The Influence of Fatigue on EMG and Impact Acceleration in Running
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Abstract
This research was aimed at analyzing the effects of fatigue on the ability of the human musculoskeletal system to attenuate the heel strike generated shock waves and to correlate any existing changes in the EMG signal with other fatigue measures. Twenty-two healthy male subjects participated in this research. Each subject was instrumented with a light-weight accelerometer placed externally over the tibial tuberosity and half of the subjects also with two pairs of bipolar surface EMG electrodes placed over the quadriceps and gastrocnemius. The subjects ran on a treadmill for 30 min at a speed corresponding to their individual anaerobic threshold level and the acceleration and EMG data were sampled. Fatigue was monitored by the respiratory end-tidal CO2 pressure. The obtained results show that whenever general fatigue occurred, the amplitude of acceleration signal steadily increased. However, in both the time and frequency domains, EMG did not correlate with fatigue. Thus, one may conclude that when fatigued, the human musculoskeletal system becomes less capable of handling the heel strike induced shock acceleration.

Key words: accelerometer, electromyography, fatigue, running, shock acceleration.

Basic Appl Myol 7 (2): 111-118, 1997

Continuing popularity of running stems from its simplicity. Running is the exercise of choice for millions of people, mainly since there is no need for special equipment and there is an abundance of exercise locations. However, running is also accompanied by an increase of musculoskeletal injuries. These injuries may be caused by a number of factors, such as unsuitable running surfaces as suggested by several researchers [6, 13, 29]. The dynamic loading on the human musculoskeletal system due to foot strikes was also implicated as one of the factors in the development of injuries associated with running. Dynamic loading at heel strike may also be substantially enhanced in pathologies such as those characterized by partial or full immobilization of the ankle joint complex [30]. Recent findings [33] suggest that fatigue of the subject may be the cause of dynamic loading increase.

One of the important functions of the human musculoskeletal system is to attenuate and dissipate shock waves initiated with foot-ground contact. These shock waves are initiated by any type of motion, such as walking, running, stair climbing, etc. The generation, propagation, and attenuation of shock waves is a normal physiological process that accompanies about any human motion. The fundamental research work by Radin et al. [26] showed that the high impact forces associated with heel strike may cause damage to articular cartilage. It was suggested that overloading of the functional capacity of the human musculoskeletal system may lead to the development of osteoarthritis. Radin et al. [27] indicated that osteoarthritis results mainly from poorly handled mechanical load, rather than from a disease. Voloshin and Wosk [32] showed that low back pain may result from overloading the human musculoskeletal system because of its reduced shock absorbing capacity.

Heel strike initiated shock waves are measured mainly by skin mounted accelerometers [4, 17, 18, 35]. The principal method of attachment to the skin is by strapping the accelerometer to bony prominences such as tibial tuberosity, forehead, and sacrum.

It is a well-known fact that fatigue of the muscles affects not only their own performance, but also other related functions. For instance, muscles were shown to act to lower the bending stress on bone and to attenuate the peak dynamic loads that can damage musculoskeletal tissues [28]. Thus, athletes have been advised to avoid impulsive loads for which they are unprepared, and to not participate in athletics when their muscles are fatigued.
When the muscle's ability to perform is diminished, articular cartilage and ligaments become more vulnerable to excess dynamic loading \[1, 31, 36\]. Fatigue may cause modifications in the body dynamics which will lead to the loss of the muscles inherent ability to protect internal tissues from excessive shock waves.

Muscular fatigue has been studied extensively in the past via use of the electromyographic (EMG) signals. Temporal \[5, 15, 21\] and frequency \[9, 19, 23\] characteristics of the EMG signal were used as measures of muscle fatigue.

