THE YALE JOURNAL OF BIOLOGY AND MEDICINE 54 (1981), 243-248

Pulmonary Dysfunction in Ultramarathon Runners*

DONALD A. MAHLER, M.D.[a] AND JACOB LOKE, M.D.[b]

[a]*Assistant Professor of Medicine, Pulmonary Section, Yale University School of Medicine;* [b]*Associate Professor of Medicine, Pulmonary Section, Yale University School of Medicine, New Haven, Connecticut*

Received June 11, 1981

To assess the effects of *extreme* exercise on lung function we measured maximal expiratory flow volume (MEFV) curves in fifteen runners (mean age, 35.3 years; range, 26–46) before and after an 80.6 to 100 km (50–62.2 mile) road race. Mean running time for 80.6 km was 7 hr, 42 min. Post-race testing showed significant decreases of 12.4 percent in forced vital capacity (FVC), 9.5 percent in forced expiratory volume in one second (FEV_1), 13.7 percent in peak expiratory flow (PF), and 28.4 percent in flow at 50 percent of FVC (MEF50). By 2.5 hours after the race lung function had improved. The reduction in flow rates after ultramarathon running may be due to airway obstruction. In contrast, the decrease in FVC with gradual recovery of lung function after rest and nourishment suggests the development of respiratory muscle fatigue.

INTRODUCTION

Although long distance running involves severe metabolic demands [1,2] and provokes strenuous cardiorespiratory responses [3], the effects of prolonged exercise on lung function are not well defined. Over fifty years ago Gordon et al. [4] reported a decrease in the forced vital capacity (FVC) of the lungs in runners completing the 1923 Boston Marathon. Using maximal expiratory flow-volume (MEFV) curves we recently confirmed this finding and showed in addition that airway obstruction did not occur in marathon (42.2 km) runners competing at above freezing ambient temperatures [5].

However, the exact mechanism for the change in post-race FVC has not been determined, and whether *extreme* endurance exercise results in further alterations in pulmonary function is unknown. To address these questions, we measured MEFV curves in runners before and after an 80.6 to 100 km ultramarathon race (50 to 62.2 miles). To evaluate for recovery of respiratory dysfunction, testing was repeated at 2.5 and 24 hours.

METHODS

Pre- and post-race lung function was measured in fifteen runners at the Lake Waramaug Ultramarathon (LWU) which was held on May 6, 1979, in Preston, Connecticut. The course was a 7.66-mile loop, and the distance of the race was 80.6 or 100 km (50 or 62 miles). Humidity of the ambient air was 54 percent, and the temperature ranged from 10.0°C to 18.3°C. Testing was performed in a building located within 0.05 km from the start and finish of the race. On the morning of race

*Presented in part at the Annual Meeting of the American College of Sports Medicine, Las Vegas, Nevada, May 1980 (Med Sci Sports Exer 12:105, 1980).

day each runner completed a questionnaire on respiratory symptoms, training, and previous running performances. Informed consent was obtained.

Pulmonary function was measured with a pneumotachygraph integrated flow-volume device connected to a Brush 500 High Performance X-Y recorder (Gould Inc., Cleveland, OH), which displayed a maximum expiratory flow-volume curve [6]. Volume was calibrated with a three-liter syringe and flow with a precision bore flowrator tube (Fischer & Porter, Warminister, PA) having a range of 0–10 liters/sec. Four MEFV curves were measured in each runner in the seated position before and immediately after completion of the marathon. From the MEFV curve, forced expiratory volume in one second (FEV_1), FVC, peak expiratory flow (PF), and flow rate at 50 percent of FVC (MEF50) were obtained. The value of these indices was taken from the MEFV curve yielding the largest sum of FVC and FEV_1 [7]. The post-race MEF50 was measured from total lung capacity at the same volume at which pre-race MEF50 was determined. The results of pre-race lung function was compared to the predicted normal values of Higgins and Keller [8].

Post-race testing was performed 10 to 15 minutes after each runner finished the race, at which time the subject was breathing comfortably. Ten of the runners were available for retesting of lung function 2.5 hours later, while two runners were also tested 24 hours after the race.

The results from pre- and post-race MEFV curves were compared using the paired t-test; $p < 0.05$ was considered significant.

RESULTS

Anthropometric and training characteristics are listed in Table 1. Thirteen males and two females with a mean age of 35.3 years (range, 26–46 yr) participated in the study; the majority were experienced long distance runners, nine of whom had previously completed an ultramarathon race. One runner had a history of episodic wheezing. Twelve subjects completed 80.6 km while three athletes ran 100 km; the mean running time for 80.6 km was 7 hr, 42 min (range, 5 hr, 51 min to 9 hr, 39 min).

