Effects of marathon running on running economy and kinematics

Accepted: 20 March 2000

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

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 2020 J · m⁻¹ · s⁻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), skeletal muscle troponin I (sTnI), blood hemoglobin and hemocrit 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 x 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 isofrom of troponin I (cTnI) by using two independent immunoenzymatic assays. One assay detects all TnI isofoms, 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).