Biomechanical effects of dissecting flexor carpi ulnaris

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Our aim was to determine whether the length and function of the flexor carpi ulnaris muscle were affected by separating it from its soft tissue connections. We measured the length of flexor carpi ulnaris before and after its dissection in ten patients with cerebral palsy. After tenotomy, tetanic contraction shortened the muscle by a mean of 8 mm. Subsequent dissection to separate it from all soft tissue connections, resulted in a further mean shortening of 17 mm ($p < 0.001$). This indicated that the dissected connective tissue had been strong enough to maintain the length of the contracting muscle. Passive extension of the wrist still lengthened the muscle after tenotomy, whereas this excursion significantly decreased after subsequent dissection.

We conclude that the connective tissue envelope, which may be dissected during tendon transfer of flexor carpi ulnaris may act as a myofascial pathway for the transmission of force. This may have clinical implications for the outcome after tendon transfer.

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In order to transfer a tendon, the connective tissue which surrounds the donor muscle is dissected until the line of pull to the receptor tendon is in a straight line. Animal experiments have shown that these fascial connections act as path-

ways for the transmission of force between the muscle and its environment.¹⁻⁴ The force of a muscle was found to be transmitted not only through its tendon, but also through connective tissue to adjacent muscles and non-contractile tissues. Consequently, this pathway of biomechanical interaction may significantly affect individual muscle function.^{1,2} Since the success of tendon transfer surgery depends on the function of the donor muscle after transfer, we have studied the passive and active changes in the length of the muscle of flexor carpi ulnaris (FCU) during surgical dissection.

The prerequisites for myofascial connections to act as a pathway for the transmission of force are that the connective tissue is strong enough to transmit force and that it can transmit it to a distal target through adjacent muscles and other extramuscular tissues.

In order to test the hypothesis that the connective tissue which is dissected is strong enough to transmit force, we measured the length of the FCU muscle after simple tenotomy and after subsequent unloaded maximal tetanic contraction. Further measurements were made after dissection of the muscle and again after maximal tetanic contraction. Immediately after tenotomy, the muscle was expected to shorten passively until its passive elastic force equalled the opposing resistance from the surrounding fascia.^{2,3} Maximal tetanic contraction was expected to shorten the muscle further until a new equilibrium between the active force of the muscle and the fascial resistance was reached. If progressive dissection was to result in further passive and active shortening of the FCU, it would follow that the dissected connective tissue had been able to prevent the muscle from shortening and therefore had resisted the active force exerted by the muscle. To test the hypothesis that the inter- and extramuscular connective tissue actually transmits passive force, the length of the FCU muscle was measured before and after moving the wrist passively from maximal flexion to maximal extension. For the hypothesis to be proven, the muscle would have to lengthen passively while the wrist was moved from flexion to extension with the myofascial pathways intact, even after it had been completely disconnected from the wrist. After subsequent division of the myofascial pathways by progressive dissection, FCU would no longer lengthen by passive extension of the adjacent muscles which remain connected across the wrist.

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Fig. 1

Diagram showing the mean shortening of the muscle of FCU after tenotomy and after dissection, before and after tetanic contraction (ML, muscle length).

We present the results of this experiment carried out during transfer of the FCU in patients with cerebral palsy.

Patients and Methods

We carried out transfer of FCU to the tendon of extensor carpi radialis brevis to correct a flexion deformity of the wrist in ten patients with cerebral palsy. The four boys and six girls had a mean age of 14 years (6 to 19). Ethical approval was granted and informed consent obtained from all patients.

Operative technique. Under general anaesthesia, a longitudinal incision was made over the distal third of the forearm from the pisiform bone along the ulnar border of FCU. Care was taken not to damage the fascial surroundings of the belly of the FCU muscle. The distal part of the tendon was prepared for tenotomy. The insertion of the distal muscle fibres into the tendon was marked with a fine suture. A Kirschner wire was placed in the medial humeral epicondyle to mark the origin of FCU, and the length of the FCU muscle was defined as the distance between this wire and the marker in the distal tendon (Fig. 1).

Length of FCU muscle before and after muscle dissection. The length of the muscle was measured with the wrist in neutral (0° flexion) and the elbow at a constant angle of flexion. We undertook three series of measurements of the length of the muscle: 1) with the FCU intact; 2) after division of the tendon of FCU and its paratenon just proximal to the pisiform bone, with complete release from its insertion; and 3) after careful dissection of the belly of the FCU muscle from its fascial surroundings throughout its length allowing transfer of the tendon and muscle to the dorsal aspect of the wrist in a straight line. The length of the muscle was measured before and after maximal tetanic contraction which was induced using two electrodes (RedDot 2560 skin electrodes; 3M Corporation, Maplewood, Minnesota) placed on the skin over the ulnar nerve in the cubital tunnel and a peripheral nerve stimulator (AMC-Medical

Technical Development, Amsterdam, The Netherlands). Supramaximal tetanic contraction of the ulnar nerve was provoked using biphasic, intermittent pulses of an amplitude of 125 mA at a stimulus frequency of 50 Hz, a pulse duration of 0.1 ms, and a stimulus duration of 1000 ms, with the limb free to move. All measurements were made without the use of a tourniquet or muscle relaxants.