All subjects had a normal pre-race FVC. The pre-race FVC was 5.00 ± 0.21 liters (mean ± SEM) and the post-race FVC was 4.38 ± 0.20 liters, a 12.4 percent decrease ($p < 0.001$) (Fig. 1). PF was decreased by 13.7 percent, from 9.12 ± 0.48 1/sec to

TABLE 1
Physical Characteristics and Running Experience in 15 Runners Completing the 1979 Lake Waramaug (Connecticut) Ultramarathon (50 miles and 100 km)

	Mean	Range
Age (yr)	35.3	(26–46)
Height (cm)	177.6	(163–188)
Training distance (km/week)	118	(58–203)
Number of marathons completed	21.2	(1–144)
Best marathon time (hr:min)	3:07	(2:37–3:45)
50-mile ultramarathon time (hr:min)	7:42	(5:51–9:39)

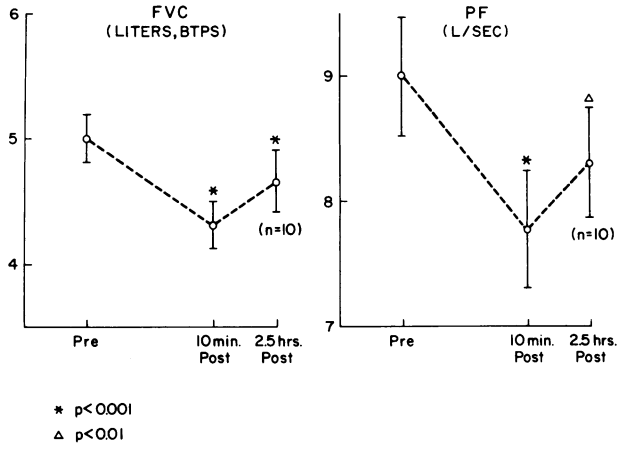

FIG. 1. Pre- and post-race forced vital capacity (FVC) and peak expiratory flow (PF) in 15 ultramarathon runners.

7.77 ± 0.48 l/sec ($p < 0.001$) (Fig. 1). The mean pre-race FEV_1 (3.79 ± 0.21 liters) was also significantly reduced (-9.5 percent) after the race (3.43 ± 0.18 liters) ($p < 0.005$) (Fig. 2). Similarly, MEF50 was decreased significantly (-28.4 percent) from 4.33 ± 0.31 L/sec to 3.10 ± 0.24 L/sec ($p < 0.005$) (Fig. 2). There was no significant difference between the pre-race (77.5 ± 2.2 percent) and post-race (78.3 ± 1.5 percent) FEV_1/FVC ratio.

None of the runners experienced shortness of breath. The runner with a history of wheezing had a 9.3 percent decrease in FVC, but FEV_1 and MEF50 did not change after the ultramarathon.

Repeat testing 2.5 hours later in ten runners showed an increase of 3 percent, 6 percent, and 9 percent from immediate post-race measurements in FEV_1, FVC, and MEF50, respectively. Two subjects were also retested 24 hours after the ultramarathon at which time lung function had returned to pre-race values.

DISCUSSION

The results of this study demonstrate a reduction in pulmonary function after *extreme* endurance exercise. Except for the individual with a history of wheezing, all runners had a decrease in FVC, PF, FEV_1, and MEF50. The percentage decrease in FVC after the ultramarathon (-12.4 percent) was greater than we have observed after a 20 km (-4.2 percent) [9] or 42.2 km (-3.9 percent and -5.9 percent) [5] race,

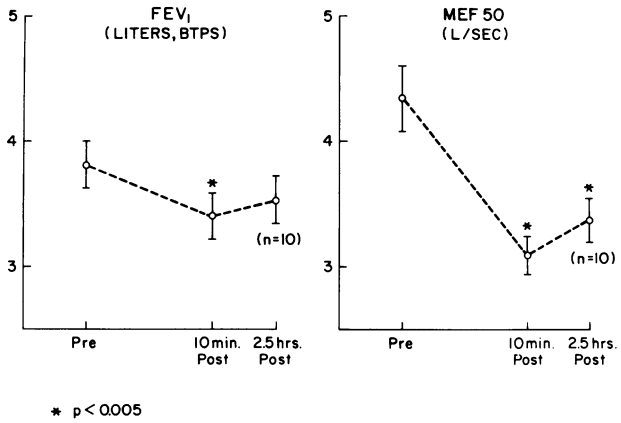

FIG. 2. Pre- and post-race forced expiratory volume in one second (FEV_1) and flow at 50 percent of FVC (MEF50) in 15 ultramarathon runners.

