps response (positive force) to the strongest biceps response (negative force) during triceps brachii vibration (solid bars). A triceps response was obtained in six of the muscles. Contrary to expectation, contraction of the biceps brachii muscle or a flexion response was obtained in the other five muscles during triceps vibration. A biceps brachii response with vibration of the triceps tendon was more frequent in the subjects who had greater paralysis of the elbow extensors. The mean MVC of these subjects was only 4% that of healthy subjects, whereas it was 13% for the subjects who exhibited a TVR in the triceps brachii muscle.

When the biceps brachii tendon was vibrated outside the apparatus with the arm of the subject slightly extended and supported by an experimenter, all of the subjects tested felt an illusion of movement. However, when the arm was inside the setup, only three subjects reported an illusory extension of the arm in 4 of the 15 trials (27%) that involved biceps brachii tendon vibration. This relatively infrequent sensation of movement was accompanied either by a clear EMG response in the biceps brachii muscle (62% of trials; see Fig. 2B as an example) or, less frequently, by a total lack of response in either the biceps or triceps brachii muscles (Fig. 2C, shaded bars show force; EMG data are not shown).

Effects of tendon vibration during elbow extensor MVCs. Figure 3 illustrates the general increase in triceps brachii surface EMG and extension force observed when the triceps brachii tendon was vibrated during MVCs performed by subject 4l. Without tendon vibration, the maximum voluntary elbow extensor force developed by this subject was ~20 N (Fig. 3A), but this force was doubled during triceps tendon vibration (Fig. 3B). Notice that the period of vibration preceding the onset of the MVC was also accompanied by an increase in triceps surface EMG and extension force (Fig. 3B).
The mean data for this subject during the three different experimental situations are given in Fig. 3, C–E. Compared with the MVC values (diamonds), vibration of the triceps brachii tendon (squares) gave rise to a large increase in both the extension force and the triceps surface EMG, but only a small increase in biceps surface EMG. In contrast, vibration of the biceps brachii tendon (triangles) did not change the performance of this subject, as there were only small increases in both the triceps and biceps brachii surface EMG.

Figure 4 shows the mean (±SE) data for all subjects who developed an elbow extensor MVC of at least 1% of that of able-bodied subjects. Vibration of the triceps brachii tendon (CVT) facilitated the elbow extensor force in four muscles (Fig. 4A, open bars), particularly for subject 41. In the other muscles tested, triceps tendon vibration either did not change the performance, or the elbow extensor force decreased. Even subject 1r showed no change in performance with triceps brachii tendon vibration. The results from this subject were unexpected for two reasons. First, the twitches superimposed on the MVC of this subject were the biggest that we obtained in the present study (only 29% voluntary activation, Fig. 1, C and D). The response induced by triceps tendon vibration at rest was also the strongest encountered (Fig. 2, A and C). In comparison, vibration of the biceps brachii tendon decreased the extension force in most of the subjects (Fig. 4A, shaded bars).

Figure 4, B–D, gives a general view, for all of the subjects, of the force, as well as the triceps and
biceps brachii muscle activities during elbow extensor MVCs with either triceps or biceps brachii vibration (CVT and CVB, respectively), compared with the MVC without vibration. There was a general tendency for the force to increase during triceps brachii tendon vibration and to decrease during biceps tendon vibration (Fig. 4B). Except for subject 4t, the triceps brachii surface EMG did not change much with vibration of either tendon (Fig. 4C). In contrast, the biceps brachii muscle was generally activated more during vibration and particularly during biceps vibration (Fig. 4D). One subject (subject 5r) behaved differently from the others in that the extension force increased with vibration of both the triceps and biceps brachii tendons (Fig. 4A). In both situations, this improvement was related to less involvement of the biceps brachii muscle during the MVC (lowest points in Fig. 4D).

Fig. 4. Influence of tendon vibration during elbow extensor MVCs. A: mean (±SE) elbow extensor force, relative to data from control subjects (31), generated during MVCs without tendon vibration (solid bars) and with triceps brachii (CVT; open bars) or biceps brachii tendon vibration (CVB; shaded bars). Three values were averaged per contraction (see Fig. 3B) for each subject who developed >1% of control MVC force. B-D: force data from A reported as absolute values, together with the corresponding triceps and biceps brachii surface EMG, respectively. All MVC data with biceps (CVB; left) or triceps brachii tendon vibration (CVT; right) are normalized to the MVC values without vibration (middle). Dotted lines refer to subject 4t, the data shown in Fig. 3.
DISCUSSION

The present data showed that triceps brachii tendon vibration at rest evoked a TVR in just over one-half of the triceps tested, particularly in muscles of SCI subjects with stronger elbow extensor MVC forces. Vibration applied to the tendon of triceps brachii during elbow extensor MVCs (>1% of control force) also enhanced the elbow extension force in one-half of the muscles that were evaluated. Conversely, biceps brachii tendon vibration at rest always evoked a TVR in biceps brachii, but biceps tendon vibration during elbow extensor MVCs resulted in no improvements in the extensor force. These data suggest that triceps, but not biceps brachii, tendon vibration provides a way to increase the contraction strength of some of these elbow extensor muscles that have been weakened and partially paralyzed by chronic cervical SCI.