The present study was undertaken to analyze the effect of fatigue on the ability of the human musculoskeletal system to attenuate heel strike generated shock accelerations and to correlate any existing changes in the EMG signal with other fatigue measures. Two questions were tested in this work is: 1) Does fatigue hamper the ability of the human musculoskeletal system to protect itself from overloading by heel strike generated shock waves? 2) What quantitative characteristics of the EMG signal may be used for detection of muscle fatigue while running at the anaerobic threshold (AT) level? Knowledge of the influence of fatigue on the magnitude of dynamic loading on the human musculoskeletal system and the development of convenient means for quantifying it may allow the development of proper training procedures and exercises, and possibly lead to reduction of the damage to the musculoskeletal tissues.

**Methods**

*Subjects and experimental protocol*

Twenty two male subjects of 30.8 ± 5.1 years of age, height 173.9 ± 7.3 cm, and with body mass 70.4 ± 9.2 kg, volunteered to participate in this study. All subjects were in an excellent state of health. They all practiced calisthenics at least twice a week. No previous histories of muscle weakness, neurological disease, or drug therapy were ever recorded. Each subject provided informal consent according to the local ethical committee's guidelines. To assure uniformity of the testing conditions all subjects were provided with the same manufacturer and type running shoes.

Running tests were performed on a treadmill (Quinton Q55) to allow reproducible monitoring of the heel strike induced shock waves, oxygen consumption, and speed of running. During running, the subject breathed through a mouthpiece attached to a turbine device. The subject's expired gas was continuously sampled by a Sensor-Medics 4400 metabolic cart for breath-by-breath determination of gas exchange and ventilatory variables. The instrument was calibrated before every test. Exercise values for oxygen consumption (VO2), minute ventilation (VE), carbon dioxide production (VCO2), end-tidal carbon dioxide pressure (PETCO2), ventilatory equivalent for oxygen (VE/VO2) and ventilatory equivalent for carbon dioxide (VE/VCO2) were calculated as an average of the breath-by-breath data during a time span of 30 sec.

Before beginning the experiment, each subject was subjected to an incremental load on the treadmill with an increasing speed to determine his AT. Anaerobic threshold was determined non-invasively as the onset of initial increase in each of the ventilatory equivalent for oxygen (VE/VO2) and ventilatory equivalent for carbon dioxide (VE/VCO2) \[34\]. The running speed was initially 1 m/s and was increased every 30 sec by increments of 0.22 m/s until the point of AT determination. Figure 1 shows a typical record of VE/VO2 and VE/VCO2. The point of increase in both ventilatory equivalents defines the speed corresponding to AT level.

The running test lasted 30 min at the speed corresponding to the AT level of each subject. It is well known that if the subjects exercised long enough at a work rate corresponding to the AT, a decrease of PETCO2 may result \[34\]. If present, a decrease of PETCO2 indicates a decrease in performance, leading to fatigue \[22\]. According to PETCO2 by the end of the 30 min of running on the treadmill all subjects were divided into two groups. One group (n = 10) had a significant decrease (p < 0.05) of PETCO2 at the end of the 30 min run and it was defined as the fatigue group. The other group (n = 12) did not show a significant change of the amount of PETCO2 at the end of the test (Fig. 2) and was defined as the non-fatigue group. Age, height, body mass and running speed were not significantly different between the groups (Table 1). The average speed for the fatigue group (2.76 ± 0.29 m/sec) was not significantly different from the non-fatigue group (2.75 ± 0.48 m/sec).

Each subject was instrumented with a light-weight (2.3 grams) accelerometer which was externally attached above the tibial tuberosity. The accelerometer was aligned...
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along the longitudinal axis of the tibia to provide axial components of tibial acceleration. The accelerometer was attached by a metal holder strapped on the skin. Such attachment is capable of faithfully measuring the amplitude of a shock wave, as it was shown by Kim et al. [14]. Two pairs of small bipolar disposable Ag/AgCl snap surface electrodes (10 mm diameter, Promedico Ltd.) were placed over the quadriceps and gastrocnemius of the subject’s right leg to monitor EMG activities of those muscles during running. However, only half of the subjects (12) were instrumented with the surface electrodes for EMG evaluation due to procedural constraints.

