Maximal oxygen uptake “classical” versus “contemporary” viewpoints

ULF BERGH, BJORN EKBLOM, and PER-OLOF ÅSTRAND

Defense Research Establishment, Stockholm, Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, SWEDEN

ABSTRACT

BERGH, U., B. EKBLOM, and P.-O. ÅSTRAND. Maximal oxygen uptake “classical” versus “contemporary” viewpoints. Med. Sci. Sports Exerc., Vol. 32, No. 1, pp. 85–88, 2000. In two articles Timothy Noakes proposes a new physiological model in which skeletal muscle recruitment is regulated by a central “govenor,” specifically to prevent the development of a progressive myocardial ischemia that would precede the development of skeletal muscle anaerobiosis during maximal exercise. In this rebuttal to the Noakes’ papers, we argue that Noakes has ignored data supporting the existing hypothesis that under normal conditions cardiac output is limiting maximal aerobic power during dynamic exercise engaging large muscle groups. Key Words: MAXIMAL AEROBIC POWER, LIMITING FACTORS, PLATEAU

N oakes (11,12) has undertaken the very important but often thankless task of challenging existing models and beliefs. Specifically, he questions the models of limitations of maximal oxygen uptake and its role in endurance performance. To stress the necessity of such challenges, Noakes cites a number of distinguished scientists. His main points are:

1. A theory or a statement has to be refutable, meaning that one must identify at least one observation that is incompatible with that theory;
2. it is usually unwise to accept a conjecture for which there is already a single logical refutation in the literature;
3. we tend to interpret all information within a certain paradigm, obscuring the true meaning of that information; therefore the paradigm itself should be questioned;
4. information should be judged on its own merits, without considering the status of the one bringing it forward;
5. one should be aware of the distinction between association and causality; and
6. Scientific “truths” are subject to change.

NEW MODEL

Noakes has identified a number of theories/models in exercise physiology that in his opinion become “ugly and creaking” if subjected to an adequate examination. Most of these are included in what he calls “The Cardiovascular/Anaerobic Model of Exercise Physiology and Athletic Performance.” He proposes an alternative model “The Regulated Skeletal Muscle Contraction Model of Exercise Physiology and Athletic Performance.”

According to the former model the oxygen delivered by the heart limits the aerobic power of the muscles. Higher work rates can be maintained because power is yielded anaerobically but only for a limited period of time (seconds to minutes depending on the intensity of exercise). Eventually, the accumulation of anaerobic metabolites will slow down the rate of ATP-generation, thereby impairing muscle performance. The capacity for prolonged work at submaximal intensities is strongly influenced by maximal oxygen uptake. Consequently, changes in maximal oxygen uptake would affect performance in exercise lasting longer than a few minutes.

The latter model states: skeletal muscle recruitment and contractile function are regulated by a hierarchy of controls specifically to prevent damage to any number of different organs. During maximal exercise, progressive myocardial ischemia preceding skeletal muscle anaerobiosis must be thwarted so that neither the heart nor the skeletal muscle develops irreversible rigor and necrosis with fatal consequences. One postulate from that model is that oxygen partial pressure of blood in the vessels of the heart and the heart muscle itself would be independent of altitude. Consequently, peak cardiac output would decrease with increasing altitude.

QUESTIONING MAXIMAL OXYGEN UPTAKE

Noakes argues that maximal oxygen uptake might be a somewhat “elusive” quantity. Therefore, it might not serve very well as a characteristic for the functional capacity of
the cardiovascular system. In line with this reasoning is the observation that oxygen uptake can differ significantly between different modes of exercise, even if the classical conditions are fulfilled (large muscle groups, breathing sea level atmospheric air, and sufficient exercise duration). It should be noted, however, that changing exercise mode rarely changes oxygen uptake more than 15%. Hence, there is an upper limit for the attainable oxygen uptake. Therefore, we need to specify the exercise mode for the sake of comparison, especially when using maximal oxygen uptake to characterize athletes. As suggested earlier by many, it might be better to exchange the term “maximal oxygen uptake” for “peak oxygen uptake.”

**OUR REBUTTAL AND PURPOSE**

In spite of good and justifiable intentions and the fact that Noakes has identified several crucial points that have to be addressed (as well as indicating how these should be addressed), neither the lie of reasoning nor the conclusions are, with a few exceptions, quite convincing. In our opinion, the main reasons for this are:

1. He has not identified unequivocally what observations are compatible and, more importantly, incompatible with different models and theories;
2. he has not evaluated different models according to the same standards; for example, a much heavier burden of proof is placed on theories included in the so-called “Cardiovascular/Anaerobic model…” than on other models and theories;
3. he has excluded vital sources of information and included data, the relevance of which are debatable;
4. he switches between accepting and rejecting paradigms.

