RUNNING KINEMATICS AND SHOCK ABSORPTION DO NOT CHANGE AFTER BRIEF EXHAUSTIVE RUNNING

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ABSTRACT

Abt, JP, Sell, TC, Chu, Y, Lovalekar, M, Burdett, RG, and Lephart, SM. Running kinematics and shock absorption do not change after brief exhaustive running. *J Strength Cond Res* 25(X): 000–000, 2011—Because of the nature of running, the forces encountered require a proper coordination of joint action of the lower extremity to dissipate the ground reaction forces and accelerations through the kinetic chain. Running-related muscle fatigue may reduce the shock absorbing capacity of the lower extremity and alter running kinematics. The purpose of this study was to determine if a bout of exhaustive running at a physiologically determined high intensity, changes running kinematics, impact accelerations, and alters shock attenuating capabilities. It was hypothesized that as a result of fatigue induced by an exhaustive run, running kinematics, impact accelerations at the head and shank, acceleration reduction, and shock attenuation would change. A within-subject, repeated-measures design was used for this study. Twelve healthy, competitive male and female distance runners participated. Subjects performed 2 testing sessions consisting of a $\dot{V}O_2$max treadmill protocol to determine the heart rate at ventilatory threshold and a fatigue-inducing running bout at the identified ventilatory threshold heart rate. Kinematic data included knee flexion, pronation, time to maximum knee flexion, and time to maximum pronation. Acceleration data included shank acceleration, head acceleration, and shock attenuation. No significant differences resulted for the kinematic or acceleration variables. Although the results of this study do not support the original hypotheses, the influence of running fatigue on kinematics and accelerations remains inconclusive. Future research is necessary to examine fatigue-induced changes in running kinematics and accelerations and to determine the threshold at which point the changes may occur.

KEY WORDS pronation, knee flexion, fatigue, cardiovascular, running gait

INTRODUCTION

The popularity of running has increased dramatically over the last few decades as a result of the associated health benefits. Unfortunately, a parallel rise in the rate of running-related overuse musculoskeletal injuries has occurred. The risk of injury accompanied with running can be associated with both the repetitive nature of running and the impacts the body suffers during running. The repetitive nature of running is obvious: An average individual running 20 $\text{miles}\cdot\text{wk}^{-1}$ for 1 year will endure approximately 1.3 million impacts to the lower extremity (4). The impact ground reaction forces experienced during running have been estimated between 1.5 and 2 body weights (18) and impact accelerations between 6 and $8G$ (4). Such impacts are high but not extreme. In addition, epidemiological studies have estimated overuse injuries of the lower extremity in recreational runners between 30 and 70% during any given year (25), whereas acute injuries are not as frequent (11). It is not surprising that more research has focused on the repetitive aspect of running as a contributor to injury. As a result, previous studies have used a long-duration, moderate-intensity marathon protocol to investigate the potential kinematic changes because of fatigue and the underlying mechanisms (20).

However, overuse running injuries result from the exposure to repetitive forces, which produce a combined fatigue effect and ultimately inhibit the remodeling and repair process (7). According to such a mechanism, the key element contributing to overuse running injuries is fatigue and the repetitive nature of running is just a potential factor that can induce fatigue. The repetitive nature of running requires efficient movement patterns to prevent injury by absorbing the impact forces (26), reducing impact accelerations, and promoting proper coordination between muscle activity and the responding joint motions (8,21). Repetitive impact in similar but relatively lower-intensity activities, such as walking, is

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sufficient to induce short-term structural changes in the tendinous tissues (2). Running-related muscle fatigue can impair the efficiency of movements, reduce the shock absorbing capacity of the lower extremity (5,27), alter running kinematics (27), and increase the risk of injury.