Data acquisition and analysis

For each 30 min test, seven files of acceleration data were generated. The EMG data files were recorded starting at Minute-5 from the beginning. The PETCO2 data were also acquired at the breath-by-breath sampling rate and stored for processing. Data processing was simplified due to the constant speed of running during each test. Software was developed that utilized the given speed and location of the first heel strike. After this it proceeded automatically and detected all consequent occurrences of the heel strike.

Since there was always a possibility of “bad” data (i.e. subject stumbled or misplaced the foot), all results were shown on the monitor and had to be confirmed by an operator.

The analyzed variables were the maximum amplitudes of the accelerations recorded at foot strike on the tibial tuberosity (Fig. 3a); mean and RMS of the EMG signal in the time domain and mean and median frequency of the same signal in the frequency domain, and PETCO2 (Fig. 2). For each subject the maximum accelerations recorded in one file were measured and averaged. This was done for each of the seven files. After this, the acceleration data were normalized with respect to the record at the beginning of the experiment.

The EMG signal (Figs. 3b, 3c) was filtered by a Butterworth filter, with a cut-off frequency of 500 Hz. This filtered signal was rectified and the beginning and the end of each cycle was identified (as the start and the end of the muscle activity region) and marked in the data file. As demonstrated in Fig. 3, the EMG cycle could be easily

Table 1. Mean (± S.D.) physical characteristics and running speeds of the subject groups.

<table>
<thead>
<tr>
<th>Group Type</th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Bodymass (kg)</th>
<th>PETCO2 at 1 min (mmHg)</th>
<th>PETCO2 at 30 min (mmHg)</th>
<th>Speed (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>non fatigue</td>
<td>29.7 ±6.1</td>
<td>175.5 ±9.2</td>
<td>68.7 ±8.5</td>
<td>43.6 ±2.8</td>
<td>42.6 ±2.7</td>
<td>2.75 ±0.48</td>
</tr>
<tr>
<td>fatigue</td>
<td>32.0 ±3.4</td>
<td>172.1 ±3.6</td>
<td>72.3 ±10.0</td>
<td>44.2 ±2.0</td>
<td>40.3 ±2.1</td>
<td>2.76 ±0.29</td>
</tr>
</tbody>
</table>

* PETCO2 was significantly different from values at the first minute of running (p < 0.05).
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Figure 3. Typical record of acceleration and EMG patterns recorded during running. (a) Acceleration of the tibial tuberosity; (b) EMG of quadriceps; (c) EMG of gastrocnemius.

determined by using the foot strike shock signals monitored by the accelerometer. The mean and RMS values for each cycle were calculated and averaged for each subject. The FFT analysis of the signals was also performed to calculate the Power Spectral Density (PSD) of the signal, using the Hanning window. The obtained spectra provided data for the Mean Frequency (MNF) and Median Frequency (MNF) calculations.

Blood samples

Fingertip blood samples (50 µl) were taken before and after each run from ten randomly chosen subjects from both groups (six from the fatigue and four from the non-fatigue group). Blood lactate concentrations were determined using a standard enzymatic method (Boehringer Mannheim).

Statistics

The obtained data were processed via conventional statistical routines available in “MATLAB”, version 4.2b. Differences between results were tested (t-test) and the level of significance was determined at p < 0.05.

Results

Data obtained for each subject were normalized with respect to the initial values of each test. This was done in order to provide means for averaging, since the absolute values of acceleration and EMG data were different for each subject and, therefore, direct averaging would conceal the changes due to fatigue.

Accelerometric data

A summary of the normalized acceleration and PETCO₂ data during the running tests for the fatigue group is presented in Table 2. The maximum amplitude of the acceleration constantly increased with the time. The PETCO₂ level confirms that this group was fatigued toward the end of the tests. Conversely, in the non-fatigue group, there was no significant change in the obtained data (acceleration, PETCO₂ level) with the time, as shown in Table 3.