Therefore, our main purpose is not to discuss every issue addressed by Noakes. We will briefly comment on some of the more fundamental points to demonstrate the consequence of his reasoning. In addition, we are presenting information excluded by Noakes, information of vital importance to elucidate what models are most compatible with presently available data. Finally, the discussion must be diverted from the route of establishing who is right to enter the route of searching for the “truth.”

**POINT ONE**

The first limitation is that Noakes is not stating clearly what observations would be compatible and incompatible with the models he is evaluating. To successfully evaluate different theories, it is necessary to clearly define different models and theories and to identify the postulates of different models both in terms of compatibility and incompatibility. Part of this problem is demonstrated in Noakes’ discussion about “the role of oxygen delivery in determining endurance performance” and “the role of muscle factors in determining exercise performance” (12). His line of reasoning seems to be based on the assumption that the cardiovascular model postulates that maximal oxygen uptake is the sole determinant of endurance performance. Hence, it should explain fatigue under all conditions. However, few scientists of today would support that type of model because the evidence to refute it has been overwhelming for decades. Thus, it is not fair to claim that this is an established model. So the logic of bringing it up seems to be: if a factor fails to be the only determinant, it is without importance. In fact, it might even be the most important one.

**POINT TWO**

The second problem is the use of different standards. For example, Noakes does not accept that the observed changes of peak oxygen uptake following experimentally induced changes of the volumes of oxygen leaving the heart is really indicating that the capacity of the central circulation is a limitation for peak oxygen uptake (3–5). He argues that it is not proven that other changes which might produce that effect do not occur.

Applying the same standards, it would not be possible to use the absence of a plateau in oxygen uptake as an indication of a regulated cardiac output, in view of the fact that numerous studies have demonstrated the presence of a plateau. Furthermore, Noakes reports that about 50% of all studies, mainly those using incremental procedure, fail to demonstrate a plateau (see below). He devotes time finding plausible explanations why the other studies have, erroneously, found a plateau in oxygen uptake. But there is no discussion about the influence of incremental procedures on the probability to find a plateau, if there is one. The point is that it is absolutely necessary to use the same standards both qualitatively as well as quantitatively. Otherwise, models and theories will not be judged on their merits. The risk of biased opinion is obvious.

**POINT THREE**

The third problem is that relevant information is not included. The main omissions are discussed below.

**The influence of muscle mass on peak oxygen uptake.** In his response Noakes ignores many studies supporting the belief that central circulation limits the peak oxygen uptake during exercise engaging a large muscle mass (13). For example, adding maximal arm exercise to maximal leg exercise does not increase peak oxygen uptake more than expected from widened arterio-venous difference, mainly from the part of the cardiac output that supplied the arm muscles (7,14) (Fig. 1).

Figure 1 presents a good summary of such observations. It is interesting to note that the same work rate and oxygen uptake (4 L·min⁻¹) could be maintained for twice as long when both arms and legs were activated (estimated oxygen demand of about 5 L·min⁻¹). Evidently the heart could tolerate a prolongation of the exercise period when a larger mass of skeletal muscles was activated. Blood pressure was not measured, but there are no reasons to believe that it was
Importance of the arterial oxygen content and pressure for heart muscle performance. Noakes discusses the effect of chronic exposure to high altitude on cardiac output. He points out that at altitude cardiac output is not increased during exercise to compensate for the reduced arterial O2 content. Apparently he is not aware of the study by Stenberg et al. (16) showing that at acute exposure to an altitude of 4000 m there was an increase in cardiac output at submaximal exercise as compared with the data at sea level. During maximal exercise blood pressure, stroke volume and cardiac output were about the same at sea level and at altitude despite a reduction in O2 saturation to 70%. It should be noted that the ECG recordings were all normal. This is incompatible with the theory that the performance of the heart muscle is regulated to avoid ischemia.

In accordance with the conditions regarding the heart muscle, the fact that running performance is higher at altitude, aiming at avoiding muscle ischemia.

POINT FOUR

Noakes has included information on the correlation coefficient between maximal oxygen uptake and running performance. However, he has chosen studies with a quite small running performance range. Based on the absence of a significant correlation, he argues that one cannot infer that maximal oxygen uptake is important for endurance performance. However, using a small range in the dependent variable will inevitably decrease the correlation coefficient. Very often the number of observations are small. Hence, the probability of finding an insignificant result will increase while the chances of detecting a true relationship will decrease.

Current models explaining endurance performance do not attribute differences in performance to one single factor. Hence, the postulates are that a given level of endurance performance can be achieved by persons who differ in peak oxygen uptake, ability to work close to that peak, or mechanical efficiency, for example. Thus, a given peak oxygen uptake can produce different levels of performance.