With different running durations and intensities, runners may experience diverse types of fatigue, because the stress placed on different systems in the body can vary. Although most previous studies used a marathon protocol, running at a higher intensity exhausts runners faster and may fatigue the cardiovascular system before the neuromuscular response is compromised. Because fatigue, instead of high repetitions, is the key element inducing kinematic and shock-attenuation changes, we may observe some changes in such variables that can be related to increased risk of running injuries after a brief but high-intensity run. Derrick et al. (4) applied a higher-intensity protocol, having subjects running at the speed of their maximum-effort 3,200-m run. They found altered impact accelerations and kinematics, and reached the conclusion that kinematic changes after a fatigued running bout might be a compensatory mechanism to reduce the likelihood of injury and not as a result of fatigue. Derrick et al. (4), however, arbitrarily chose the exercise intensity, which therefore was hard to interpret in physiological terms. An objective intensity criterion based on the runner's physiological response would help to relate the potential effects of fatigue to the stress placed on the cardiovascular system. Morgan et al. (17) used 90% $\dot{V}O_2$max as the running intensity but focused on the fatigue effects 1 or more days after running instead of immediate changes. In the case of brief and high-intensity run, the runners' physiological and kinematic responses may vary and could be explained with different mechanisms than in a marathon run. Nicol et al. (20) reported that functional performance deficits after marathon running were the result of inhibited neural input to the muscle and deterioration of contractile muscle efficiency but failed to establish the relationship between kinematics and fatigue. Kyrolainen et al. (13) noted performance adaptations as a compensatory mechanism to account for impaired neuromuscular function during the run. Although they noticed increased stride frequency and decreased stride length, the decreased running economy after fatigue could not be explained by the changed kinematics. As such, the theories remain conflicting explaining kinematic changes after a marathon run in the context of overuse running injuries. The kinematics or shock-attenuation changes and their implications on fatigue and injuries after a brief, high-intensity run are still unclear and require further study.

Therefore, the purpose of this study was to determine if a bout of exhaustive running at a physiologically determined high-intensity changes running kinematics, impact accelerations, and alters shock attenuating capabilities. It was hypothesized that as a result of fatigue induced by such exhaustive run, running kinematics, impact accelerations at the head and shank, acceleration reduction, and shock attenuation would change.

METHODS

Experimental Approach to the Problem

A within-subject repeated measures design was used to assess changes in running kinematics and accelerations after an exhaustive run. Subjects reported for 2 testing sessions ($\dot{V}O_2$max session, Exhaustive run session) separated by 1 week. The $\dot{V}O_2$max session was held to determine the ventilatory threshold of the subject and the corresponding pace for the exhaustive run. The exhaustive run session included pre and postexhaustion run kinematic and acceleration measurements. The kinematic-dependent measures were knee flexion and pronation and time to maximum knee flexion and time to maximum pronation. The accelerometer-dependent variables are peak shank and head accelerations, percent reduction in accelerations, and shock attenuation.

Subjects

Twelve healthy, competitive male and female distance runners (age: 24.5 ± 4.1 years, height: 1.74 ± 0.09 m, mass: 65.2 ± 9.8 kg, $\dot{V}O_2$max: 68.9 ± 7.6 $ml\cdot kg^{-1}\cdot min^{-1}$) participated in this study. Subject eligibility was determined by a history of running for at least 3 years, minimum training mileage of 48 $km\cdot wk^{-1}$, minimum pace of 10.7 $km\cdot h^{-1}$, and no history of injury within the previous 3 months that limited training. This study received University Institutional Review Board approval (IRB #0303039), and all subjects provided written informed consent before participation.

Procedures

Day 1. After completion of informed consent, subjects performed a maximal oxygen uptake test to determine the speed associated with ventilatory threshold (22,28). Subjects were fitted with the K4b^2 portable metabolic system (K4b^2, COSMED USA Inc, Chicago, IL, USA) and heart rate monitor (Polar USA, Lake Success, NY, USA) to assess oxygen consumption and heart rate during a maximal oxygen uptake test. The K4b^2 contained individual oxygen and carbon dioxide analyzers to assess cardiovascular and pulmonary function during physical activity on a breath-by-breath basis.

A Modified Astrand protocol was used for this study. The protocol consisted of a 5-minute warm-up at a comfortable self-selected pace. An initial 3-minute workload at 0% grade was self-selected by the subjects. The adopted speed permitted the subject to perform 12–15 minutes of exercise before exhaustion and was approximated from the subject's daily training 45-minute run time. The incline was increased 2.5% every 2 minutes while the speed remained constant. The subjects were instructed to continue running until exhaustion (defined as the inability to continue the test because of cardiovascular or peripheral inhibition). Heart rate and $\dot{V}O_2$ were monitored continuously throughout the test. The test was self-terminated by the subject and verified for (a) a plateau in $\dot{V}O_2$ is achieved with increasing intensity, (b) respiratory exchange ratio is >1.1, and (c) heart rate is within 95% of heart rate max (defined as 220 − age).

