ABSTRACT: To show that human muscle may adapt to tendon transfer, adaptation of flexor carpi ulnaris (FCU) function was studied by measuring active and passive length–force characteristics at initial operation and at reoperation in a case of extension deformity secondary to FCU tendon transfer. At reoperation, FCU was 20 mm shorter; active force decreased ~10%, indicating atrophy; and passive force increased, reflecting increased stiffness. FCU fiber length was unchanged. The presented case shows that human forearm muscle may adapt to a transferred function.

ADAPTATION OF THE PROPERTIES OF SPASTIC MUSCLE WITH WRIST EXTENSION DEFORMITY

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Transfer of the flexor carpi ulnaris muscle (FCU) to the extensor carpi radialis brevis muscle (ECRB) is a widely used surgical procedure to correct spastic wrist flexion deformity in patients who are not able to actively extend their wrist to the neutral position.1,6 The Green transfer is based on the availability of the FCU as a strong, active working muscle under voluntary control, and the synergy of its function in its new position. It has been acknowledged only in the last 20 years that besides muscle availability and functional synergy, characteristics of muscle function should be considered to optimize the outcome of tendon transfer.3,5,11,13

Some authors caution about the risk of over-correction of the flexion deformity, resulting in a hyperextension deformity that compromises the long-term result21 (M.A. Tonkin, personal communication). An explanation for this might be the adaptability of the muscle tissue to the new conditions. Unfortunately, the limited access to human muscle in vivo does not permit extensive studying of such adaptability. As such, adaptation of muscle function after tendon transfer has never been demonstrated in human muscles.

We previously developed a reliable technique for in vivo measurements of the length–force relationship of human muscle.17 The length–force relationship reflects the muscle’s morphological properties. Muscle cross-sectional area is reflected by the maximal force,3,11 fiber length is reflected by the available excursion,2,10 and the length and stiffness of the intra-, inter-, and extramuscular connective tissues is reflected by the passive force that is exerted at the tendon.14,16

In 2001 we performed a Green transfer to correct a spastic wrist flexion deformity in a patient with cerebral palsy and intraoperatively measured the FCU length–force characteristics to study spastic muscle properties.18 Three years later, the same patient presented with a gradually progressive wrist extension deformity. Revision surgery offered a unique opportunity to test whether transposition of the FCU had altered the muscle’s morphological properties.

MATERIALS AND METHODS

Patient. In 2001, a then 20-year-old man with spastic flexion deformity of the wrist caused by cerebral palsy underwent transposition of the FCU to the ECRB as described by Beach et al.1 Preoperatively, the wrist was held in 85° of flexion without active extension. The grasp and release pattern of the hand was nonfunctional in this position of the wrist. Passively, the wrist could be moved between 50° and 110° of flexion. During surgery, however, general anesthesia allowed the wrist to be moved between 90° flexion and 50° extension. During surgery, the flexor carpi radialis muscle and the long extrinsic finger flexors were fractionally lengthened. After its...
release and transposition, the FCU was inserted in the ECRB tendon at a length that intraoperatively held the hand in a neutral position withstanding the force of gravity. The patient gave informed consent for intraoperative force–length measurements in accordance with a protocol that had been approved by our institutional Medical Ethical Committee.17

Ten months postoperatively, the results of surgery were considered satisfactory to both the surgeon and the patient. The flexion deformity was absent and the wrist was held in a resting position of 25° extension, with a limited range of motion between 10° and 45° extension.

In 2004 the patient returned with a wrist deformity of 50° extension without active range of motion. The deformity progressed gradually over time and hampered the already limited grasp–release pattern that was improved after the primary operation. Revision surgery to release the FCU from ECRB was decided. During reoperation, force–length measurements of FCU were repeated. At 12-month follow-up after FCU release, a satisfactory correction of the extension deformity was still present. Active range of motion was absent, but the wrist was held in 15° extension, which was satisfactory to serve as an assisting hand with a limited but functional grasp–release pattern.

Measurement of Length–Force Characteristics. Our method of in vivo measurement of active and passive length–force curves of the FCU has previously been validated and described in detail.17 A series of maximal tetanic contractions of the FCU were induced at subsequent muscle lengths by supramaximal transcutaneous electrical stimulation of the ulnar nerve (140 mA, 50 Hz, 0.1-ms pulse duration, 1,000-ms stimulus duration) using two gel-filled skin electrodes (RedDot 2560; 3Com, Minneapolis, Minnesota) that were pasted on the skin directly overlying the cubital tunnel of the elbow. A strain gauge was attached to a metal ring sutured on the released distal tendon of the FCU and to a metal bar that was attached to a Kirschner wire in the medial epicondyle. The strain gauge was kept aligned with the FCU. Just prior to and during stimulation, the strain gauge signal was analog-to-digital converted and stored in a computer. Force measurements were obtained at a series of muscle lengths, varying from that corresponding to well shorter than the length at maximal flexion of the wrist to that corresponding to well beyond the length at maximal wrist extension. During measurement the elbow was held in 30° flexion and the wrist was held in a neutral position.