EMG signals

Calculated mean and median frequencies were in the range of 60-120 Hz, as reported earlier by a number of researchers [8, 15]. A summary of the normalized EMG data for the gastrocnemius muscle during the running tests for the fatigue group is presented in Table 4. There was a decrease in the time domain, but it was not statistically significant. No change was noted in the frequency domain results. The gastrocnemius data for the non-fatigue group presented in Table 5, also do not indicate a significant variation with time. Data for the quadriceps muscle for the fatigue and non-fatigue groups are presented in Tables 6

<table>
<thead>
<tr>
<th>Time</th>
<th>5 min</th>
<th>10 min</th>
<th>15 min</th>
<th>20 min</th>
<th>25 min</th>
<th>30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acceleration</td>
<td>1.17±0.17</td>
<td>1.44±0.30</td>
<td>1.48±0.28</td>
<td>1.49±0.23*</td>
<td>1.50±0.24*</td>
<td>1.62±0.32*</td>
</tr>
<tr>
<td>PETCO₂</td>
<td>1.00±0.02</td>
<td>0.98±0.02</td>
<td>0.96±0.02</td>
<td>0.94±0.03</td>
<td>0.92±0.01*</td>
<td>0.91±0.01*</td>
</tr>
</tbody>
</table>

* Significantly different from the values at Minute-5 (P < 0.05)

<table>
<thead>
<tr>
<th>Time</th>
<th>5 min</th>
<th>10 min</th>
<th>15 min</th>
<th>20 min</th>
<th>25 min</th>
<th>30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acceleration</td>
<td>0.99±0.15</td>
<td>0.99±0.11</td>
<td>0.97±0.18</td>
<td>0.96±0.20</td>
<td>0.97±0.15</td>
<td>0.99±0.14</td>
</tr>
<tr>
<td>PETCO₂</td>
<td>0.99±0.02</td>
<td>1.00±0.04</td>
<td>1.00±0.03</td>
<td>1.00±0.03</td>
<td>1.00±0.03</td>
<td>0.98±0.03</td>
</tr>
</tbody>
</table>
Table 4. Normalized EMG parameters (gastrocnemius) vs time for fatigue group (n = 5) (Mean ± S.D.)

<table>
<thead>
<tr>
<th>Time</th>
<th>10 min</th>
<th>15 min</th>
<th>20 min</th>
<th>25 min</th>
<th>30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.925 ±0.051</td>
<td>0.912 ±0.056</td>
<td>0.885 ±0.092</td>
<td>0.905 ±0.056</td>
<td>0.874 ±0.080</td>
</tr>
<tr>
<td>RMS</td>
<td>0.914 ±0.050</td>
<td>0.905 ±0.051</td>
<td>0.874 ±0.080</td>
<td>0.886 ±0.057</td>
<td>0.860 ±0.080</td>
</tr>
<tr>
<td>Mean frequency</td>
<td>1.050 ±0.053</td>
<td>1.068 ±0.076</td>
<td>1.054 ±0.064</td>
<td>1.076 ±0.083</td>
<td>1.091 ±0.104</td>
</tr>
<tr>
<td>Median frequency</td>
<td>1.064 ±0.066</td>
<td>1.060 ±0.108</td>
<td>1.058 ±0.071</td>
<td>1.074 ±0.112</td>
<td>1.089 ±0.132</td>
</tr>
</tbody>
</table>

Table 5. Normalized EMG parameters (gastrocnemius) vs time for non fatigue group (n - 6) (Mean ± S.D.)

