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ORIGINAL ARTICLE

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Effects of marathon running on running economy and kinematics

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Abstract The present study was designed to investigate interactions between running economy and mechanics before, during, and after an individually run marathon. Seven experienced triathletes performed a 5-min submaximal running test on a treadmill at an individual constant marathon speed. Heart rate was monitored and the expired respiratory gas was analyzed. Blood samples were drawn to analyze serum creatine kinase activity (S-CK), skeletal troponin I (sTnI), and blood lactate (B-La). A video analysis was performed (200 frames · s^{-1}) to investigate running mechanics. A kinematic arm was used to determine the external work of each subject. The results of the present study demonstrate that after the marathon, a standardized 5-min submaximal running test resulted in an increase in oxygen consumption, ventilation, and heart rate ($P < 0.05$), with a simultaneous decrease in the oxygen difference (%) between inspired and expired air, and respiratory exchange ratio ($P < 0.05$). B-La did not change during the marathon, while sTnI and S-CK values increased ($P < 0.05$), peaking 2 h and 2 days after the marathon, respectively. With regard to the running kinematics, a minor increase in stride frequency and a similar decrease in stride length were observed ($P < 0.01$). These results demonstrate clearly that weakened running economy cannot be explained by changes in running mechanics. Therefore, it is suggested that the increased physiological loading is due to several mechanisms: increased utilization of fat as an energy substrate, increased demands of body temperature regulation, and possible muscle damage.

Key words Fatigue · Energy expenditure · Muscle damage · Catecholamines · Troponin

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Introduction

Since the pioneering work of Fenn (1923), many attempts have been made to characterize the economy and efficiency of animal and human locomotion. Margaria et al. (1963a) measured the oxygen consumption in successive knee flexion exercises with a variable interval time between flexion and extension of the lower limbs. The efficiency was greater when the shortening of the muscle immediately followed the stretching. Taylor et al. (1982) have shown that the big red kangaroo becomes more economical as the speed of hopping increases. Therefore, it is possible that implanted mechanical energy may be temporarily stored in the series of elastic components of active muscle, for utilization in a subsequent muscle action (Asmussen and Bonde-Petersen 1974).

Numerous factors influence the economy and efficiency of running, such as age (e.g., Daniels et al. 1978), gender (e.g., Bransford and Howley 1977), air resistance (e.g., Costill and Fox 1969), body temperature (e.g., Rowell et al. 1969), body mass (e.g., Cureton et al. 1978; Bergh et al. 1991), maximal aerobic power (e.g. Mayhew 1977), and muscle fiber distribution (e.g. Bosco et al. 1987). In addition, values of mechanical efficiency are affected by the methods used to measure and calculate the mechanical work and energy expenditure (e.g., Cavagna et al. 1965; Margaria et al. 1963b; Margaria 1968; Asmussen and Bonde-Petersen 1974; Cavagna and Kaneko 1977; Kaneko et al. 1981; Ito et al. 1983).

Most of these studies have been performed under non-fatiguing conditions, and with the primary interest being the physiological aspects of running economy or efficiency. One might, however, expect that changes in

running mechanics have substantial influence on metabolic energy cost, and vice versa. However, only a few attempts have been made to characterize the interaction between running economy and running mechanics in fatiguing conditions (Williams et al. 1987). In our laboratory, Nicol et al. (1991a) observed that marathon running reduces the maximal sprint performance by 16%, the maximal knee extension torque by 22%, and the maximal drop jump performance by 16%. These impairments in maximal performance were associated with a reduction in the neural input to the muscle and a deterioration in the efficiency of the contractile function (Nicol et al. 1991b). Furthermore, during submaximal constant-speed running, the electromyographic (EMG) activity of the leg extensor muscles is increased due to marathon-induced fatigue (Komi et al. 1986).

In natural human locomotion, the neuromuscular system is acting simultaneously with many other physiological functions. Therefore, the present study was designed to examine how changes in running economy can be characterized by combining biomechanical and physiological factors both during and in recovery from a marathon run.

Methods

Subjects

Seven experienced triathletes (one woman and six men) volunteered to run a marathon. Their mean (SD) age was 29 (5) years, their body mass ranged from 82.0 (11.2) kg to 79.3 (10.6) kg (before and after the marathon, respectively), and their height was 1.82 (0.07) m. Their training included running an average 160 (21) km · month^{-1}, and their personal records for running a marathon race varied from 2.45 to 3.20 h. The subjects were fully informed about the procedures and of all possible risks involved in this study. The study was approved by the University Ethical Committee.