Data Analysis. The muscle’s operating length range (OLR) is defined as the range of the FCU length from maximal passive wrist flexion to maximal passive extension and calculated during both operations. Using an Excel 2000 software package (Microsoft, Redmond, Washington), the muscle’s passive and active length–force characteristics at both the first operation and the second operation were plotted. Subsequently, both active and passive force were presented as the distance to optimum length. Data of the active force profile were fitted to a second-degree polynomial function ($R^2 = 0.977$ for the first, and $R^2 = 0.945$ for the second active length–force profile) and extrapolated to the x-axis to estimate the available active excursion of the FCU. This available active excursion was defined as the length range at which the FCU is able to actively exert force.

RESULTS

The range of motion and the corresponding muscle lengths at the first and second operation differed, but the FCU operating length range was similar at both operations (Table 1).

The measured FCU maximum active force at optimum length at the first operation was 95.1 N and at the second operation 93.5 N; this difference is smaller than the estimated measurement error of our method of 2.8%.17 However, other features of both the active and passive length–force relationship of the flexor carpi ulnaris muscle at the first operation markedly differed from that at the second operation (Fig. 1A).

The muscle optimum length shifted ~20 mm to a shorter length at the second operation (i.e., ~8% of the total muscle length). Consequently, at a length corresponding to the neutral position of the wrist, the active force was ~37 N lower at the second operation (i.e., 40% of maximum active force).

Active force was ~10% reduced at nonoptimal lengths, up to 30% at the shortest measured FCU length (Fig. 1B). By contrast, the FCU available ex-

| Table 1. Intraoperative passive wrist range-of-motion with corresponding FCU lengths, and the operating length range at the two operations. |
|---|---|---|---|---|
| Flexion (Deg) | Extension (Deg) | Lflex (cm) | Lext (cm) | OLR (cm) |
| First operation | 90 | 50 | 24.9 | 27.2 | 2.3 |
| Second operation | 10 | 90 | 25.1 | 23.0 | 2.1 |

Lflex, FCU length at maximal flexion angle; Lext, FCU length at maximal extension angle; OLR, FCU operating length range.
cursion only slightly differed at both operations, between 58 mm at the first operation and 55 mm at the second operation.

The range of FCU length at which it exerted over 70% of its maximum active force ranged from ~24.0 cm to 26.5 cm at the first operation (3.0 cm), and from 23 to 25 cm (2.0 cm) at the second operation, a decrease of 33%.

The passive force was increased at a neutral position of the wrist (Fig. 1A). After correction for the shorter muscle length at the second operation, passive force was the same at both operations at short lengths up to optimum length. At higher lengths, passive force was markedly higher at the second operation (Fig. 1B).

**DISCUSSION**

The altered length–force characteristics of the present study show that the FCU has shortened. This indicates that, indeed, the spastic FCU may adapt to transposition by shortening its length. This has functional consequences, as the range of lengths at which the FCU exerted high force decreased dramatically. Additionally, the steeper incline of the passive force at the second operation indicates that the FCU had become less extensible over optimum length, due to an increased stiffness or shortening of the intra-, inter-, and extramuscular connective tissues. This may further explain the extension deformity in this patient.

The 8% shortening of the FCU after its transposition may be explained by muscle atrophy because of the related ~10% reduction of the active force around optimum length that is expected from a loss of the cross-sectional area due to the atrophy. The measured maximum active force was not different, however, in opposition to this explanation.

As the available active excursion is mainly determined by the muscle-fiber length, the small difference in the available active excursion between the first and second operation indicates that the shortening of the FCU is not caused by a shortening of the muscle fibers. The extrapolation of the force data by the second-degree curve-fitting in the present study is a quite approximate method, and the relative length ranges over which forces were measured during the two operations were substantially different, limiting the credibility of the estimated excursion. However, we feel confident that such a method differentiates between muscle fiber lengths that would explain the FCU shortening of 20 mm. Moreover, the available excursion from the present study is only slightly less than that predicted from the muscle architecture of a recent anatomical study that showed the average FCU muscle fiber length to be 63 mm. In addition, the findings from the present study are in accordance with previous study of spastic calf muscles that showed them to be atrophied, rather than to have shortened muscle fibers after surgical aponeurectomy. The decrease in passive extensibility over optimum length in the case presented here may be explained either by an increase or shortening of connective tissue within the FCU and directly adjacent to it, or increased stiffness of intracellular noncontractile structures such as titin, and requires further study.

Using our novel method to directly measure the active and passive length–force characteristics of human spastic muscle, the present study shows that, at least in the selected case, human forearm muscle adapts to a transferred function. We cannot explain why this adaptation occurs. Possibly, the moment that the transferred spastic FCU exerts is greater than that exerted by the flexor muscles due to a greater moment arm that allowed for the FCU to be...
short while holding the wrist in extension. It is, however, unexplained why the shortening of the FCU did not occur in the years prior to the first surgery, because the fixed, extremely flexed position of the wrist obliged the FCU to remain in a short position. This supports the assumption that specific conditions that were applied to the FCU after the tendon transfer triggered the change of muscle function, as opposed to the underlying pathology of cerebral palsy. These specific conditions include the stretch to the muscles,5,19 muscle excitation,7 and the formation of intra-, inter-, and extramuscular connective tissues.8,20 Unfortunately, we lack information on any of these conditions, and we do not know why this particular case developed hyperextension after FCU transfer, whereas most patients do not. There is still little knowledge on the mechanisms of muscle adaptation, but our case shows the clinical need for studying them, as they may influence the outcome of tendon transfer surgery.

REFERENCES