Health & Fitness Journal of Canada

Volume 4 **June 1, 2011** **Number 3**

COMMENTARY
An Early Study of the Oxygen Cost of Breathing

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Abstract

Dr Roy Shephard's 1966 paper "The oxygen cost of breathing during vigorous exercise" (Shephard, 1966) is revisited. At the time of the study, modern understanding of the mechanical work of breathing had been established, but estimates of the associated oxygen cost were found to be variable. At least in part, the variability could be ascribed to differences in the method adopted to increase breathing (voluntary hyperventilation, CO_2-driven, added instrumental dead space) and a lack of precision in the measurement of small inspired-expired O_2 concentration differences. An experimental design was adopted to minimize the effect of such factors. The results suggested that during heavy exercise, at ventilations less than 90 l/min, the O_2 cost of breathing was less than 2 ml/min/l V_E, but at higher ventilation induced by hyperventilation at breathing frequencies of 50 and 100 breaths/min, the O_2 cost increased to 4 ml/min/l V_E.

The study remains a model of carefully conducted physiology. Even after several decades, controversy remains as to whether the oxygen cost of breathing contributes to exercise limitation in healthy subjects and patients with obstructive lung disorders (COPD).

Health & Fitness Journal of Canada 2011;4(3):13-16

Keywords: respiratory physiology, medicine

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Introduction

Roy Shephard and myself could not be called close contemporaries, since he obtained his medical degree 3 years before me. In those three years, he showed evidence of the prodigious productivity for which he became famous. In 1955 he published the results of his PhD thesis in the Journal of Physiology, and already able to cite six of his own papers. However, we were both lucky enough to cut our scientific teeth in the heyday of British respiratory physiology, when young researchers benefitted greatly from the regular meetings of the Medical Research Society (MRS) and Physiological Society. There was even a Breathing Club which met on Saturday mornings following MRS meetings, faithfully attended by many across the UK. His thesis work on "The carbon dioxide balance-sheets of the body" showed his clear interest in integrative physiology (Shephard, 1955) . I came to the field some ten years later, and it more-or-less dominated my research interests for the rest of my career. By the mid 1960s Roy's chosen path in exercise and fitness was firmly established, and in 1966 he published an important study on the oxygen cost of breathing during vigorous exercise (Shephard, 1966). The paper is well-worth reading to this day, which is what I decided to do, when asked to contribute a short paper on Roy's contributions to pulmonary physiology.

Arthur Otis, with his colleagues Wallace Fenn and Hermann Rahn, had elucidated the mechanical factors contributing to the work of breathing, clearly laid out in his seminal 1954 paper in *Physiological Reviews* (Otis, 1954). At the same time, Richard Riley wrote an incisive editorial (Riley, 1954), in which he argued that the metabolic cost of the increased work of breathing, in terms of O_2 consumption and CO_2 production, might contribute to respiratory failure. In this concept, the work of breathing might increase to an extent that the associated metabolic costs would exceed any increase in pulmonary uptake of O_2 and output of CO_2. Furthermore, there was the possibility that even in healthy individuals, ventilation in heavy exercise might reach a similar "limiting value", and thus provide a pulmonary limitation to maximal exercise.