Procedure

The experimental design included different repeated tests before, during, and after the marathon, which was run individually because of the complexity of the measurements. A marathon speed was chosen for each runner on the basis of her or his actual training state, and a taper was individually introduced before the run. For preventing the influences of warm-up on the measurements, the subjects performed an individually standardized warm-up before each test. One week before the marathon the subjects performed a 5-min submaximal running test on a treadmill at their individual constant speed [3.82 (0.33) m · s^{-1}]. This speed was utilized in all testing conditions: at the beginning, after 13 km, after 26 km running, at the end of the marathon, 2 h after, 2 days after, 4 days after and 6 days after the marathon. A cyclist continuously paced the marathon run at the preselected running speed. The marathon was run along a circular route with a relatively constant gradient, and environmental conditions were fairly stable (temperature +10 °C, and relative humidity 70%) for all subjects. During the marathon, the runners were allowed to drink and eat according to their own experience. They drank 2.2 (0.9) l of sport drinks and they ate small amounts of carbohydrates during the run.

The maximal oxygen uptake of the subjects [5.26 (0.93) l · min^{-1}; 65.0 (7.6) ml · kg^{-1} · min^{-1}] was tested on the treadmill several weeks after the marathon run. These tests were performed to investigate the relative load of the marathon run. In the maximal test, the treadmill speed (1° inclination) was increased from 9 km · h^{-1} by steps of 1 km · h^{-1} every 3 min until 15 km · h^{-1} was reached. After that the inclination was increased by 1° every 3 min without increasing the speed. Respiratory variables were analyzed continuously (Sensor Medics, Vmax 229, Yorba Linda, Calif., USA), and after every 3 min, blood samples were taken from a fingertip for analyzing blood lactate (B-La). The mean maximal lactate concentration at the end of the test was 6.25 (1.48) mmol · l^{-1}.

Metabolic measurements

Heart rate was monitored (Polar Sport Tester, Kempele, Finland) throughout the experiment, while the expired respiratory gases were analyzed (Sensor Medics) only during the treadmill tests. To calculate the energy expenditure, an energy equivalent of 20202 J · l^{-1} oxygen was applied when respiratory exchange ratio (R) was 0.82. Changes of ±0.01 in R caused ±50 J changes in energy expenditure (McArdle et al. 1996, p 147). Before and after the marathon, as well as during the recovery period, blood samples were drawn from the ulnar vein for analyzing serum creatine kinase (S-CK), plasma skeletal troponin I (sTnI), blood hemoglobin and hematocrit as well as plasma catecholamines: epinephrine (E) and norepinephrine (NE). For determination of B-La, blood samples were taken from the fingertip before and after the marathon.

Kinematic measurements

While the subjects were on the treadmill, their running was recorded by a video camera (NAC, HSV-200, Japan), which was located 10 m away to the right side of the midpoint of the running lane. The camera was set at a height of 1.2 m above the ground. The operating rate was 200 frames · s^{-1}, and the shutter speed was set to 1/1000 s to ensure sharp images of running. The camera view, which was calibrated using a 3.0 × 2.0 m calibration frame, was set to cover 3.0 m of running space. The frame was parallel with the performance lane and at the midway of the optical axis of the camera.

The speed of the treadmill was measured by means of an optical encoder. The external work of the subjects was determined by a kinematic arm, which is a device used for the three-dimensional recording of human movement (Belli et al. 1992). This device consists of four rigid bars that are linked together by three joints equipped with optical transducers. One end of the kinematic arm was connected to a fixed reference point, while the other end, which was fixed to the back of the subject and near the center of the gravity of the whole body, could move freely in the three spatial directions. For more details of this method see Belli et al. (1992). The kinematic measurements were recorded at two intervals of 20 s during the submaximal test.

Blood analysis

In addition to conventional analysis of blood hemoglobin and hematocrit, the percentage changes in the volumes of blood, plasma, and red cells were calculated according to the method of Dill and Costill (1974). B-La was analyzed using an enzymatic method (Biochemica Boehringer, Mannheim, Germany), and S-CK was analyzed using a commercial test kit (Biochemica Boehringer). sTnI is the inhibitory protein of the protein-tropomyosin complex, which regulates the interaction of actin and myosin in striated muscles. Two optimal pairs of high-affinity monoclonal antibodies were selected to determine the concentrations of both sTnI and the cardiac isoform of troponin I (cTnI) by using two independent immunoenzymetric assays. One assay detects all TnI isoforms, while the other assay is specific only for cTnI. A more detailed description of this method has been published elsewhere (Larue et al. 1993; Rama et al. 1996; Sorichter et al. 1997).

