Measurements of tennis players’ specific forearm muscle force imbalance to assess the potential risk of lateral epicondylitis

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Abstract

Several studies suggested that lateral epicondylitis could potentially develop among tennis players presenting a muscle force imbalance, defined as a functional ratio weakness between agonist and antagonist muscles and/or a predominance of synergic muscles during the movement. Consequently, the measurement of muscle force capability and the assessment of imbalance between antagonist and agonist muscle groups could be useful in the context of clinical follow-up, ascertaining the subject’s functional capabilities to return to tennis practice. The purpose of the present study was to develop a method to quantify subject’s specific imbalance in forearm muscles to improve the knowledge about lateral epicondylitis. The study included a group of healthy male tennis players (n=11, no history of musculoskeletal problems) and specific tennismen profiles (recovered lateral epicondylitis (n=2), expert (n=1) and woman (n=1)). Maximal isometric net torques in both flexion and extension were measured at the metacarpophalangeal and wrist joints using an isokinetic ergometer. Tennis players’ specific fingers and wrist muscle force imbalance between agonistic and antagonistic prehension muscles were estimated using a two-step calculation procedure including (i) an EMG-calibration procedure in order to integrate the muscle coactivation in net joint torques calculation and; (ii) the calculation of the antagonist/agonist torque ratio from corrected joint torques. The results showed dissimilar muscle force imbalances between the healthy men group and recovered lateral epicondylitis players. The observed differences between healthy players and recovered lateral epicondylitis suggested that the proposed method of muscle force imbalance estimation may provide a useful assessment of functional recovery in lateral epicondylitis.
1. Introduction

Lateral epicondylitis, also called tennis elbow (TE), is one of the commonest tendinopathy localized at the muscular insertion of finger and wrist extensor muscles, i.e. extensor carpi radialis brevis and extensor digitorum communis, respectively. The incidence of TE in tennis player career has been reported as comprised between 35 and 51% (Abrams et al. (2012)). In the most serious cases, this disease could lead to a partial or complete rupture of the common wrist extensor tendon.

Many factors are identified to significantly contribute to TE. On one hand, the transmission of the initial shock wave, and subsequent vibrations, from the racquet to the wrist and elbow joints has been hypothesized as the likely mechanisms of TE (Knudson and Blackwell (1997)). This could be influenced by the use of an inappropriate material (i.e. tennis racquet head size, resonance frequency, weight or stringing) or the level of expertise. On the other hand, TE is thought to result from overuse of the finger/wrist extensor muscles. The eccentric stretching of finger/wrist extensor muscles caused by an extreme nonneutral posture of the wrist, including a faulty wrist kinematics during the backhand stroke, has been identified as a risk factor (Knudson (2004)). However, a predominant activity of the finger/wrist extensor muscles has been measured in all tennis strokes. Nirschl (1973) specifically identified biomechanical factors in faulty forehand stroke technique which could contribute to TE. Rossi et al. (2014) suggested a relation between TE and a combination of forceful and repetitive cocontractions of the extensor muscles in grip tasks for both backhand and forehand strokes. Indeed, the gripping task associated to the handle squeezing results in the activation of both pluri-articular finger/wrist flexor and extensor muscles (Goislard et al. (2012)), even though this task only entails the flexion of metacarpophalangeal (MCP) and phalangeal joints. This suggests that, during prolonged practise, repetitive high level of finger/wrist extensor muscle solicitations in all tennis strokes could provoke the overuse of these extensor muscles and affect the tendinous tissue of their origins in the most extreme cases.

As a consequence, several authors (Strizak et al. (1983), Alizadehkhaiyat et al. (2009)) suggested that a potential muscle force imbalance, defined as a functional weakness ratio between agonist and antagonist muscles or a predominance of synergic muscles during the movement, could be the cause of progressive tendinous damages. TE would be ascribed an incapability to develop the adequate muscle force by the finger/wrist extensor muscles. Assessing muscle force imbalance between extensor and flexor muscles in both wrist and MCP joints therefore appears crucial to investigate the muscular adaptive mechanism caused by tennis practice and the potential implications for prevention of TE and clinical follow-up.