ENDURANCE PARADIGM

An established paradigm is that muscle cells, skeletal as well as heart, need oxygen to produce power for extended periods of time. A higher power production requires more oxygen. A study published by Karlsson et al. (9) determined the treadmill speed that exhausted the well motivated subject at 4.0 min. In five of six subjects the speed could be reduced to 75–80% of that maximum without a significant decline in oxygen uptake. How was the energy demand of the additional 20–25% in speed covered if not by anaerobic processes?

Noakes seems to agree with the notion that oxygen is needed, but he does not acknowledge this in the discussion of the role of oxygen delivery in determining endurance performance. If he had, he would have appreciated that a low peak oxygen uptake is insufficient to produce high levels of power during prolonged exercise. Unless the model that states “more power is required to run faster” is rejected, one must accept that the probability of sustaining a high running speed for more than a few minutes increases with increasing peak oxygen uptake.

METHODOLOGICAL CONSIDERATIONS

Noakes quotes C. Taylor (17) “. . . In fact in 50% of cases no deviation in the linear increase of oxygen intake occurs at exhaustion.” Noakes did not continue the quotation: “. . . and in the remaining cases this value accelerated more often than fell off ” (italics by the present authors). This is an unusual response, a finding that can raise questions about the applied protocol.

Why not also quote H. L. Taylor et al. (18): “Only 7 men in 115 subjects have failed to reach the criteria established for the plateau of oxygen intake.” Their protocol
was running at a fixed speed and a 2.5% increase in grade of the treadmill every third minute up to exhaustion. The expected increase in oxygen uptake per grade increase was on average 4.18 ± 1.07 mL·kg⁻¹·min⁻¹. The criteria that peak oxygen uptake was reached was a recorded increase in oxygen uptake by less than 2.1 mL·kg⁻¹·min⁻¹. Improved running economy cannot explain the plateau. It should have been observed also at submaximal running speeds.

The coefficient of reliability was 0.95 in 69 test-retest determinations. Twelve men whose physical activity did not vary widely were tested five times over a period of 1 yr. The measured peak oxygen update was remarkably constant. Noakes does not comment on Figure 3 in Bassett and Howley’s paper (2) showing a definite plateau in peak oxygen uptake in six out of nine subjects tested on a cycle ergometer.

WHAT LIMITS MAXIMAL CARDIAC OUTPUT?

A crucial question is: what factor limits cardiac output during maximal exercise? Stroke volume is the main difference between different well-trained individuals during maximal exercise. Stroke volume is well maintained or even increased (10) during maximal exercise compared with submaximal work rate, indicating that the oxygen supply to the heart is adequate. Furthermore, a plasma expansion can increase stroke volume and thereby cardiac output compared with peak values attained before expansion. Kanstrup and Ekbloom (8) found that plasma expansion by infusion of dextran (on average a 700 mL increase in plasma volume) reduced the hemoglobin (Hb) concentration, but normal peak O₂ could be reached because an increase in stroke volume and cardiac output compensated for the reduced Hb concentration and therefore O₂ content per milliliter of arterial blood. These observations have been confirmed by Kirp et al. (10). An increase in preload can lead to an increased end-diastolic volume of the heart. In the former experiments the mean arterial blood pressure was slightly reduced after plasma expansion. Hammon et al. (6) tested the hypothesis that the pericardium, by restricting heart size, limits maximal cardiac output and oxygen consumption. Pigs who perform maximal treadmill running after a pericardectomy increased both maximal cardiac output (29%) and maximal oxygen uptake (31%) after the operation. These results were associated with an estimated 33% increase in end-diastolic volume. The conclusion was that the limit to cardiorespiratory performance during exercise engaging large muscle groups lies not in oxygen utilization in the peripheral muscles, but in oxygen delivery from the heart. Furthermore, removal of pericardium is associated with myocardial hypertrophy.

Noakes must be credited for restarting the relevant discussion about limiting factors for maximal oxygen uptake and other questions regarding endurance performance and also for reminding us of fundamental scientific principles. However, Noakes’ line of reasoning has not honored those principles very well. Rather, his two papers (11,12) demonstrate the consequences of violating those fundamental principles. Therefore, neither his evaluation of “classical versus contemporary viewpoints” nor his rejection of what he calls “the cardiovascular/anaerobic models” becomes convincing. To achieve better insight we need to follow Noakes’ advice: to critically examine available data and to do that according to sound scientific principles. Otherwise, the risk of biased conclusions is evident. The consequences may be the survival of false theories as well as the rejection of true hypothesis.

REFERENCES


Address for correspondence: Bjorn Ekbloom, P.O Box 5626, S-114 86 Stockholm Sweden. E-mail: bjorn.ekblom@fyfa.ki.SE.