Last Word on Viewpoint: Evidence that reduced skeletal muscle recruitment explains the lactate paradox during exercise at high altitude

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TO THE EDITOR: Surely the art of science is to see the complete picture. Some respondents focus on the findings that peak blood lactate concentrations and muscle activation are reduced in hypoxia and ignore the related phenomenon that exercise in hypoxia terminates at low cardiac outputs and blood lactate concentrations. I provided a unifying explanation for these phenomena.

Dr. Marcora (see Ref. 3) states that many intracellular factors, not just the extent of skeletal muscle recruitment, determine lactate production and removal in hypoxia; Professors Grussi and Perrey (see Ref. 3) make essentially the same point. Professor Kayser and, I suspect, Professor Perrey agree that all the phenomena I describe can be explained by a common determinant—reduced central motor command.

Dr. Amann (see Ref. 3) focuses on phenomena that occur near the end of exercise not at its beginning. He, Dr. Lundby, and to some extent, Professor Richalet (see Ref. 3), misunderstand my argument. A focus on the factors associated with the termination of exercise is a natural consequence of the mindset imposed by the VO2max test. Testing for VO2max has produced a brainless model of exercise physiology (4).

Dr. Amann found that within seconds of exposure to a reduced FiO2, skeletal muscle activation and muscle power output fell (1). These changes occurred too rapidly to be explained by direct effects of skeletal muscle or cardiac hypoxia as proposed by Professor Richalet. Rather they must be due to a central (neural) control mechanism acting in an anticipatory manner (5). Similarly the exercise intensity chosen (by the brain) at the onset of exercise will determine when that exercise bout ends. Studies of the anticipatory factors determining the initial exercise intensity as well as the response to interventions imposed acutely during exercise, are likely to be more revealing than is a continuing focus on factors associated [not necessarily causally (6)] with exercise termination.

Dr. Lundby argues that the premature termination of exercise at altitude due to “different fatigue mechanisms” explains the lactate paradox. He does not detail those mechanisms. Professor Wagner (see Ref. 3) believes that my interpretation lacks experimental support. He questions whether there is proof that a falling cerebral O2 availability causes the brain to direct “the legs to quit.” Professor Nybo’s (see Ref. 3) work has established that “it is correct that (cerebral) hypoxia may impair muscle recruitment (by the brain).” Professor van Lieshout (see Ref. 3) finds this explanation “attractive,” while Professor Marino (see Ref. 3) considers it “the only logical explanation.”

Professor Brooks (see Ref. 3) correctly points out that I overlooked the article by Reeves and colleagues. He argues that “ . . . the presence of the Lactate Paradox during maximal exercise at altitude is one of the strongest demonstrations of a central limitation to exercise performance.” He also writes that “It is perhaps regrettable that the brain’s role in exercise physiology has been overlooked.”

That summarizes what I am trying to say (4, 6).

REFERENCES