The blood samples required for the determination of plasma catecholamine concentration were centrifuged at 2100 *g* and 3 °C with an anti-oxidant solution containing ethyleneglycoltetraacetic acid and reduced glutathione. The plasma was removed and frozen at −80 °C. Catecholamines from 500 µl plasma were extracted into 30 mg Al_2O_3 in 3 ml of tris-HCL buffer (pH 8.65) in 5-ml conical test tubes. 3,4–Dihydroxybenzylamide hydrobromide (Sigma, St. Louis, Mo., USA) was used as an internal standard to correct for absolute recovery variations in catecholamines. After washing four times with 2 ml H_2O, the catecholamines were eluted into 100 µl of 0.2 M $HCLO_4$ solution. Catecholamines in the eluates (50 µl) were measured by high-pressure liquid chromatography with an electrochemical detector (Esa Coulochem Multi-Electrode, model 5100 A). An ESA Catecholamine HR-80 column (C-18 reversed phase column, 80 × 4.0 mm, 3 µm) was used for the analysis of NE and E, and methanol-phosphate buffer, pH 2.2 (ESA Cat-A-Phase reagent) was used as the mobile phase. The flow rate was 0.6 ml · min^{-1}.

For calibration purposes, known catecholamine standards were treated in the same way as samples, and the peak height ratios (relative to the peak height of the internal standard) of unknown catecholamines were compared to those of synthetic standards (D-2500 Chromato-Integrator, Merck Hitachi, Japan). The detection limit of catecholamine standards in the described method was about 0.2 nmol · l^{-1}, and the inter-assay coefficient of variation was 4–6%.

Delayed-onset muscle soreness of the leg muscles was evaluated by a questionnaire. All of the subjects completed the questionnaire daily, with the perceived leg soreness rated on a five-point scale ranging from 0 (no pain) to 5 (very painful).

Statistics

A multivariate analysis of variance for repeated measurements was utilized to test the main effects of experimental conditions on selected variables. When significant *F*-values were found, a post hoc test was applied to determine the specific condition means that differ from one another. Mean and standard deviation (SD) were calculated by conditions. Finally, Pearson's correlation analysis was utilized to study relationships between the changes in different variables.

Results

The results of the treadmill tests (see Fig. 1) demonstrate that oxygen consumption [43.3 (4.1) ml · kg^{-1} · min^{-1} vs 50.1 (5.9) ml · kg^{-1} · min^{-1}], energy expenditure [876 (84) J · kg^{-1} · min^{-1} vs 996 (122) J · kg^{-1} · min^{-1}], ventilation [83.4 (12.8) l · min^{-1} vs 103.4 (13.0) l · min^{-1}], and heart rate [145 (8) beats · min^{-1} vs 166 (9) beats · min^{-1}] increased ($P < 0.05$) during the marathon run. Simultaneously, *R* [0.82 (0.03) vs 0.75 (0.03), $P < 0.05$] and the amount of oxygen extracted for any volume of air exhaled [true O_2%; 5.21 (0.48) vs 4.68 (0.35), $P < 0.05$] decreased. The recovery follow-up revealed that the true O_2% had already returned to baseline levels 2 h after the marathon, while it took 4–6 days for the other above-mentioned variables to recover to the premarathon level.

Plasma volume and B-La [2.2 (0.3) mmol · l^{-1} vs 2.7 (0.6) mmol · l^{-1}] did not change statistically significantly [6.3 (7.5)%] during the marathon. In the follow-up tests, the plasma volume increased [7.3 (7.7)%, $P < 0.05$] until 2 days after the marathon, while B-La was fairly constant, varying from 1.6 to 2.0 mmol · l^{-1}. Figure 2 demonstrates that S-CK and sTnI values increased, peaking 2 days [1147 (520) U · l^{-1}] and 2 h [164.3 (74.8) ng · ml^{-1}] after the marathon, respectively. S-CK-activity recovered within 6 days, while sTnI concentrations had recovered almost fully by 2 days post-marathon. The perceived muscle soreness developed during the marathon [4 (1)] and remained elevated until the 5th day.