<table>
<thead>
<tr>
<th>Time</th>
<th>10 min</th>
<th>15 min</th>
<th>20 min</th>
<th>25 min</th>
<th>30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.006 ±0.074</td>
<td>0.994 ±0.104</td>
<td>1.011 ±0.123</td>
<td>1.035 ±0.101</td>
<td>0.973 ±0.098</td>
</tr>
<tr>
<td>RMS</td>
<td>1.003 ±0.073</td>
<td>0.989 ±0.099</td>
<td>1.003 ±0.127</td>
<td>1.024 ±0.102</td>
<td>0.966 ±0.113</td>
</tr>
<tr>
<td>Mean frequency</td>
<td>0.995 ±0.043</td>
<td>1.020 ±0.062</td>
<td>1.018 ±0.062</td>
<td>1.095 ±0.181</td>
<td>1.042 ±0.076</td>
</tr>
<tr>
<td>Median frequency</td>
<td>0.988 ±0.050</td>
<td>1.016 ±0.068</td>
<td>1.001 ±0.058</td>
<td>1.057 ±0.076</td>
<td>1.045 ±0.070</td>
</tr>
</tbody>
</table>

Table 6. Normalized EMG parameters (quadriceps) vs time for fatigue group (n = 5) (Mean ± S.D.)

<table>
<thead>
<tr>
<th>Time</th>
<th>10 min</th>
<th>15 min</th>
<th>20 min</th>
<th>25 min</th>
<th>30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.028 ±0.075</td>
<td>1.076 ±0.063</td>
<td>1.085 ±0.062</td>
<td>1.102 ±0.091</td>
<td>1.116 ±0.075</td>
</tr>
<tr>
<td>RMS</td>
<td>1.032 ±0.079</td>
<td>1.085 ±0.072</td>
<td>1.082 ±0.069</td>
<td>1.103 ±0.111</td>
<td>1.112 ±0.086</td>
</tr>
<tr>
<td>Mean frequency</td>
<td>1.020 ±0.067</td>
<td>1.015 ±0.064</td>
<td>1.040 ±0.077</td>
<td>1.021 ±0.042</td>
<td>1.032 ±0.084</td>
</tr>
<tr>
<td>Median frequency</td>
<td>1.037 ±0.053</td>
<td>1.050 ±0.059</td>
<td>1.085 ±0.111</td>
<td>1.069 ±0.081</td>
<td>1.082 ±0.111</td>
</tr>
</tbody>
</table>

Table 7. Normalized EMG parameters (quadriceps) vs time for non fatigue group (n = 6) (Mean ± S.D.)

<table>
<thead>
<tr>
<th>Time</th>
<th>10 min</th>
<th>15 min</th>
<th>20 min</th>
<th>25 min</th>
<th>30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.041 ±0.055</td>
<td>1.014 ±0.056</td>
<td>1.047 ±0.057</td>
<td>1.051 ±0.114</td>
<td>1.143 ±0.129</td>
</tr>
<tr>
<td>RMS</td>
<td>1.043 ±0.054</td>
<td>1.031 ±0.069</td>
<td>1.050 ±0.059</td>
<td>1.056 ±0.112</td>
<td>1.152 ±0.150</td>
</tr>
<tr>
<td>Mean frequency</td>
<td>0.974 ±0.086</td>
<td>0.975 ±0.061</td>
<td>0.994 ±0.080</td>
<td>0.988 ±0.064</td>
<td>1.023 ±0.035</td>
</tr>
<tr>
<td>Median frequency</td>
<td>1.009 ±0.071</td>
<td>1.024 ±0.046</td>
<td>1.056 ±0.081</td>
<td>1.041 ±0.108</td>
<td>1.102 ±0.025</td>
</tr>
</tbody>
</table>

Discussion

This study was aimed at investigating the dynamic loading on the human musculoskeletal system as a function of fatigue. The results presented in this study were obtained while running on a motor driven treadmill. While such a setup simplified data acquisition, it may result that the locomotion patterns differ from the ones corresponding to overground running. The accelerometer for measurement of the axial component of tibial acceleration was attached externally to the tibia. While this may provide somewhat

Blood samples

The average blood lactate concentration for all subjects was 1.9 ± 0.5 mole/liter at the beginning of the test and it increased to 4.6 ± 1.4 after the test, the difference was significant at p < 0.05. There was no significant difference between the blood lactate concentration for members of the fatigue and non fatigue groups.
reduced amplitudes versus the ones measured by an accelerometer attached directly to the bone [16], the bone attachment was not a method of choice in the present group. In this work the obtained data were normalized for each subject with respect to his own initial measurements, thus any errors due to loss of amplitude or high frequency components of the acceleration signal are not significant for the purpose of this study.

