Shock Transmission and Fatigue in Human Running

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The goal of this research was to analyze the effects of fatigue on the shock waves generated by foot strike. Twenty-two subjects were instrumented with an externally attached, lightweight accelerometer placed over the tibial tuberosity. The subjects ran on a treadmill for 30 min at a speed near their anaerobic threshold. Fatigue was established when the end-tidal CO₂ pressure decreased. The results indicated that approximately half of the subjects reached the fatigue state toward the end of the test. Whenever fatigue occurred, the peak acceleration was found to increase. It was thus concluded that there is a clear association between fatigue and increased heel strike–induced shock waves. These results have a significant implication for the etiology of running injuries, since shock wave attenuation has been previously reported to play an important role in preventing such injuries.

Key words: accelerometry, end-tidal CO₂ pressure, fatigue, running, shock absorption

Running is the exercise of choice for millions of people all over the world and across the age spectrum. One of the main reasons for its popularity stems from its simplicity. However, running also carries the risk of increased musculoskeletal injuries. These injuries may be caused by a number of factors that lead directly to increased loading, including shoe design (Cook, Kester, Brunet, & Haddad, 1985), variations in running surfaces (Freehery, 1986), and fatigue (Dickinson, Cook, & Leinhardt, 1985).

One of the important functions of the human musculoskeletal system is to attenuate and dissipate shock waves initiated with foot–ground contact. Those shock waves are initiated by most types of motion, such as walking, running, and stair climbing. Radin et al. (1973) showed that the high impact forces associated with the heel strike may damage articular cartilage. Radin, Paul, and Rose (1975) indicated that osteoarthritis results mainly from poorly handled mechanical load, rather than from a disease. Voloshin and Wosk (1982) showed that low back pain may result from overloading the human musculoskeletal system and reducing its shock-absorbing capacity. Collins and Whittle (1989a) reported a relationship between heel strike impulsive loads and injuries.
Heel strike–initiated shock waves are measured mainly by skin-mounted accelerometers (Clarke, Cooper, Hamill, & Clark, 1985; Light, MacLellan, & Kleeneman, 1980; Loy & Voloshin, 1991; Wosk & Voloshin, 1981). These and other authors used various methods of accelerometer-to-skin attachment, mainly by strapping the accelerometer to bony prominences (e.g., tibial tuberosity, forehead, and sacrum).

It is well known that fatigue of the muscles affects not only their performance but other related functions as well. Force plate measurements have suggested a possible effect of fatigue on heel strike transients (Dickinson et al., 1985). Muscles lower the bending stress on bone and attenuate the peak dynamic loads that can damage musculoskeletal tissues (Jefferson, Collins, Whittle, Radin, & O’Connor, 1990; Radin, 1986).

The effect of stride rate variations on musculoskeletal loading has also been studied. Martin and Marsh (1992) reported that a decreased stride rate in walking is accompanied by an increased vertical force at touchdown. Clarke et al. (1985) showed that in running, decreasing the stride rate increased peak shank decelerations immediately following foot contact.

A goal of the present study was to analyze the effect of fatigue on the ability of the human musculoskeletal system to attenuate heel strike–generated shock waves. Fatigue hampers the ability of the human musculoskeletal system to protect itself from overloading due to heel strike–generated shock waves. Loss of protection may be manifested in an increased shock wave amplitude as measured on the tibial tuberosity. Also, to accommodate modifications in the running pattern, there may be a change in stride rate. Understanding the influence of fatigue on the magnitude of dynamic loading on the human musculoskeletal system will enhance shoe design and floor design, allow the development of proper training procedures and exercises, and will reduce damage to the musculoskeletal tissues.

**Methods**

**Subjects and Experimental Protocol**

Twenty-two male subjects, age (mean ± SD) 30.8 ± 5.1 years, height 173.9 ± 7.3 cm, and body mass 70.4 ± 9.2 kg, volunteered to participate in this study. All subjects were in excellent health and practiced calisthenics at least twice a week, although they were not elite athletes. They had no previous histories of muscle weakness, neurological disease, or drug therapy. Each subject provided informal consent according to the local ethical committee’s guidelines. To ensure uniformity of the testing conditions, all subjects were provided with the same type of running shoes by the same manufacturer.

Running tests were performed on a treadmill (Quinton Q55) so we could accurately monitor 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 respired 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 (\(\dot{V}O_2\)), minute ventilation (\(V_E\)), carbon dioxide production (\(\dot{V}CO_2\)), end-tidal carbon dioxide pressure (\(PETCO_2\)), ventilatory equivalent for oxygen (\(V_E/\dot{V}O_2\)), and ventilatory equivalent for carbon dioxide (\(V_E/\dot{V}CO_2\)) were calculated as an average of the breath-by-breath data during 30 s.