The ventilatory threshold was determined by plotting ventilation ($L \cdot min^{-1}$) against $\dot{V}O_2$ ($ml \cdot min^{-1} \cdot kg^{-1}$) and heart rate ($b \cdot min^{-1}$) against $\dot{V}O_2$ ($ml \cdot min^{-1} \cdot kg^{-1}$) (1). The ventilation–$\dot{V}O_2$ plot was first split into thirds with the upper 66% of the trial and lower 66% of the trial being plotted independently. Linear regression lines were independently applied to the upper and lower portions of the ventilation–$\dot{V}O_2$ plots. A vertical line was then drawn from the *x*-axis through the heart rate–$\dot{V}O_2$ plot to determine the corresponding ventilatory threshold heart rate (HRmax: 186.7 ± 10.5 $b \cdot min^{-1}$, Ventilatory Threshold: 90.2 ± 2.5% of HRmax).

Day 2. One week after the maximal oxygen uptake test, subjects reported to perform an exhaustive run. Raw coordinate data were collected using the Peak Motus 3D Motion Analysis System (Software Version 7.2.3, Vicon Motion Systems Inc, Centennial, CO, USA) interfaced with 8, high-speed optical cameras (Pulnix Industrial Product Division, Sunnyvale, CA, USA). Coordinate data were collected at 120 Hz. The accuracy of this system had been determined as 4.68 ± 1.02 mm for linear measurement and 0.56 ± 0.40° for angular measurement (6). Spherical reflective markers (diameter 0.019 m, 0.0125 m) were placed unilaterally on the dominant limb at according to Kadaba et al. (9).

Two 21 g, triaxial accelerometer modules (Model 2422-010, Silicon Designs, Inc., Issaquah, WA, USA) were used to measure shank acceleration, head acceleration, % acceleration absorption, and shock attenuation. The shank accelerometer was also used to identify the discrete point of impact during the running trials. The accelerometer modules contained 3 orthogonal accelerometers within an anodized aluminum case and an integrated circuit sense amplifier. The accelerometers provided analog voltage signals with a full scale acceleration of $\pm 25G$. The accelerometers were fixed to the tibial flare and center of the forehead with a double sided disc, prewrap, and tape to prevent skin movement and acceleration artifacts that would interfere with the shank and head accelerations. Acceleration data were collected at 1,200 Hz.

Subjects performed a 5-minute accommodation running warm-up before data collection. After the warm-up, the treadmill pace was elevated until the heart rate that corresponded with the ventilatory threshold (HR_{VT}: 168.7 ± 11.7 $b \cdot min^{-1}$). The pace was 3.3 ± 0.4 $m \cdot s^{-1}$ and was kept constant throughout the testing. The heart rate response was verified for 1 minute before initiating data collection. Five-second trials were collected every minute throughout the test. Treadmill inclination was set at 0% during the data collection procedures. Subjects wore their current training shoes to simulate a functional running situation (7). The exhaustive run was terminated when subjects felt they were not able to continue. The test–retest reliability of this testing procedure was demonstrated with an intraclass correlation coefficient (ICC) and *SEM* variables (ICC: 0.78–0.85, *SEM*: 1.5–1.7°) and accelerometer variables (ICC: 0.84–0.91, *SEM*: 0.16–0.39*G*).

Data Reduction

Joint kinematics of the ankle and knee were calculated based on raw coordinate data. A custom designed MATLAB program (MATLAB 6, The Mathworks Inc., Natick, MA, USA) processed and filtered the raw 3D coordinate data and calculated the dependent variables. Frontal plane and sagittal plane kinematic data were filtered with a fourth order, zero lag Butterworth filter with a cutoff frequency of 16 Hz (24). The raw voltage collected for each trial from the shank and head accelerometers was converted to acceleration (*G*), based on the authenticated conversion formulas provided by the manufacture. Initial contact was identified by the minimum amplitude before the peak amplitude of the acceleration data. Accelerometer and kinematic data were time synchronized within the Peak Motus software. Knee flexion and ankle pronation were calculated as the maximum values following initial contact with the time to each variable recorded. Accelerometer data included peak shank and head accelerations and percent reduction in the time domain and shock attenuation in the frequency domain. Peak shank and head accelerations are the maximum acceleration values after initial contact recorded at the shank and head accelerometers, respectively. Acceleration reduction is the percent reduction in acceleration values between the shank and head. Shock attenuation is the change in the power spectral densities between the head and the shank, calculated with the formula described in Derrick et al. (4)