Studies aiming to investigate upper limb muscle force imbalance commonly focused on the measurement of the maximal isometric net strength/torque and electromyography (EMG) method during wrist movements (Alizadehkhaiyat et al. (2009)). Nevertheless, the assessment of muscle force imbalance from EMG signals or dynamometry has significant limits. First, EMG data alone could lead to erroneous conclusions on muscle activation because of normalization issues and possible distortions of the signal. Second, the measurement of maximal muscle strength/torque largely underestimated the muscle forces actually produced during the isometric task since the coactivation required to the joint mechanical equilibrium was not considered. To overcome these limitations, alternative methods of muscle force estimation associated dynamometry with the use of EMG signals, which provide information on the level of muscle coactivation (Rao et al. (2009)). Furthermore, the above studies only focused on muscle force imbalance at the wrist joint whereas De Smedt et al. (2007) identified that TE results in a tendinosis of not only the extensor carpi radialis brevis the extensor digitorum communis.

In the present study, we aimed to propose a novel method to investigate specific muscle force imbalance in finger and wrist muscle groups for various profiles of tennis player. Specific muscle force imbalances were computed from MCP and wrist joint torque measurements and an EMG-based calibration procedure in order to represent muscle cocontraction required to the joint mechanical equilibrium during the isometric maximal task. Three specific tasks were used to assess the muscle force capability of distinct finger and wrist muscle groups.
Nomenclature

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>TE</td>
<td>tennis elbow</td>
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<tr>
<td>MCP</td>
<td>metacarpophalangeal joint</td>
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<td>EMG</td>
<td>electromyography</td>
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<td>RTE</td>
<td>recovered tennis elbow</td>
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<td>RMS</td>
<td>root mean square</td>
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2. Material and methods

2.1. Participants

Fifteen subjects (14 men and 1 woman; mean age: 20.8 ± 1.4 years old; mean height: 177.0 ± 6.6 cm; mean weight: 67.6 ± 7.8 kg; hand size: 19.2 ± 1.0 cm) volunteered for the experimentation. Participants were composed of experienced tennismen without history of TE (n=11), expert (n=1), woman (n=1) and recovered tennis elbow (n=2, RTE* and RTE**, two different nonsurgical treatments to TE) subjects. The inclusion criteria in recovered tennis elbow group was that tennis elbow was diagnosed at least 10 months before the experimentation.

2.2. Procedure

A purpose-built wrist and hand isokinetic ergometer (Bio2M, Compiègne, France) inspired from Schweizer et al. (2003) was used for (i) MCP, (ii) wrist and (iii) combined MCP-wrist maximal flexion/extension torque measurements. The ergometer was used in static condition, fixed vertically and adjusted in relation to subject height (Fig. 1). Measurements were done with the wrist fixed in neutral position and the shoulder in 0-15° abduction and neutral rotation. For each condition, specific modules were used:

- For measurements of wrist flexion/extension torque (T1), a specific module enwrapped the hand palm proximally to the MCP joint line. The torque measurements were performed at the wrist joint.
- A second module was used to measure the combined action of fingers and wrist muscles (T2). This module enwrapped the hand palm and the four fingers (index, middle, ring and little) from their tip to a line located proximally to the MCP joints. Similarly to the previous condition, measurements were done around the wrist joint.
- Measurements of MCP flexion/extension torque (T3) were done with a third module which enwrapped the four fingers from their tip to a line located distally to the MCP joints. The torque measurements were performed at the line of MCP joints.

Subjects were asked to apply a maximal torque at the MCP or the wrist joint during 6s. Subjects forced on module surfaces with the palmar side of the fingers for flexion and dorsal side for extension. For each condition, two consecutive trials were performed from both extension and flexion. Maximal trials were considered for the analysis.