although less than the change noted in runners at the 1923 Boston Marathon (−17 percent) [4]. In previous studies we have shown that flow rates did not change after running 20 km and 42.2 km at above freezing ambient temperatures [5,9]. In contrast, at the LWU there were significant decreases in FEV_1, PF, and MEF50, while the FEV_1/FVC ratio was unchanged.

Based on previous investigations [10,11] we assumed that total lung capacity remained constant after the LWU and measured the post-race MEF50 at 50 percent of the pre-race FVC. Since it is possible that TLC may change after endurance exercise, we also measured the post-race MEF50 at 50 percent of the post-race FVC which should approximate mid-flow rates at the same relative lung volume. Using this method, there was also a significant decrease in the post-race MEF50 (3.60 ± 0.26 L/sec; $p < 0.005$).

It is interesting that the runner with a history of wheezing did not experience a decrement in flow rates after the ultramarathon. Although use of bronchodilator medication could account for the preservation of lung function, the runner denied any drug therapy. Subsequent testing in the Pulmonary Function Laboratory demonstrated the presence of reversible airway obstruction consistent with asthma. One possible explanation for the findings at the LWU is that the release of endogenous catecholamines during exercise might contribute to bronchodilation, or at least prevent bronchoconstriction, in asthmatic individuals [12].

Air flow is dependent on resistance of the airways and the driving pressure of the thorax [13]. Any decrease in flow rates may be due to an alteration in the state of the airways and/or a reduction in the effective driving force. Although the FEV_1/FVC ratio remained constant after the ultramarathon indicating that large airway obstruction did not develop, small airways dysfunction cannot be excluded. Another mechanism for the decrease in flow rates may be related to a change in the elastic properties of the lungs. However, information on compliance of the lungs during exercise is conflicting with decrease [11,14], increase [15], and no change [16,17] being reported.

The decreases in lung function after the LWU, excluding the change in MEF50, may also be due to fatigue of the respiratory muscles. The force generated by the thoracic musculature is one of the major determinants in the performance of the MEFV curve [18], and weakness or fatigue of these muscles can cause a decrease in FVC, FEV_1, and PF [19,20]. Direct measurement of ventilatory muscle function would be necessary in order to document respiratory muscle fatigue in runners.

Muscle function during prolonged exercise is dependent on several factors, including skeletal muscle respiratory enzymes [21], energy sources [22], circulation [23], and the contractile properties of the muscle [24]. Of these, muscle glycogen appears necessary for sustained muscular effort [25]. Previous investigators have shown that physical exhaustion coincided with extremely low muscle glycogen levels despite the availability of circulating free fatty acids [26–28]. Although the accumulation of blood lactate during exercise has been associated with muscular distress and exhaustion [1], long distance running is primarily an aerobic process and only low levels of lactic acid are found in runners at the finish of a marathon race [2]. In fit individuals, anaerobic metabolism with associated lactate production occurs at a relatively high intensity of exercise or maximal oxygen consumption [29]. Accordingly, the ultramarathon runners could achieve high levels of ventilation for prolonged time periods without the development of lactic acidosis, reflecting efficient gas exchange and transport. Thus, it appears that fuel metabolism and efficiency of

respiration are important factors in muscle performance during endurance exercise.

Serial measurements throughout a marathon race have shown that minute ventilation averaged over 80 L/min in two proficient runners [30]. Such increased levels of ventilation during 6 to 10 hours of ultramarathon running might lead to respiratory muscle fatigue which presumably reflects a general muscle phenomenon associated with prolonged exertion. The greater decrease in FVC at the LWU (80.6 km) compared to the marathon distance (42.4 km) [5] as well as the partial recovery of respiratory function after 2.5 hours of rest and nourishment after the ultramarathon support this concept.

ACKNOWLEDGEMENTS

The authors thank Dr. D.P. Schilder for critical review of the manuscript and A. Mahler, L. Pantalena, and J. Virgulto for technical assistance.