Statistical Analyses

Differences in dependent variables were assessed with SPSS 11.5 (SPSS Inc., Chicago, IL, USA). Dependent variables were calculated for the 2 5-second trials at $time_0$ and $time_{100\%}$ of the exhaustive run. Dependent variables were calculated for every step captured and then averaged within each 5-second trial. Dependent *t*-tests were used to determine differences in maximum ankle pronation, maximum knee flexion, time to maximum ankle pronation, time to maximum knee flexion, peak shank and head accelerations, percent reduction in accelerations, and shock attenuation, between the initial and final phases of the exhaustive run. Statistical significance was determined at $p \leq 0.05$. A power analysis based on previous data (4,15) demonstrated that 12 subjects were required to achieve a statistical power of 0.80 at this significance level.

RESULTS

The mean time to termination during the exhaustive run was 17.8 ± 5.7 minutes. Kinematic and acceleration variables

Table 1. Summary of kinematic and acceleration variables before and after the exhaustive run.*

	Prefatigue	Postfatigue	*p* Value
Maximum knee flexion (°)	49.3 ± 6.6	49.2 ± 6.2	0.48
Maximum ankle pronation (°)	9.2 ± 5.1	9.7 ± 6.2	0.36
Time to max knee flexion (ms)	106.1 ± 22.7	117.7 ± 20.4	0.13
Time to max ankle pronation (ms)	102.9 ± 21.0	109.9 ± 21.0	0.27
Peak shank acceleration (*G*)	7.5 ± 1.1	7.7 ± 1.3	0.19
Peak head acceleration (*G*)	2.7 ± 0.4	2.8 ± 0.5	0.22
Acceleration reduction (%)	63.2 ± 7.9	62.9 ± 9.6	0.42
Shock attenuation (db)	−14.2 ± 3.7	−13.7 ± 3.1	0.18

*Values are given as mean ± *SD*.

before (prefatigue) and after the exhaustive run (postfatigue) are presented in Table 1. No significant prefatigue, postfatigue differences existed for these variables.

Discussion

The purpose of this study was to determine if a bout of exhaustive running at a physiologically determined high-intensity changes running kinematics, impact accelerations, and alters shock attenuating capabilities. Repetitive impact forces are encountered during running and can lead to overuse injury. Approximately 70–80% of the impact forces during running are absorbed by the knee (10). Dissipation of such impact forces may be critical to prevent overuse injuries as impact forces are distributed throughout the body. To dissipate such forces, a proper coordination of joint action of the lower extremity is required. Such coordination is achieved with the combination of muscle stabilization and joint movement (4,14). Ankle (subtalar joint) pronation and the accompanying tibial internal rotation further reduce the magnitude of the impact forces as they are distributed throughout the kinetic chain over a longer period of time (3,10,23). Fatigue may alter the joint actions and neuromuscular coordination and therefore reduce the efficiency of impact dissipation. It is also possible that the altered joint actions are compensatory adjustments in response to decreased capacity of impact dissipation following fatigue. The mechanism by which impact dissipation occurs may also contribute to the development of overuse injuries as the body adjusts to reduce the impact. Greater muscle activation may have assisted in maintaining the accelerations experienced during impact despite the onset of fatigue. The onset of fatigue may further increase the magnitude of stress placed on the soft tissue structures in an effort to attenuate the impacts. With further accumulated fatigue near exhaustion, the kinematic compensation would not be able to keep the impact dissipation in control. As results, we may expect to observe altered kinematics and accelerations with fatigue at the exhaustive level. However, the hypotheses of this study were not supported, because no changes were demonstrated after the exhaustive run for any kinematic or acceleration variables.