The concentration of lactic acid in the blood increased during the test. This increase, however, was mild. It is well known [2] that concentration of lactic acid starts to increase as soon as the load exceeds about 50-60% of the individual's maximal aerobic power. Above this load, lactic acid increase is considerably more dramatic. This confirms that all the subjects from the fatigue and non-fatigue groups were indeed running at 50-60%, i.e., near the aerobic/anaerobic transition region.

The result that the amplitude of the heel strike initiated shock wave will increase with fatigue has been previously predicted [12, 20]. The analysis of our data showed that even though the running speed was set such that the subject will run at the AT in order to cause fatigue, this was not so in all subjects. This may be explained by the fact that in the less experienced subject due to overestimation of the running speed and expected effort the breath rate and oxygen consumption became exaggerated [33].

Bobbert et al., [3] suggested that the fatigue of the runner may lead to modification of the mechanics of the landing phase due to changes in preactivation levels of muscles. It was also found that the transfer of mechanical energy between the eccentric and concentric phases is drastically reduced during muscle fatigue [10]. Such changes may be involved in the development of injuries.

Although not studied in the present work, the possibility that different aged subjects may response to fatigue differently should be explored. Based on the data of 7 subjects aged 23 to 50, Fyhrie et al. [7] hypothesized that the loss of muscle strength and endurance with age prevents forceful muscle contractions when synergistic control of muscles is lost following fatigue. In contrast, muscle strength is maintained in younger people even though coordinated muscle action is impaired, perhaps causing increased impact loading. Their data also suggested that strain rate rather than strain magnitude is implicated in the etiology of stress fracture. The pattern of age dependence in heel strike impact after fatigue may thus explain the observations that stress fractures are age-related and occur more frequently in younger people.

It was expected that the fatigue will be reflected in the decrease in the mean and/or median frequency of the EMG signal, as suggested in a number of previous publications [9, 23]. However, this was not confirmed by the obtained data here. The more detailed analysis reveals that all conclusions on the decrease of the median frequency were obtained at the close to maximum voluntary contraction (MVC) conditions in static tests [8, 24] or during high load dynamic tests on the exercise bicycle [9]. It was found that the decrease in the load level significantly reduced the correlation between the mean power frequency and the subjective muscle fatigue [25]. Repetitive loading during long time span did not introduce changes in the power spectral characteristics of surface EMG [19] as well as low level of the load of the knee extensors [9]. It may be concluded that the frequency shift can not be used as a valid indicator of the fatigue of muscles subjected to a relatively low level, intermittent load. This may be also supported by the measurements of the recovery time constant of the resting membrane potential of mouse soleus. It was found [11] that the recovery time was about one 1 min after 960 stimuli.

Failure of the EMG measurements to indicate fatigue raises the question what fatigue are we talking about? The data obtained tend to point that it is a deterioration of coordinated muscle action with loss of synergistic control of muscles, rather than failure of the individual muscle. Therefore, we may conclude from this study that PETCO2 measurements may reflect this stage better than EMG.

The obtained results show that the amplitude of the acceleration amplitude is steadily increasing with running time in the fatigue group. Thus, one may conclude that the human musculoskeletal system becomes less capable to handle the heel strike induced shock waves when there is a significant fatigue. This condition may promote development of osteoarthritis. Therefore it may be advantageous to assure that the majority of training and exercise is performed in a way that does not lead to severe fatigue.

Acknowledgement

This work was supported in part by the Segal Foundation and by the Fund for the Promotion of Research at the Technion. One of the co-authors (AV) is grateful to the Henry Goldberg Lectureship Fund for the support during his sabbatical leave to Technion, Haifa where this work was performed. The co-author (OV) was supported by the Center for Absorption in Science, Ministry of Immigrant Absorption, State of Israel.

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