Both NE and E peaked after the marathon, being 71.1% and 72.3%, respectively, higher as compared to

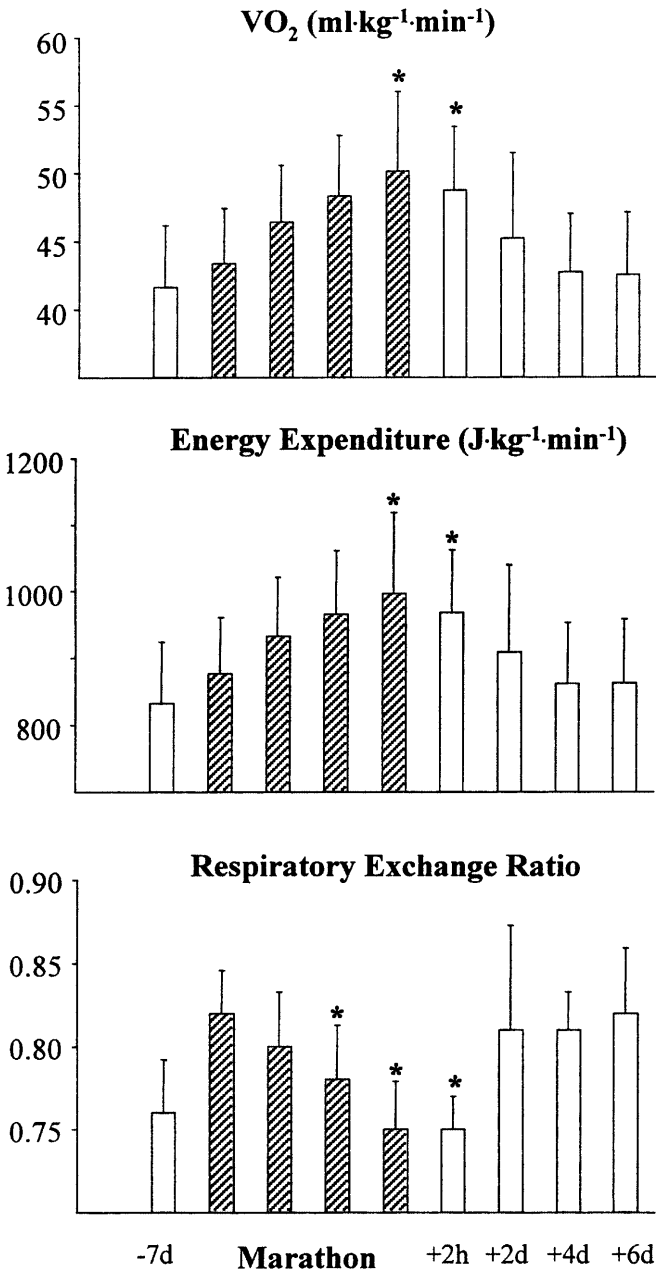

Fig. 1 Mean (±SD) oxygen uptake ($\dot{V}O_2$), energy expenditure and respiratory exchange ratio during different experimental periods: 7 days before the marathon (*−7d*), during the marathon (*Marathon*, *hatched bars*), 2 h after the marathon (*+2h*), and 2, 4 and 6 days after the marathon (*+2d*, *+4d* and *+6d*, respectively). *$P < 0.05$

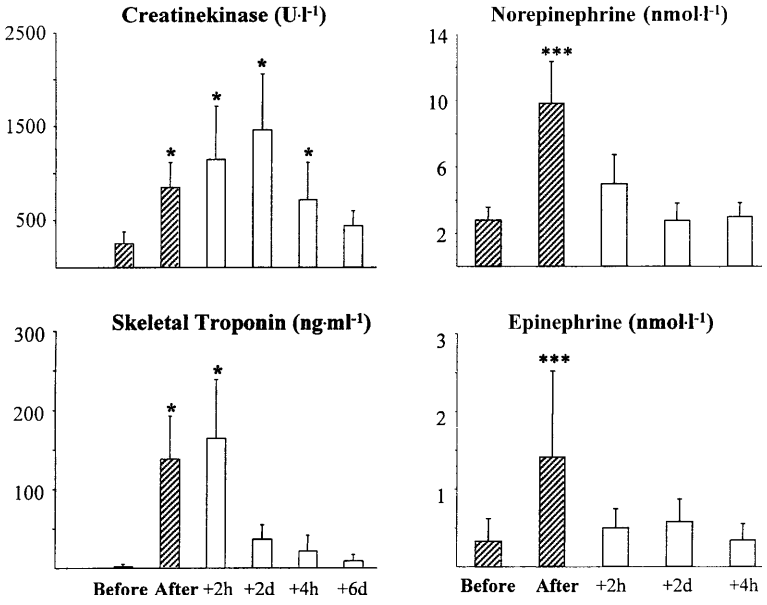

Fig. 2 Mean (±SD) creatine kinase activity, skeletal troponin, norepinephrine and epinephrine values before, immediately after, and at various other time intervals after the marathon run. *$P < 0.05$

the values measured before the marathon. Their follow-up values recovered to close to the premarathon values within 2 h (Fig. 2).