Before beginning the experiment, each individual was subjected to an incremental load on a treadmill with an increase in speed to determine his anaerobic threshold (AT).
Anaerobic threshold was determined noninvasively as the common onset of increase of the ventilatory equivalent for oxygen ($V_{\text{E}}/VO_2$) and ventilatory equivalent for carbon dioxide ($V_{\text{E}}/VCO_2$) (Smith & O'Donnel, 1984). Running load consisted of 30-s runs at incremental speeds of 0.22 m/s beginning at 1 m/s and continuing until the point of AT determination.

The running exercise test was performed for 30 min at a speed corresponding to the AT level. However, it is well known that if a subject is exercised long enough at a work rate corresponding to the absolute AT, PETCO$_2$ will decrease (Wasserman, Whipp, Koyal, & Beaver, 1973). Thus, decreased PETCO$_2$ indicates a decrease in performance, leading to fatigue (Muza, Lee, Wiley, McDonald, & Zechaman, 1983). The distinction between the fatigue and nonfatigue groups emerges from the PETCO$_2$ measurements. Thus, at the end of the 30 min of running on the treadmill, all subjects were divided into two groups according to PETCO$_2$. The pretest of ventilatory equivalent versus speed of running determined only the vicinity of the AT speed: As became evident later on, in some subjects (fatigue group) the pretest speed was above the breakpoint and in the others (nonfatigue group) it was below the breakpoint. Age, height, body mass, and running speed of the groups are shown in Table 1. Several subjects performed the test twice, on different days, to assess the repeatability of the data. Data obtained for each subject were normalized with respect to his own performance at the beginning of the test. This was done to provide means for averaging, since the absolute values of acceleration and stride rate were different for each subject and, therefore, direct averaging would conceal the changes due to fatigue.

Each subject was instrumented with a lightweight (2.3 g) accelerometer attached above the tibial tuberosity. The uniaxial accelerometer was aligned along the longitudinal axis of the tibia to provide axial components of tibial acceleration. The accelerometer was attached externally to the tibial tuberosity by a metal holder strapped on the skin. Such an attachment, as proven in earlier works (Kim, Voloshin, Johnson, & Simkin, 1993), can accurately measure shock wave amplitude.

### Instrumentation

A lightweight accelerometer (PCB, Type A303) was used to measure acceleration patterns due to the heel strike. The accelerometer was connected to a power unit (PCB, Model...
480B) that provided an analog signal. The signal was fed to a PC-based data acquisition system. During the test, acceleration data were acquired for 30 s every 5 min by an A/D converter (CODAS, Data Instrumentations, Inc., Akron, OH) with a sampling rate of 1667 Hz per channel. This setting provided information on about 30 heel strikes over a 20-s range. The data were stored for off-line processing. To minimize possible gait modifications, the subject was not aware of when exactly the data were acquired.

**Data Acquisition and Analysis**

For each 30-min test, seven files of acceleration data were generated. 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 used the given running speed and the location of the first heel strike to detect all consequent occurrences of heel strike. Since there was always a possibility of “bad” data (e.g., a subject stumbled or misplaced his foot), all results were shown on the monitor and confirmed by an operator.

The main variables were the maximum amplitudes of the accelerations recorded on the tibial tuberosity, stride rate (Figure 1), and PETCO₂ (Figure 2). For each subject, the maximum accelerations and stride rates recorded in one file were measured and averaged. This was done for each of the seven files. After this, the data were normalized with respect to the record at the beginning of the experiment.

**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 $p < .05$.

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**Figure 1** — Typical acceleration pattern recorded on the tibial tuberosity during running. A = maximum acceleration; T = time length of the stride. For each test, the extraction procedure of multiple stride values of these variables is as detailed in the text (Data Acquisition and Analysis).
Results

The groups of subjects were as follows (Figure 2). Ten subjects whose PETCO₂ significantly decreased ($p < .05$) at the end of the 30-min run were defined as the fatigue group. Twelve subjects who did not show a significant change in the amount of PETCO₂ at the end of the test were defined as the nonfatigue group. The normalized PETCO₂ are summarized in Table 1. The average speed for the fatigue group (2.76 ± 0.29 m/s) was not significantly different from the nonfatigue group (2.75 ± 0.48 m/s) (Table 1).