REFERENCES

1. Costill DL: Metabolic responses during distance running. J Appl Physiol 28: 251–255, 1970
2. Costill DL, Fox EL: Energetics of marathon running. Med Sci Sports 1:81–87, 1969
3. Fox EL, Costill DL: Estimated cardiorespiratory responses during marathon running. Arch Environ Health 24:316–324, 1972
4. Gordon B, Levine SA, Wilmaers A: Observations on a group of marathon runners with special reference to the circulation. Arch Intern Med 33:425–434, 1924
5. Mahler DA, Loke J: Lung function after marathon running at warm and cold ambient temperatures. Am Rev Respir Dis 124:154–157, 1981
6. Virgulto J, Bouhuys A: Electronic circuits for recording of maximum expiratory flow-volume (MEFV) curves. J Appl Physiol 35:145–147, 1973
7. Gardner RM (chairman): Report of snowbird workshop on standardization of spirometry. ATS News 3:20, 1977
8. Higgins MW, Keller JB: Seven measures of ventilatory lung function. Am Rev Respir Dis 108:258–272, 1973
9. Mahler DA, Snyder P, Loke J: Pulmonary function in runners before and after a 20 kilometer road race. Conn Med 44:549–552, 1980
10. Maron MB, Hamilton LH, Maksud MG: Alterations in pulmonary function consequent to competitive marathon running. Med Sci Sports 11:244–249, 1979
11. Stubbing DG, Rengally LD, Morse JLC, et al: Pulmonary mechanics during exercise in normal males. J Appl Physiol 49:506-510, 1980
12. Jones RS: Assessment of respiratory function in asthmatic children. Brit Med J 2:972–975, 1966
13. Bates DV, Macklem PT, Christie RV: Respiratory function in disease. Philadelphia, WB Saunders Co, 1971, pp 27–40
14. Gilbert R, Auchincloss JH Jr: Mechanics of breathing in normal subjects during brief, severe exercise. J Lab Clin Med 73:439–450, 1969
15. McIlroy MB, Marshall R, Christie RV: The work of breathing in normal subjects. Clin Sci 13:127–236, 1954
16. Chiang ST, Steigbiegel NH, Lyons HA: Pulmonary compliance and non-elastance during treadmill exercise. J Appl Physiol 20:1194–1198, 1965
17. Granath A, Horie E, Linderholm H: Compliance and resistance of the lungs in the sitting and supine positions at rest and during work. J Clin Lab Invest 11:226–234, 1959
18. Leith DE, Mead J: Mechanisms determining residual volume of the lungs in normal subjects. J Appl Physiol 23:221–227, 1967
19. Derenne J-Ph, Macklem PT, Roussos CH: The respiratory muscles: mechanics, control, and pathophysiology (Part III). Am Rev Respir Dis 118:581–601, 1978
20. Gibson GJ, Pride NB, Newsom Davis J, et al: Pulmonary mechanics in patients with respiratory muscle weakness. Am Rev Respir Dis 115:389–395, 1977
21. Hickson RC, Bomze HA, Holloszy JO: Linear increase in aerobic power induced by a strenuous program of endurance exercise. J Appl Physiol 42:372–376, 1977

22. Felig P, Wahren J: Fuel homeostasis in exercise. N Engl J Med 288:774–777, 1973
23. Monod H, Scherrer J: The work capacity of a synergic muscular group. Ergonomics 8:329–337, 1965
24. Faulkner JA, Maxwell LC, Ruff GL, et al: The diaphragm as a muscle: contractile properties. Am Rev Respir Dis 119 (2):89–92, 1979
25. Hermansen L, Haltman E, Saltin B: Muscle glycogen during prolonged severe exercise. Acta Physiol Scand 71:129–130, 1967
26. Hultman E: Studies on muscle metabolism of glycogen and active phosphate in man with special reference to exercise and diet. Scan J Clin Lab 19 (Suppl 94): 1–63, 1967
27. Pernow B, Saltin B: Availability of substrates and capacity for prolonged heavy exercise in man. J Appl Physiol 31:416–422, 1971
28. Saltin B, Karlsson J: Muscle glycogen utilization during work of different intensities. In Muscle Metabolism During Exercise. Edited by B Pernow, B Saltin. New York, Plenum, 1971, pp 289–300
29. Davis JA, Frank MH, Whipp BJ, et al: Anaerobic threshold alterations caused by endurance training in middle-aged men. J Appl Physiol 46:1039–1046, 1979
30. Maron MB, Horvath SM, Wilkerson JE, et al: Oxygen uptake measurements during competitive marathon running. J Appl Physiol 40:836–838, 1976