Contrary to our results, significant differences following an exhaustive run were found by Derrick et al. (4). Compared across the total duration of their exhaustive run, these variables were only different between the start and the finish, indicating that the change in the dependent variables occurred as the fatigue became more prominent, probably near exhaustion. For kinematics, greater maximum knee flexion and greater maximum ankle pronation were observed after an exhaustive run. They also found increased shank acceleration and increased acceleration reduction, whereas head acceleration and shock attenuation remained unchanged (4). The increased acceleration reduction, according to Derrick et al. (4), was likely a result of compensatory kinematic adaptations that resulted. With the knee angle shifted toward more flexion and the knee angular velocity increased after fatigue, it is possible that the knee joint provided less resistance against impacts with decreased stiffness. Decreased joint stiffness following fatigue was described in the previous literature (5,12). The increased ankle pronation may be another sign of such compensation (3,10,23). Mercer et al. (14) also evaluated changes in shock attenuation and impact accelerations during fatigued running. Unlike Derrick et al. (4), no significant differences were demonstrated in any of their collected variables. The lack of changes within shank accelerations, head accelerations, and shock attenuation within this study is consistent with the findings of Mercer et al. (14) and contrary to Derrick et al. (4).

The inconsistent results among these studies may be attributed to the different running protocol for fatigue and data collection. Mercer et al. (14) used a graded running during which the speed started from 1.3 $m \cdot s^{-1}$ with 0.22-$m \cdot s^{-1}$ increment every minute and the grade increased from 3 to 7.5%. They, however, collected their data in 2 separated 3.8 $m \cdot s^{-1}$, 3- to 5-minute run before and after the graded running. Similarly, Kyrolainen et al. (13) chose a marathon-run model at the self-selected pace for fatigue but several separated 3.82 ± 0.33 $m\cdot s^{-1}$ running trials were used before, during, and after the marathon run for data collection. Derrick et al. (4) used each subject's average speed measured in a 3,200-m maximum-effort run as the speed of the exhaustive run and collected data at the beginning, at the middle, and at the end of the run. The design of this study was similar to Derrick et al. (4). Collecting data in separated trials or during the exhaustive run may lead to different results.

Besides the design of data collection, the intensity of the exhaustive run may play an important role on the inconsistent results. For example, one may argue that, compared to a marathon-run protocol, our protocol was relatively short and may not induce sufficient fatigue to cause neuromuscular compensations, which were inferred with kinematics and acceleratory changes. It should be noticed that, however, the intensity of our protocol was higher than that of a marathon run. As neuromuscular fatigue response depends on different tasks (19) we may not find the same changes observed by others. In the current case, it is possible that the high stress placed on the cardiovascular system forced our subjects to terminate the exhaustive run before the fatigue accumulated in the neuromuscular system reached the threshold. In addition, the treadmill pace was set at the beginning of the trial when the heart rate reached the ventilatory threshold level. With continued running at the fixed pace, it was likely that the heart rate kept increasing to well above the ventalitory threshold level. Although the heart rate, unfortunately, was not monitored throughout the run, such increased heart rate could further stress a the cardiovascular system. However, the different results between this study and Derrick et al. (4) may not be completely explained by intensity. Instead of directly setting a speed, we used the heart rate at ventilatory threshold as the target for the exhaustive run. Although it is plausible to consider such physiologically determined intensity places greater cardiovascular stress, the average speed when our subjects reached their ventilatory threshold (3.3 ± 0.4 $m\cdot s^{-1}$) was coincidentally comparable to the speed that Derrick et al. (4) used (3.4 ± 0.4 $m\cdot s^{-1}$). As a result, our exhaustive run terminated at 17.8 ± 5.7 minutes, also similar to 15.7 ± 1.7 minutes in Derrick et al. (4). Our acceleration data were in the ranges more comparable to Derrick et al. (4) than to Mercer et al. (14), even though no significant pre and postfatigue difference was detected.

It is, therefore, also possible that if the subjects could have run for a longer period, significant changes in kinematics and accelerations would be observable. That is, physiological characteristics of subjects may also contribute to our results. Mizrahi et al. (16) used a protocol similar to ours by setting the treadmill at 5% higher than the speed at which their subjects hit the ventilatory threshold. The speed was 3.5 ± 0.2 $m\cdot s^{-1}$, at which their subjects, surprisingly, were able to finish a 30-minute run. Mizrahi et al. (16) found altered kinematics at the beginning and the end of the run, but the kinematic variables they calculated were different to ours. Derrick et al. (4) and Mizrahi et al. (16) also found significantly increased shank acceleration. However, with much longer exercise time at a comparable intensity, the observed shank acceleration increased from 6.9 ± 2.9 to 11.1 ± 4.2*G*. Although the prefatigue value was similar to ours and Derrick et al.'s, the postfatigue value was much higher. However, no obvious subject differences were identified in the available demographic, training experience, or physiological profiles in these studies.