A typical performance of a subject from the fatigue group is illustrated in Figure 3, a–c. The maximum amplitude of the acceleration constantly increased with time (Figure 3a), while the stride rate was decreasing (Figure 3b). The PETCO₂ level (Figure 3c) confirms that this subject was fatigued toward the end of the experiment. Conversely, for a subject in the nonfatigue group, there was no significant change in the obtained data (acceleration, stride rate, PETCO₂ level) with time (Figure 3, d–f).

Three subjects repeated the test at various days and running speeds. On each occasion, the speed of running was set as described earlier in the Subjects and Experimental Protocol section; however, due to changes in the subjects’ reactions to the experimental setup, slightly different speed levels corresponding to the AT level were obtained on different days. Raw data for a subject who ran twice at different speeds are shown in Figure 4. Both runs qualified him as a member of the nonfatigue group and showed acceleration patterns that were not significantly different from each other. Another subject also ran twice; however, both tests placed him in the fatigue group. The absolute values of the acceleration appear to depend on running speed (Figure 5), but both sets of data have the same pattern: an increase of acceleration amplitude with time. Another subject ran twice: one run placed him in the nonfatigue group, while the second placed him in the fatigue group (Figure 6), as was his definitive inclusion. The acceleration data followed the same pattern: no significant change versus time for a subject in the nonfatigue group and increase in the acceleration amplitude in the fatigue group. Normalized stride rates
Figure 3 — Typical averaged raw results of 30 foot strikes for (a–c) fatigue group (Subject I.G.) and (d–f) nonfatigue group (Subject B.R.). (a, d) maximum amplitude of the acceleration (mean $\pm$ SD); (b, e) stride rate (mean $\pm$ SD); (c, f) PETCO$_2$ (one reading was obtained every 30 s). Vertical bars indicate standard deviations. *Significant difference ($p < .05$) with respect to the data at the beginning of running.

Figure 4 — Average raw results of acceleration change (mean $\pm$ SD) versus time for 30 foot strikes of Subject O.R. running at speeds of 2.16 (dashed line) and 2.24 (solid line) m/s. Both times he was in the nonfatigue group (mean $\pm$ SD). Vertical bars indicate standard deviations.
Figure 5 — Average raw results of acceleration change (mean ± SD) versus time for 30 foot strikes of Subject O.L., running at speeds of 2.50 (dashed line) and 2.91 (solid line) m/s. Both times he was in the fatigue group. Vertical bars indicate standard deviations. *Significant difference (p < .05) with respect to the data at the beginning of running.

Figure 6 — Average raw results of acceleration change (mean ± SD) versus time for 30 foot strikes of Subject P.B., running at speeds of 2.50 and 3.13 m/s. Running at 2.50 m/s (dashed line) placed him the nonfatigue group, while higher speed (solid line) placed him in the fatigue group, as was his definitive inclusion. Vertical bars indicate standard deviations. *Significant difference (p < .05) with respect to the data at the beginning of running.

(Figure 7) show a significant (p < .05) decrease toward the end of the test for the fatigue group, while no change was detected for the nonfatigue group. Normalized maximum accelerations (Figure 8) also did not change significantly for the nonfatigue group; however, significant increase was noted for the fatigue group, as summarized in Table 1.
Figure 7 — Mean stride rate (±SD) for both groups (solid line = fatigue group, \(n = 10\); dashed line = nonfatigue group, \(n = 12\)). Vertical bars indicate standard deviations. *Significant difference \((p < .05)\) with respect to the data at the beginning of running.

Discussion

This study was aimed at investigating dynamic loading on the human musculoskeletal system as a function of fatigue. The results presented in this study were obtained while subjects ran on a motor-driven treadmill. While such a setup simplified data acquisition, the locomotion patterns obtained may differ from patterns for overground running. The runners in the present study were constrained to run at a constant speed, regardless of
whether they were fatigued or not. This may not be the case in overground running: When fatigue begins, runners may slow down as a protective means. The result could be moving away from the state of fatigue, in which case the acceleration data might not increase. According to the results presented in this study, for the acceleration data to increase, fatigue should be present. Thus, the results of this study can be extrapolated to overground running if fatigue indeed prevails. Also, most running injuries in overground running are lower extremity injuries, with a predominance of knee injuries (van Mechelen, 1992), and our results indicating an increase in acceleration data in the tibial tuberosity therefore tend to support this extrapolation.

The accelerometer for measuring the axial component of tibial acceleration was attached externally to the tibia. While this may provide somewhat reduced amplitudes versus the ones measured by an accelerometer attached directly to the bone (Lafortune, 1991), 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 this study.