In submaximal running kinematics, only minor changes were observed during the experimental period. Mean stride frequency increased from 2.85 (0.15) Hz to 2.97 (0.14) Hz ($P < 0.01$), while stride length shortened ($P < 0.01$). The other kinematic parameters were fairly constant throughout the conditions. Mean contact time (246–264 ms), external mechanical work, and power were maintained at a quite constant level in every test situation. Angular displacements (Fig. 3) and velocities were also quite stable in every condition.

Discussion

The main findings of the present study demonstrate that oxygen consumption, ventilation, and heart rate increased during the marathon, while true O_2% and R decreased simultaneously. Plasma volume and B-La did not change during the marathon while S-CK and sTnI values increased, peaking 2 days and 2 h after the marathon, respectively. Plasma E and NE concentrations were significantly increased immediately after the marathon. In running kinematics, only minor changes were observed in the tests that were submaximal and close to the marathon running speed.

Muscle damage

During marathon running the muscles are under substantial strain that may lead to muscle damage (Hikida et al. 1983; Warhol et al. 1985). This may be due to two different mechanisms (Ebbeling and Clarckson 1989). The first is based on increased levels of metabolites in muscles (Newham et al. 1983). In the present study, the B-La level was, however, quite constant. The other mechanism of muscle damage addresses the mechanical disruption of the muscle cell (Armstrong et al. 1983). Thus in the present study, the increased levels of the indirect markers of muscle damage, S-CK and sTnI, immediately after the marathon, were probably caused by disruption of the muscle cell membrane.

CK activity is the most frequently used marker of muscle damage. Twenty-four hours after the end of the marathon, the total CK value has been demonstrated in other studies to increase to 414% (Nuviala et al. 1992). This value is similar to that obtained in the present study (Fig. 2). However, CK does not enter the circulation directly, but passes to the lymph system via the interstitial fluid (Lindena et al. 1979). The peak value of CK activity is, therefore, usually seen 2–4 days after the strenuous exercise. In the present study, the peak value of S-CK was reached in 2 days, and it was maintained at an elevated level for up to 4 days after the marathon run. S-CK activity returned to initial levels 6 days after the marathon, by which time the symptoms of muscle soreness had disappeared. The rate of influx and the clearance rate of the enzyme by the reticulo-endothelial system determines the speed at which the CK value in the blood returns to its baseline level (Komulainen 1994).

In evaluating the quantity of muscle damage and its timing, more interest has been focused on the contractile proteins, such as myosin heavy chain (MHC) fragment concentrations (Mair et al. 1992). MHC also shows a delayed increase after exercise-induced muscle damage (Mair et al. 1995), which causes difficulties in formulating an early diagnosis. Therefore, sTnI has been used as an early and skeletal-muscle-specific marker for exercise-induced muscle damage (Sorichter et al. 1997). In the present study, sTnI concentration was increased