A Biopac MP 150 system (Biopac Systems, Inc. Santa Barbara, CA, US) and Acqknowledge software were used for EMG data acquisition. The raw EMG signal was recorded and differentially amplified, then digitized at a sampling rate of 2000 Hz. Forearm EMG activity was recorded for extensor carpi radialis, extensor digitorum communis, flexor carpi radialis and flexor digitorum superficialis. After appropriate skin preparation, 9 mm diameter surface electrodes Ag/AgCl (Oxford Instruments Medical, Surrey, UK) were applied to the skin over the muscles with a 20 mm inter-electrode spacing. Correct placement of the electrodes was confirmed with functional tests including ulnar deviation, finger and wrist flexion, and finger and wrist extension. EMG signals were filtered off-line using a zerolag Butterworth filter (order 4, bandpass from 10 to 400 Hz).
2.3. Assessment of muscle force imbalance

Averaged joint torque was calculated within a 750 ms window centered on the peak torque. Within this time interval, the EMG root mean square (RMS) value was computed. In order to assess the muscle activation, RMS value computed for each muscle was normalized to the largest RMS value recorded for the corresponding muscle among all trials.

One trial of right upper limb postural fitting task with maximum voluntary contraction was performed by the subjects in order to measure the basic ratio of coactivation between forearm extensor and flexor muscles (Falconer and Winter (1985)). During this task, the subjects were asked to statically cocontract all hand and forearm muscles in a similar posture than the one used for torque measurements. From this postural fitting task, a calibration procedure of torque measurement was performed to account for the basic level of maximal coactivation as:

\[
\bar{T} = \frac{T}{1 - \left( \frac{a_{antago, task}}{a_{ago, task}} \times \frac{a_{ago, posture}}{a_{antago, posture}} \right)}
\]

where \(\bar{T}\) and \(T\) were respectively the adjusted and the measured MCP or wrist torque. \(a_{ago, posture}\) and \(a_{antago, posture}\) were respectively the activation of the agonist and the antagonist muscle recorded during the upper limb postural fitting task. \(a_{ago, task}\) and \(a_{antago, task}\) were respectively the activation of the agonist and antagonist muscles recorded during torque measurement. The extension/flexion ratio \(r_j\) at the joint \(j\) (\(j = \text{wrist, MCP, combined wrist-MCP}\)) was calculated from the adjusted extension and flexion torques as:

\[
r_j = \frac{T_{ext}}{T_{flex}}
\]

\(\bar{T}\) and \(r_j\) were computed during the six tasks (T1, T2 and T3 for flexion and extension) for each subject. Mean and standard deviation (SD) were computed for the healthy man group results. Torque and ratio were also computed for the specific subject profiles (RTE, expert and woman subjects). Since no statistical test could be performed for the latter, we highlighted the results higher than mean (+1SD) or lower than mean (-1SD) of the healthy man group data.
3. Results

The values of muscle coactivation between extensor and flexor muscles varied in all subjects and were ranged from 0.08 to 0.75 in flexion and from 0.02 to 0.24 in extension (Table 1). Muscle coactivation in each extension tasks was lower than coactivation in flexion tasks. Values of coactivation implied that the adjusted joint torque were respectively corrected from the measured torque by 19.2 ± 12.4 % in flexion and 23.8 ± 15.6 % in extension.

Table 1. Summary of adjusted torque values (N.cm) and index of coactivation in wrist (T1), combined wrist-MCP (T2) and MCP (T3) tasks observed in the group of healthy male tennis player (mean ± SD) and, expert, woman, RTE* and RTE** tennis players.