Several limitations were identified and may also have contributed to the results. Fatigue has been reported to decrease muscle–joint–complex stiffness of the lower extremity (5). Decreasing the stiffness of the lower extremity and the energy absorbing capacity results in kinematic, kinetic, and muscle activation compensations to maintain appropriate muscle–joint–complex stiffness and performance (5). The onset of fatigue may also result in altered knee and ankle kinematics because the body is unable to eccentrically control knee flexion or ankle pronation during the stance phase of running. Unlike prolonged outdoor running at a submaximal but self-managed pace, the current test initially imparted a fixed pace with a particular gait pattern to maintain the pace of the treadmill. The use of the treadmill for the exhaustive run may have prevented the subjects from modifying their current gait pattern as would be experienced during a typical outdoor tempo run. Although the lack of changes with the kinematic variables in this study could potentially be explained by the compensatory muscle adaptations during running, the consistent shank accelerations, head accelerations, and shock-attenuation data may further suggest that running on the treadmill induced a consistent gait pattern despite the onset of fatigue. However, a treadmill was also used in Derrick et al. (4) and Mizrahi et al. (16). Second, muscle activities were not measured in this study. The lack of changes within any of the kinematic variables may also have been related to compensatory muscle adaptations within the body. It is possible that greater muscle activation prohibited the changes in joint motion and maintained the same running pattern. Shock attenuation and the accelerations at the shank and head provide an estimate of the relative impact that is associated with each ground contact but does not directly measure how the impact accelerations are dissipated throughout the body. Even though it is theorized that eccentric muscle contraction and increased joint motion are responsible for the dissipation of impact accelerations, it is not possible to determine the exact location or joint sequencing in which shock is attenuated without muscle activation data also acquired. Finally, the associated movement of ankle pronation and the time to maximum ankle pronation occurred at approximately 10° and 85 milliseconds, respectively, after initial contact during the stance phase of running. The movement itself may have been too small to accurately assess within the time frame of which maximum ankle pronation was achieved.

Because the results of this study do not support the original hypotheses, the influence of running fatigue on kinematics and accelerations remains inconclusive. It is likely that the fatigue experienced during this study that caused the subjects to terminate testing was not the same type of fatigue experienced in a prolonged, lower-intensity running. The muscular fatigue pattern the subjects experienced was not necessarily the same as during typical tempo running. Multiple potential factors that may contribute to our current results were delineated in the discussion, suggesting the complex nature of fatigue and its effects, and the difficulty in planning and controlling such factors in research. Future research should examine fatigue-induced changes in running kinematics and accelerations at different intensities and to determine the threshold at which point the changes may occur.

PRACTICAL APPLICATIONS

The current results do not support the hypotheses that a brief but high-intensity run will induce changes in ankle and knee kinematics, head and shank acceleration, and shock attenuation. It is therefore possible that athletes can train at such intensities of shorter durations until fatigued without the risk of overuse injuries, which accompany the hypothesized changes. However, with conflicting results of Derrick et al. (4) and Mizrahi et al. (16), the risk of injury from running at such intensity cannot be ruled out completely. Considering the current conflicting results on high-intensity run and the more consistent results using marathon protocols, overuse injuries and changes in running mechanics may be impacted by volume of training–distance and time–more so than intensity of exercise. Coaches and athletes may design training schedules by manipulating intensity and carefully controlling distance to reduce the risk of injury, although caution should be taken when combining the high-intensity run with longer durations. Implementing interval running will allow for cardiorespiratory adaptations while limiting the effects of neuromuscular fatigue and possibly injury. Future research should focus on identifying potential changes in kinematics and shock attenuation after a brief, high-intensity run and the threshold at which these changes occur. These data will provide the coach and athlete with a greater degree of sensitivity to design a high-intensity training programs of optimal intensity without compromising the neuromuscular system.

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