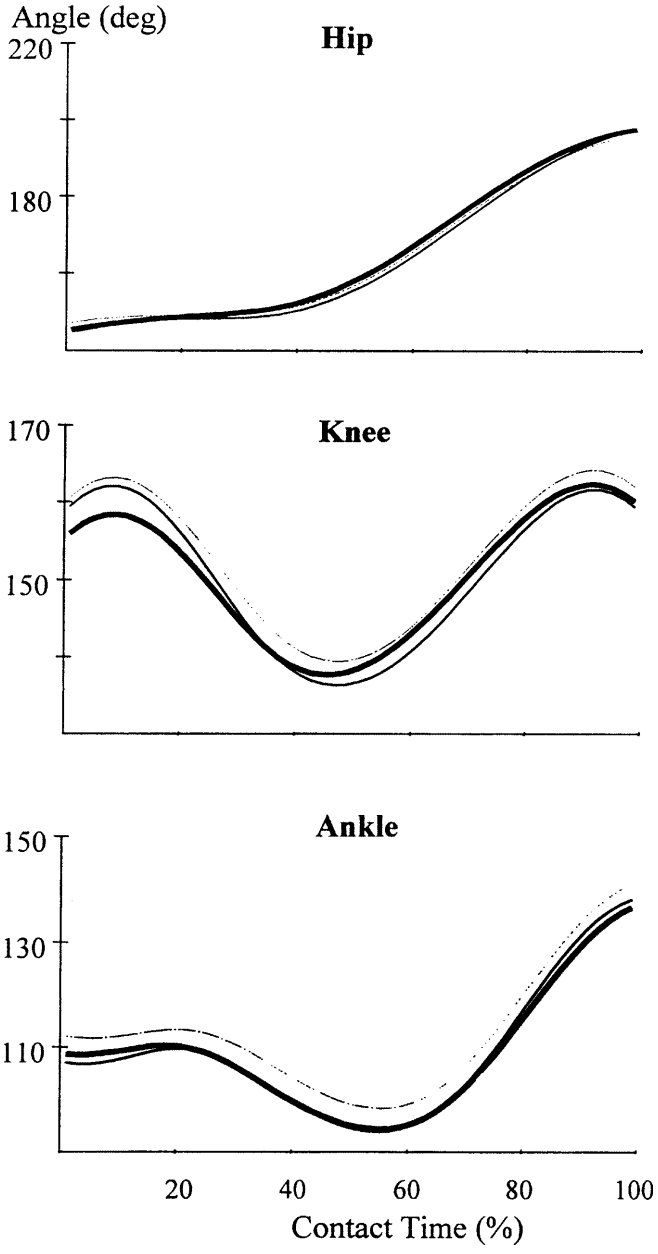

Fig. 3 Angular displacements of the hip, knee and ankle joints during the contact phase, at the beginning of the marathon (*thick black line*), at the end of the marathon (*dashed black line*) and after the 7-day recovery period (*thin black line*)

immediately after the marathon, suggesting acute disruption in the cytoskeletal structure and contractile apparatus (Sorichter et al. 1997).

In the case of muscle damage, biochemical substances, such as bradykinins and prostaglandins are known to be released, and these can stimulate the spontaneous discharge of the group III and IV mechanoreceptors and nociceptors (Kniffki et al. 1978; Rotto and Kaufman 1988). It is known that these muscle afferents have a powerful input to inhibitory interneurons (Cleland et al. 1982), the stimulation of which could lead to presynaptic inhibition of the Ia terminals or inhibition of intereneurons in the oligosynaptic pathways (Duchateau and Hainaut 1993). In both cases, this could result in a reduced neural drive to the muscle.

Further support for the marathon-run-induced impairment in the contractile properties of the skeletal muscle was found by studying changes in the same subjects' maximal performance, as reported previously (Pullinen et al. 1997; Avela et al. 1999). Maximal isometric force and the maximal rate of force production of the plantar flexor and knee extensor muscles decreased by 30% during the marathon, in both voluntary and electrically stimulated (low-frequency fatigue) conditions. These changes are not only due to the metabolic cost of the exercise, but also to exercise-induced muscle damage. In addition, it seems that the increased muscle damage has a positive correlation with the increased oxygen uptake (Kyröläinen et al. 1998). In other words, the muscle damage may partly explain the weakened running economy among the subjects in the present study.

Role of the autonomic nervous system

The autonomic nervous system regulates body temperature and controls the functions of the cardiovascular and endocrine systems. The autonomic impulses are transmitted to the body through two major subdivisions called the sympathetic and parasympathetic systems, of which nerve endings secrete the synaptic transmitter substances, acetylcholine or NE. Another part of the sympathetic nervous system is the adrenal medulla, which secretes the catecholamine hormones, E and NE. In the present study, the concentrations of these catecholamines in the plasma increased drastically during the marathon run.

Figure 4 demonstrates that the higher the increase in NE, the lower the change in the true O_2% induced by

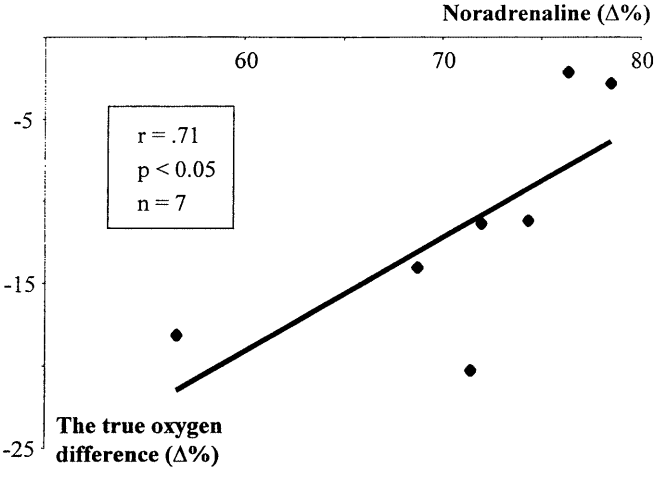

Fig. 4 The relationship between the changes in norepinephrine and the changes in the true oxygen difference (the amount of oxygen extracted for any volume of air exhaled)

