The following is the abstract of the article discussed in the subsequent letter:

Siegmund, Gunter P., Michael R. Edwards, Kelly S. Moore, Dale A. Tiessen, David J. Sanderson, and Donald C. McKenzie. Ventilation and locomotion coupling in varsity male rowers. *J Appl Physiol* 87: 233–242, 1999.—Ventilation and locomotion coupling (entrainment) has been observed and described in rowers during incremental exercise protocols but not during simulated race conditions. The purpose of this descriptive study was to examine ventilation and locomotion entrainment on a breath-by-breath and stroke-by-stroke basis in varsity male rowers during a maximal 2,000-m ergometer test. Eight of eleven rowers entrained ventilation at integral multiples of stroke rate (1:1, 2:1, or 3:1) for at least 120 consecutive seconds, with a 2:1 entrainment pattern being most common. In all 2:1-entrained subjects, inspiration occurred at catch and finish and expiration occurred during the latter portions of drive and recovery. In entrained and unentrained breaths from all rowers, peak flow rates and tidal volumes varied depending on when the breath was initiated during the stroke cycle. Entrained rowers made use of these differences and breathed in a pattern by which they avoided initiating breaths that resulted in reduced tidal volumes. The present data provides hints at certain distinct mechanisms that may induce switches between different modes of entrainment (1). In particular, amplitude and phase relations between respiration and movement affect the stability of specific frequency locking ratios and may therefore be seen as the corresponding bifurcation parameters. When searching for a maximal energy transfer, these parameters have to be adjusted properly. If the adjustment is limited and, consequently, the proper frequency relation can no longer be maintained, then the intrinsic structure of lung pressure modulation readily implies a spontaneous switch to another (sub)optimal rational frequency locking state because integer ratios always reflect (local) maxima of effective oxygen concentration.

REFERENCES


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REPLY

To the Editor: In their letter, Huys et al. have reanalyzed data from our study (6) in an attempt to elucidate a “causal principle” for LRC. They suggest that “optimization of the effective oxygen concentration can be seen as the driving mechanism that forces respiration to synchronize with rhythmic movement.” The original study showed that all rowers demonstrated LRC for some portion of the rowing test; the pattern of LRC was variable between subjects (different integral patterns; 1:1, 2:1, and 3:1) and was altered at different points during the performance task. The rowers appeared to alter their ventilation to match locomotion. The data from this descriptive paper did not permit more than speculation on the underlying causes of LRC.

The model proposed by Huys et al. fails to consider many alternate possibilities, and the notion that optimization of the oxygen concentration in the lung drives LRC appears overly simplistic given the complexity of the relationship between exercise and ventilation. The mechanisms that mediate the ventilatory responses to exercise have been studied for more than a century and remain controversial. Forster (3) suggested
this was because investigators have not yet devised an ideal preparation for its study. Most investigators have agreed on a three-component model to explain the regulation of exercise ventilation: a central medullary rhythm generator/integrator, neural inputs into this integrator from higher locomotor areas of the central nervous system and from the periphery, and the regulation of the distribution of efferent motor output to the muscles of respiration.

Although LRC has been documented in humans engaged in a variety of exercise modalities and in many exercising mammals, little is known about the neural or biomechanical basis for it (1). Evidence for a locomotor-linked neural stimulus to hyperpnea has emerged from animal models with simulated locomotion (2, 5). A feed-forward mechanism ("central command") originates in locomotor areas of the higher central nervous system; this is capable of producing parallel activation of medullary respiratory neurons and motor pathways to limb locomotor muscles and requires no feedback from the periphery. However, direct evidence for central command activation of ventilation during dynamic exercise in humans is lacking, although the rapid increase in ventilation at the onset of exercise supports the concept. Another locomotor-linked ventilatory stimulus is related to the chemical and mechanical conditions of the working muscle. Stimulation of thinly myelinated (group III) or unmyelinated (group IV) muscle afferents provokes powerful ventilatory and circulatory effects. These slowly conducting afferents respond to mechanical, chemical, and thermal stimuli (4).

Clearly, the causal mechanism for LRC in humans is complex and not well understood. Although Huys et al. have proposed one possible mechanism, it is not clear how this fits into the general model of control of exercise hyperpnea.

REFERENCES

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