<table>
<thead>
<tr>
<th></th>
<th>Man</th>
<th>Expert</th>
<th>Woman</th>
<th>RTE*</th>
<th>RTE**</th>
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<tr>
<td><strong>T1</strong> Torque</td>
<td></td>
<td></td>
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<tr>
<td>Flexion</td>
<td>15.3 ± 2.6</td>
<td>28.2 ± 4.4</td>
<td>14.1 ± 3.2</td>
<td>14.2 ± 3.0</td>
<td>27.0 ± 4.4</td>
</tr>
<tr>
<td>Extension</td>
<td>13.6 ± 3.7</td>
<td>13.5 ± 2.5</td>
<td>3.3 ± 0.5</td>
<td>8.0 ± 1.5</td>
<td>13.7 ± 2.5</td>
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<tr>
<td><strong>T2</strong> Torque</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexion</td>
<td>0.22 ± 0.13</td>
<td>0.23 ± 0.10</td>
<td>0.29 ± 0.14</td>
<td>0.24 ± 0.17</td>
<td>0.17 ± 0.08</td>
</tr>
<tr>
<td>Extension</td>
<td>0.11 ± 0.07</td>
<td>0.10 ± 0.04</td>
<td>0.11 ± 0.04</td>
<td>0.04 ± 0.06</td>
<td>0.07 ± 0.10</td>
</tr>
<tr>
<td><strong>T3</strong> Torque</td>
<td></td>
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<td></td>
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<tr>
<td>Flexion</td>
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<td></td>
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</tr>
<tr>
<td>Extension</td>
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</table>

Mean adjusted extension and flexion torques for each task (T1, T2 and T3) for the healthy man group are compared with torques in expert, RTE and woman subjects in Table 1. Wrist, MCP and combined wrist-MCP flexion torques were greater than that torques obtained in extension. Focusing in result of specific subjects, we observed that wrist flexion torque is higher in RTE subjects than in healthy man group.

Figure 2 shows that mean muscular ratios in healthy man group were 0.91 ± 0.10 for the wrist, 0.51 ± 0.10 for the MCP-wrist and 0.41 ± 0.10 for the MCP joints. Wrist muscular ratio was lower in expert, RTE and woman subjects than healthy man population. MCP and combined wrist-MCP muscular ratios in expert and RTE* subjects were close to the healthy man group mean results.

4. Discussion and conclusion

This study investigated muscle force capabilities for various profiles of tennis player. The proposed method integrated the level of muscle coactivation required to maximum isometric tasks using an EMG-based calibration procedure. This resulted in largely increasing muscular torques in comparison to measured net torque. Especially, the level of muscle coactivation varied both between flexion and extension tasks and between all tested subjects, therefore confirming that muscle coactivation should be integrated in the calculation of muscle torque capacities.

Using the proposed method, normative data of averaged wrist, MCP and combined wrist-MCP torques and ratios has been established from a healthy man group of experienced tennismen. We observed that specific subjects’ torques and ratios of clearly differed from normative data. As an example, a weaker muscle force ratio for the wrist and combined wrist-fingers tasks was found in woman player than in healthy man group. Besides, individual subject results show that wrist joint seemed to be more inclined to muscle force imbalance. Indeed, wrist flexors were stronger than extensors for individual profiles. This confirms that individual tennis player profiles present specific muscle force imbalance and the proposed method seems to be suitable for assessing these specific muscle force capabilities.

Alizadehkhaiyat et al. (2009) noted a global weakness of the wrist flexion force production and an increased activation of the ECRB for RTE subjects compared to “non-skilled” healthy players. They concluded that despite
attenuation of pain, global upper limb weakness indicated incomplete functional recovery. By contrast, we found both higher wrist flexion torque and higher wrist muscle force imbalance in experienced TE subjects who made a complete functional recovery and returned to tennis practice. These findings suggest that monitoring hand and forearm muscle force imbalance with the proposed method may provide a more comprehensive assessment of functional recovery in TE.

Our results also corroborate the hypothesis that muscle force imbalance increases with tennis practice since wrist muscle ratio was higher in expert than in healthy non-expert players. Focusing on elbow joint, Bazzuchi et al. (2008) confirmed this assumption since values of elbow muscle ratio were higher in “non-skilled” individuals than expert players.

![Fig. 2. Histogram of extension/flexion torque ratio in the group of healthy male tennis player (mean ± SD) and, expert, woman, RTE* and RTE** tennis players computed for wrist, combined wrist-MCP and MCP tasks.](image)

5. Perspectives

Future works will consist on assessing upper limb muscle force capabilities for several subjects with various tennis profiles (non-expert, expert, TE, woman, young, old...). Definition of these force capabilities may allow better understanding the biomechanical factors influencing the TE risk.

References