the marathon run. This suggests that NE, as a powerful stimulator of lipolysis in adipose tissue, has a role in the utilization of fat as an energy substrate. Therefore, free fatty acids and glycerol values have been demonstrated to increase continuously during marathon running. On the other hand, blood glucose concentration has been shown to increase and to peak at approximately 1 h, but then to decrease over time, remaining at or above resting levels (O'Brien et al. 1993).

In the present study, the increased utilization of fat toward the end of the marathon could be assessed by looking at the values of *R*. Thus, it is possible that the change in energy substrates from glycogen to fat could also explain the changes in NE concentration. The increased concentration of E due to the marathon running may be interpreted to emphasize its importance as a primary stimulator of glycogenolysis and lipolysis. The role of E might be more important at the beginning of the marathon, while NE may play a major role at the end of it. Thus, adaptations of fuel homeostasis may contribute to the maintenance of physical performance after prolonged exercise (Tuominen et al. 1996).

In addition to the changes in fuel utilization, an increase in the internal temperature regulation might also affect the running economy during a marathon run. After the marathon run, the mean rectal temperature of 30 recreational runners was shown to reach 38.9 °C (Noakes et al. 1991). Increased internal body temperature has been demonstrated to increase oxygen uptake (Saltin and Hermansen 1966) and, therefore, to weaken running economy. This might also be the case in the present study, although the body temperature was not measured.

Thermal stress could, together with elevated levels of circulating catecholamines, cause increased action of the respiratory muscles and hyperventilation and, therefore, further demands for oxygen uptake. In the present study this phenomenon can be seen in the form of increased ventilation (19%), which was greater in magnitude than the respective increase in oxygen uptake (7%). These findings are in agreement with the study of Guezennec et al. (1996).

Neuromuscular failure

Much to our surprise, running kinematics changed only slightly during the marathon run, as well as during the recovery period. Contact times, angular displacements and velocities, vertical displacements of the center of gravity of the whole body, mechanical cost and external mechanical energy (potential and kinetic) did not differ between the tests. Despite rather constant mean values, the interindividual variability was quite large. This is in agreement with earlier studies (e.g., Buckalew et al. 1985; Williams et al. 1987). On the other hand, Nicol et al. (1991c) observed some significant biomechanical changes induced by a marathon run: the knee joint was more flexed at heel strike, and the hip extension range and the hip extension velocity were increased. In the present study, the only significant changes in the running kinematics were an increased stride frequency and shortened stride length. It is possible that the increased stride frequency may have increased the amount of muscle activity by shortening the duration of relaxation. Thus, increased muscle activity could weaken running economy. However, due to the weakened neuromuscular performance it is not economical to maintain initial stride frequency and length. Therefore, the observed changes are probably an attempt to compensate for the impaired neuromuscular function.

The relationship between running kinematics and running economy seems to be controversial. According to Williams and Cavanagh (1987), running economy is the sum of the influence of many variables. However, it appears that no single kinematic variable can fully explain the decrease in running efficiency (Hausswirth et al. 1997) or in running economy (Williams et al. 1987; Nicol et al. 1991c). Thus, one could conclude that individual changes in running kinematics, as measured at marathon-running speed, could only partially explain the drastically weakened running economy.

It should be obvious, however, that during long-lasting submaximal running, decreased neuromuscular performance could be compensated for. Therefore, changes in the running mechanics during fatiguing submaximal conditions should be different to those measured in maximal effort. In an earlier study, Nicol et al. (1991c) found a clear reduction in maximal sprinting speed as well as in tolerance to high stretch load (drop jump test). In the present study, the maximal isometric force and its rate of production was clearly decreased. Figure 5 is an example from this same project, reported previously by Avela et al. (1999), demonstrating clear changes in ground reaction forces and EMGs of the leg extensor muscles during maximal stretch-shortening cycle (SSC) exercises before and after a marathon run. These maximal SSC exercises demonstrated an increased peak force reduction after an initial impact peak due to marathon running. This took place in parallel with a decline in the EMG activity of the measured muscles. Interestingly, the short-latency reflex component was significantly reduced, as also shown in Fig. 5. This reduction might be due to muscle damage, as discussed earlier. These results suggest further that in maximal SSC conditions, the tolerance of the leg extensor muscles to high-impact loads and, therefore, the ability to perform powerful muscle actions, were impaired. These findings emphasize strongly that high loading/velocity conditions should be utilized to examine the biomechanical/physiological responses of the marathon run.