SCI population. All of the subjects who participated in this study were representative of the SCI population with cervical injuries in that they had difficulty with elbow extension because their triceps brachii MVCs were weak compared with those of control subjects, cocontraction of triceps and biceps brachii was common, only some of the triceps brachii muscles could be activated selectively with radial nerve stimulation, and many of the triceps muscles were paralyzed partially (25, 31, 33). These characteristics suggest that these spinal injuries involved upper and/or lower motoneuron damage. Where there is upper motoneuron injury, vibration may enhance force by a number of mechanisms (e.g., enhanced muscle spindle afferent activity resulting in supraspinal and/or spinal excitatory reflex activation of motoneurons). Subjects with lower motoneuron damage may have denervated portions of muscle, but reinnervation is expected to occur from neighboring axons (30). Stretch reflexes are absent in reinnervated muscles (6), possibly because the afferents reinnervate inappropriate target organs (7), a situation that may impair reflex excitation of motoneurons after SCI. If reinnervation from intact axons fails, the muscle fibers that remain denervated will not be activated either by volition, reflexly by vibration, or by electrical stimulation of the radial nerve.

Tendon vibration at rest. It is well known that vibration of a muscle tendon at rest gives rise to a tonic contraction of the parent muscle when the subjects look at their arm (13, 14). This TVR is due to the high-frequency activation of muscle spindle primary endings (3, 20, 27), which, in turn, drive the α-motoneurons through monosynaptic and polysynaptic spinal pathways, as well as supraspinal pathways (13, 14, 19, 28). This reflex, which is almost always produced in muscles of healthy subjects (8, 14), was observed in about one-half of the triceps brachii muscles that we tested in people with chronic cervical SCI. Others have shown that the TVR is absent or weak in muscles influenced by SCI, particularly when there is complete cord transection (8, 16). In contrast, Dimitrijevic et al. (9) were always able to evoke a TVR in the muscles of SCI subjects, even when muscle paralysis was complete. However, stronger TVR responses occurred when there was greater preservation of motor and sensory function, as found in the present study. In hemiplegia, as well as in spastic muscles, the TVR may also be absent or weak, coexist with the activation of neighboring muscles, or involve antagonist muscles (3, 15). In the weaker elbow extensor muscles studied here, we also commonly saw a reflex response in biceps brachii with triceps tendon vibration.

Various factors may contribute to the abolition of the TVR or a weak TVR after chronic cervical SCI. Among these factors is the impairment of inputs from higher brain centers to the spinal cord (9, 11), as indicated here by the presence of triceps force increments in response to radial nerve stimulation (Fig. 1). Any disruption or reduction in the influence of afferent inputs at spinal or supraspinal levels may result in the vibration stimulus being ineffective or inadequate to bring all the motoneurons to threshold (18), especially in reinnervated muscles, in which an absence of stretch reflexes has been shown (6). The presence of a TVR in some of our triceps brachii muscles at rest (Fig. 2) does indicate that at least some muscle spindles were excited by tendon vibration, however. Vibration also induced clonus in one of the triceps brachii muscles that we studied. The TVR is also influenced by medication (Table 1), which may act to impair the ability of motoneurons to respond to asynchronous inputs by decreasing their susceptibility to temporal summation (18). The sensitivity of other afferents, such as Golgi tendon organs, muscle spindle secondary endings, and cutaneous afferents to vibration may also have changed after SCI (8, 9, 32), particularly if muscle reinnervation has occurred (7), altering the magnitude of the TVR.

Another way to activate the triceps brachii muscle in healthy subjects is to vibrate the biceps brachii tendon at rest while the subjects have their eyes closed. In the absence of visual cues, this vibration-induced activity of muscle spindle primary endings induces a sensation of illusory extension of the forearm that is accompanied by contraction of the muscle antagonist to the vibrated muscle in 70% of healthy subjects, i.e., the triceps brachii muscle (12, 26). The neural mechanisms underlying this reflex response, named the AVR, remain unclear. It has been suggested that the AVR results from a perceptual-to-motor transformation occurring at the cortical level rather than from spinal reflex mechanisms, because the motor activities are directly linked to the sensation (4, 5).

An AVR was never encountered in the present study. Rather, it was replaced by a TVR in the vibrated biceps brachii muscle. This absence of an AVR, together with the general lack of illusory movement, may be explained by the more marked dysfunction of elbow extensors compared with flexors (31). A TVR prevents the sensation of illusory movement in healthy subjects (26).
An absence of elbow extensor force improvement could be expected in those muscles that showed a biceps brachii response at rest during biceps or triceps brachii tendon vibration (Fig. 2). In contrast, the absence of improvement in the muscle that showed the largest TVR in triceps brachii at rest and the strongest superimposed twitches in response to radial nerve stimulation (subject 1), probably suggests that the same motor units were excited voluntarily and by tendon vibration. Such an overlap in the activated populations of motor units is probably also responsible for the lack of improvement observed in muscles that developed a TVR in triceps brachii at rest but were close to maximally activated voluntarily, as assessed by twitch interpolation (e.g., subject 2, Fig. 1D).

Functional implications. These data show that triceps brachii tendon vibration is a way to activate the triceps brachii muscle at rest in some SCI subjects who retain partial voluntary control of this muscle. It is unclear whether routine application of tendon vibration could prevent or retard the disuse atrophy present in some triceps brachii muscles (30), but vibration-induced contractions may be less uncomfortable than electrically induced contractions. In some subjects, triceps brachii tendon vibration during elbow extensor MVCs also improved the elbow extension force. Use of a miniaturized vibrator might help these individuals with SCI improve their muscle function (16), but this issue also needs quantitative evaluation in relation to the motor and sensory function that is preserved after the injury. Vibration-induced muscle contractions may be more effective in people who have an incomplete SCI without lower motoneuron involvement, so that the absence of stretch reflexes that occurs with muscle reinnervation (6, 7) is not a consideration. Furthermore, systematic investigation of the effects of various medications on these reflex responses induced by tendon vibration would be valuable.

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REFERENCES