Passive excursion of FCU before and after dissection.

We defined the passive excursion of the FCU as the lengthening of the muscle on passive movement of the wrist from maximal flexion to maximal extension. Immediately after each of the above three series of measurements, the length of the muscle was measured with the wrist in flexion and extension. The angles of maximal flexion and extension of the wrist before tenotomy were the same for all series of measurements, and verified using a goniometer.

Statistical analysis. The means of the measurements were compared using paired Student's *t*-test. The passive excursion of the FCU was calculated by subtracting the length of the muscle at maximal passive flexion from that at maximal passive extension of the wrist. The mean passive excursion for each series of measurements was also compared using paired Student's *t*-test.

Results

Length of the FCU muscle before and after dissection.

The mean length of the muscle with the wrist in the neutral position before tenotomy was 221 ± 36.2 mm (SD) (Table I). After tenotomy alone, it shortened to a mean of 216 ± 37.7 mm and shortened further to a mean of 213 ± 37.8 mm after subsequent maximal tetanic contraction (Fig. 1). Progressive dissection caused a further mean shortening of 204 ± 36.9 mm ($p < 0.001$). These results show that the connective tissue before dissection had been strong enough to oppose the active force of the muscle and to limit shortening. Tetanic contraction after dissection yielded the most shortening (mean 196 ± 36.3 mm). These observations support

Table I. Lengths of FCU in ten children with cerebral palsy with the FCU tendon intact, after tenotomy and after muscle dissection; before and after tetanic muscle contraction

Case	Muscle length (mm)				
	With FCU intact	After tenotomy		After dissection	
		Before	After	Before	After
1	233	229	225	222	214
2	217	211	204	200	190
3	187	183	181	164	153
4	260	258	257	246	240
5	229	225	223	220	206
6	151	143	140	136	131
7	182	177	173	166	157
8	261	261	255	247	237
9	268	263	261	245	235
10	222	213	212	197	192
Mean (SD)	221 (36.2)	216 (37.7)	213 (37.8)	204 (36.9)	196 (36.3)

Table II. Passive excursion of FCU in ten children with cerebral palsy during movement of the wrist from maximal flexion to maximal extension with the FCU intact; after tenotomy and after muscle dissection

Case	With FCU intact	After tenotomy	After dissection
1	12	12	0
2	20	14	0
3	16	16	0
4	18	17	1
5	25	21	5
6	17	16	4
7	15	9	4
8	22	18	0
9	23	22	3
10	14	13	1
Mean (SD)	18 (4.0)	16 (3.8)	2 (1.9)

the hypothesis that the connective tissue envelope is strong enough to transmit force.

Passive excursion of the FCU before and after dissection. The mean excursion on passive movement of the wrist, from maximal flexion to maximal extension, was 18.0 ± 4.0 mm (Table II). After tenotomy, the mean passive excursion was 16 ± 3.8 mm, which is 89% of the original excursion, despite the fact that FCU had been disconnected from its insertion at the wrist. This implies that other tissues crossing the joint transmit force from FCU. After dissection of all soft-tissue connections from the belly of the muscle, the same passive movement of the wrist induced only 11% of the original excursion (mean 2 ± 1.9 mm) (Fig. 2). In four patients, the passive excursion was zero. These observations support our second hypothesis that the inter- and extramuscular connective tissue acts as a pathway for the transmis-

Fig. 2

Diagram showing the mean passive excursion of FCU during movement of the wrist from maximal flexion to maximal extension before tenotomy, after tenotomy and after muscle dissection (ML, muscle length).

sion of force from the adjacent muscles to the disconnected distal tendon of FCU.

Discussion

The biomechanical properties of muscles which may be calculated from parameters of their architecture, are regarded as the most important determinants of their function.⁵⁻⁷ Thus properties, such as the available active excursion of the tendon and the force capacity calculated from the architecture of the isolated muscle belly, are used to differentiate between muscles which may be used for tendon transfer.^{5,7,8} This presumes that the function of a muscle will not change so long as its architecture is not changed. The biomechanics of a muscle also depend on its adjacent connections.¹⁻⁴ Previous studies of the brachioradialis muscle have shown that division of its antebrachial fascial connections increased its available excursion.⁸⁻¹⁰

We found that the inter- and extramuscular connective tissue around FCU also influenced the transmission of force from the muscle belly distally. This observation is in variance with the theory that fascia is too compliant to transmit force.¹¹ It proved strong enough to withstand the exerted active muscle force and stiff enough to transmit this force. Only after division of the myofascial pathways by progressive dissection did the excursion of FCU caused by passive extension from the adjacent muscles, decrease dramatically. The residual lengthening of the muscle in the other patients may be explained by the fact that the dissection of fascial connections was incomplete being only undertaken until transfer to the dorsal aspect of the wrist was in a straight line.

Recently introduced, so-called myofascial pathways of transmission of force have proved to be an important bio-

mechanical determinant of the function of muscle in patients with cerebral palsy. As such, progressive dissection of the muscle may affect the outcome of tendon transfer surgery.

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