The weakened function of the neuromuscular system seems to have influence on the running economy. Unfortunately, in the present study the EMGs were not measured during the marathon. However, it has been demonstrated that the EMG patterns change considerably in association with relatively small changes in the

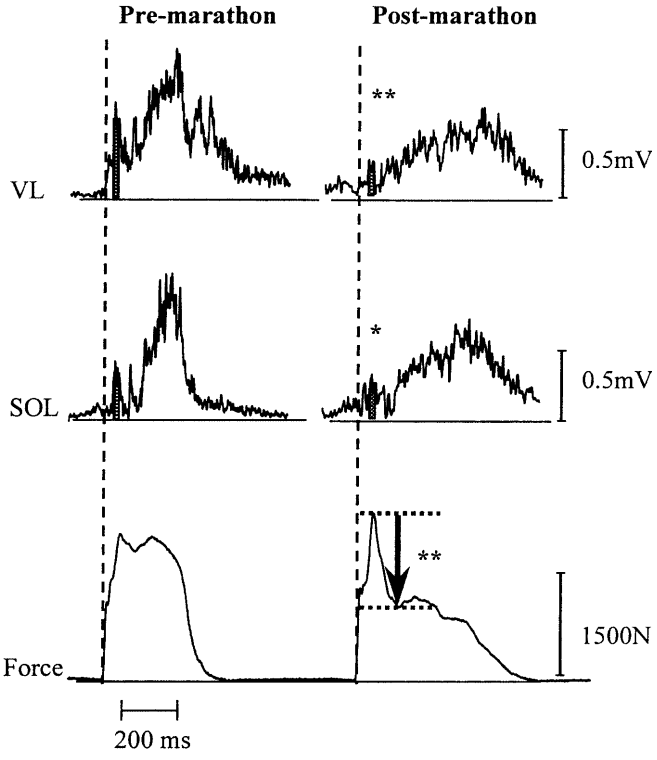

Fig. 5 The average electromyogram patterns of the vastus lateralis (VL) and soleus (SOL) muscles, and the vertical ground reaction force recorded before (*Pre-marathon*) and after (*Post-marathon*) a marathon run (taken from Avela et al. 1999). $*P < 0.05$ and $**P < 0.01$

ground reaction forces measured at marathon speed (Komi et al. 1986). Thus, at the end of the marathon, greater neural input to the muscle was required to produce the same resultant force in the push-off phase of the ground contact. Due to the linear relationship between EMG levels and oxygen uptake (Bigland-Ritchie and Woods 1974), it can be concluded that increased neural input to the muscle during the marathon run causes higher demands of oxygen uptake and, therefore, weakened running economy.

Other factors

Many hematological changes have been observed in subjects during marathon races. Hemolysis in endurance exercise may be related to structural changes in red blood cell membrane skeletal proteins (Jordan et al. 1998). Changes in plasma volume, plasma protein, osmolality, and Na^+ electrolyte concentration may be non-significant, while K^+, and plasma concentrations of renin and aldosterone increase (Pastene et al. 1996). In the present study, plasma volume decreased individually during the marathon run, while the changes in mean values were unaltered [−6.2 (7.5)%]. Pastene et al. (1996) have interpreted this finding to indicate that water production and decomplexing of water linked to muscle and liver glycogen could have compensated for a great part of the water loss. The significant increase in plasma volume, which was observed in the 2 days following the marathon run, may be attributable to both a protein shift toward the intravascular space and to renal retention of Na^+ (Pastene et al. 1996).

In conclusion, the results of this study demonstrate clearly that weakened running economy cannot be explained by minor changes in submaximal running mechanics. Therefore, the increased physiological loading that occurs during the marathon run may be due to several mechanisms: increased utilization of fat as an energy substrate, increased demands of body temperature regulation, increased neural input to the muscle, and the acute effects of muscle damage. The results further emphasize that instead of using conventional submaximal running tests, the high-intensity loading/velocity test should be utilized to observe the true weakening of the neuromuscular function that results from performing a marathon run.

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