The subjects in this study (both fatigue and nonfatigue groups) were on the threshold of aerobic/anaerobic transition. All subjects were nonprofessional athletes/runners; however, they were all able-bodied individuals who practiced calisthenics at least twice a week. This is why we designed the testing protocols so as not to exhaust the subjects, as would be required to sharply define the AT breakpoint, and used relatively low running speeds as compared to those of elite runners.

In our protocol, only near AT (and not absolute AT) could be determined. To determine AT precisely and unequivocally, more data points are essential in the ventilatory equivalent versus running speed curve. The reason is that this curve is often unstable in the transition between the aerobic and anaerobic zones. Thus, the usual ventilatory equivalent pretest (as also used in our study) is unreliable to sharply define the breakpoint between the two zones (Gaesser & Poole, 1986; Poole & Gaesser, 1985). This is unless an alternative method, such as the V-slope method, is adopted to more accurately detect the onset of increase of the ventilatory equivalent (Orr, Green, Hughson, & Bennett, 1982; Schneider, Phillips, & Stoffolano, 1993). However, application of this method involves a physically demanding effort leading to exhaustion, since it requires that the pretest be extended to running speeds far above the AT speed. This was beyond the protocols approved for our subjects by the ethical committee.

That the amplitude of heel strike–initiated shock wave will increase with fatigue has been previously predicted (Milgrom et al., 1992). However, Milgrom et al. used no measure for fatigue, and so it was expected that all subjects would behave in a similar manner. Our data analysis showed that even though the PETCO₂ suggests that the particular running speed will cause fatigue in a continued effort, this was not so in all subjects. This may be because in the less experienced subject, breath rate and oxygen consumption during AT determination became exaggerated due to overestimation of the running speed and expected effort. Indeed, some of these subjects were tested for a second time after a few days. On this second occasion, the speed as set according to the AT level happened to be higher, and the fatigue state (Figure 6) was achieved. This time the same subject was classified as a member of the fatigue group.

PETCO₂ is considered an established and reliable parameter for determining fatigue: (a) It is more sensitive than lactic acid, in particular during running 30 min (the running time in our tests) (Liu, Vargas, Stanbury, Sasse, & Light, 1995; McLellan & Cheung, 1992), and (b) it is considered a reliable parameter for determining AT (Wasserman, 1987;
Wasserman et al., 1973). Thus, the distinction between the two groups emerges from the PETCO₂ measurements, dividing the subjects into fatigue and nonfatigue groups. The pretest of ventilatory equivalent versus speed determines only the vicinity of the AT speed: In some of the subjects (fatigue group) the pretest speed was the absolute AT speed, and in the others (nonfatigue group) pretest speed was below absolute AT speed.

The results (Figures 4–6) indicated that increases in acceleration magnitude with the increase in running speed may have been expected. Indirect support for this can be found in the work of Collins and Whittle (1989b), who showed that the loading rate of the lower limb is directly and highly correlated with walking speed, and in the work of Nigg, Bahlsten, Luethi, and Stokes (1987) and Frederick, Hagy, and Mann (1981), who showed that the vertical impact force increased with increasing running velocity.

A decrease in the stride rate (in the fatigue group) with fatigue was found to be directly associated with an increase in the amplitude of the heel strike–initiated shock wave. Similar results were obtained by Clark et al. (1985); however, the decrease in the stride rate was forced by those researchers on the runner rather than being changed by fatigue. They suggested that a runner should increase his stride rate in order to minimize joint forces related to impact loading. This advice was not followed by the runners in the present study, as is obvious from the obtained results. One of the reasons may be that the fatigued muscles cannot support running at the “optimal” stride rate, even though the runners’ energy costs increase as they alter their stride rates away from their normal rates (Cavanagh & Williams, 1982). Bobbert, Yeadon, and Nigg (1992) suggested that fatigue of the runner may lead to modification of landing phase mechanics 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 (Gollhofer, Komi, Miyashita, & Aura, 1987). Such changes may be involved in the development of injuries.

The obtained results show that acceleration amplitude steadily increased with running time in the fatigue group and that there was a clear association between fatigue (as revealed by PETCO₂) and shock waves (as revealed by the acceleration). However, we cannot definitely conclude whether one resulted from the other or whether they both resulted from a third factor. We may conclude, though, that the human musculoskeletal system becomes less capable of handling heel strike–induced shock waves when the muscles are significantly fatigued. This condition may promote development of injuries (Collins & Whittle, 1989a). The results presented here have a significant implication for the etiology of running injuries. Therefore, it may be advantageous to ensure that the majority of training and exercise is performed in a way that does not lead to severe fatigue.

References


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