In the introduction to his paper, Shephard lays out the mathematical background to these concepts and reviews the difficulties inherent in their measurement. Previous estimates of the O_2 cost appeared very variable, due in part to the method of study (whether voluntary hyperventilation, hypercapnia, added dead space, etc), variable pattern of breathing (spontaneous or controlled frequency), and difficulties in measurement (especially of inspired-expired O_2 differences). Shephard argues for the use of exercise, voluntary hyperventilation and hypercapnia and controlled frequency of breathing, and describes an elaborate protocol. Over an 8-week period, ten subjects took part in sixteen runs on a treadmill at 80% of maximal aerobic capacity for 15 min, to provide adequate training and cross-over of the experimental conditions (spontaneous breathing, voluntary hyperventilation and hypercapnia, both at two frequencies of 50 and 100 b/min). Meticulous care was also taken with the analytical techniques, which involved chemical analysis of gases in the Lloyd-Haldane apparatus and van Slyke blood gas analyser, both techniques famously known to "sort the men from the boys". From these studies, he concluded that the O_2 cost of breathing was midway between previous estimates obtained in studies that used voluntary hyperventilation and hypercapnia. In the range of ventilation between 90 and 130 l/min the O_2 cost amounted to around 4 ml per litre ventilation, the "limiting ventilation" was approximately 120 l/min, and the efficiency of the respiratory muscles was in the range of 7-10%. Thus, Shephard concluded that "the margin between actual and useful ventilation [in heavy exercise] is thus quite small even in young healthy subjects". The conclusion was supported by a calculation that the cost of breathing could amount to 10% of the total metabolic cost of near maximal effort. In contrast, the oxygen cost of uncontrolled breathing during exercise at less than 90 l/min was 1-2 ml/min per litre V_E. We should also note that breathing frequencies of 50-100 are seldom encountered in exercise.

Over the many years since the publication of the paper it has been cited frequently in the exercise literature, and also used to support Riley's concept that in emphysema the oxygen cost associated with breathing during exercise might increase to an extent that it might seriously curtail the amount of oxygen available for non-respiratory work. Shephard himself noted that in emphysema it "seems quite likely that the oxygen cost of breathing could become a significant factor limiting physical performance". This seemed plausible, because a similar absolute cost of

breathing of, say, 4-500 ml/min, could constitute 50% or more of the maximal exercise O_2 uptake of a patient with severe COPD. More recently the concept has been expanded to argue that blood flow to respiratory muscles in COPD might increase to an extent that they might "steal" blood flow from exercising muscles, to provide a further limitation. However, to perhaps a surprising extent, the field remains controversial.

This is not the place to review all the factors contributing to the controversial aspects, which would have to cover issues related to experimental design and the integration between all the physiological mechanisms contributing to the function of the respiratory pump (Macklem, 1980). Even after these factors have been resolved there remain at least three points of argument; first, the magnitude of the maximal oxygen consumption by respiratory muscles; second, its contribution to exercise limitation; and third, its contribution to hypercapnia (respiratory failure). Studies of loaded breathing at rest or in exercise, where the breathing pattern is not constrained, have usually found an O_2 cost of less than 200 ml/min, consistent with a total respiratory muscle mass of less than 1 kg. Then, respiratory muscle VO_2, just as with skeletal muscle, is closely related to the mechanical work performed. Thus at a given ventilation, patients with COPD will incur a higher O_2 cost in breathing. However, the absolute increase in O_2 cost appears not to exceed 200 ml/min. For example, in the classic study by Cherniack (Cherniack, 1958) the maximum recorded in 22 COPD patients was 125 ml/min. An increase in respiratory work is appreciated as an increase in the effort expended in breathing, and when dyspnea reaches an intolerable level voluntary exercise is discontinued. Finally, underventilation may be an adaptive strategy in the face of dyspnea. Some normal subjects exhibit hypercapnia during dead-space loading in exercise, with alveolar PCO_2 increasing by as much as 20 mmHg, a value similar to that found in some patients with COPD. In these examples the hypercapnia is not accompanied by increases in CO_2 production. Thus the ventilatory limitation of exercise is less likely to be due to the attainment of a "limiting" metabolic cost of breathing than to the associated perception of respiratory muscle effort which reaches an intolerable severity (Jones and Killian, 1991).

A final word. Nowadays scientific papers tend to avoid providing insights into the author's personality. This was less often the case in the past, when the writing was not as impersonal. In a paper he wrote on his thesis topic of "carbon dioxide balance sheets" Shephard notes "Subject RJS viewed the experiments with personal interest. There was a tendency to analyse symptoms, and during the earlier observations a measure of anxiety lest the technician should fail to carry out the necessary manipulations correctly" (Shephard, 1955). A concern for the quality of experimental design and measurement is obvious in all his work, and remains the reason for his success and reputation as a leader in the field of human physiology.

Qualifications

The author's qualifications are as follows: Dr. Norman L Jones MD, FRCP, FRCP(C)

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