TO THE EDITOR: We posed the question: Can peripheral mechanisms explain why exercise terminates at submaximal levels of skeletal muscle recruitment (activation) as shown by Dr. Ekblom and his colleagues (1, 2). Few respondents addressed that challenge.

Dr. Shephard’s (see Ref. 8) response is littered with errors of fact; space limitations allow us to address just three. A. V. Hill studied neither myocardial ischemia nor ultramarathon runners; Professor Whyte’s paper addresses the development of myocardial fibrosis, not ischemia, in some ultraendurance athletes. There is indeed hard evidence, no longer possible to ignore, that at least some forms of exercise are regulated “in anticipation” by a complex, intelligent system—the central governor model (CGM) (5, 8).

Dr. Foster (see Ref. 8) does not really disagree with what we wrote. Dr. Bassett’s (see Ref. 8) assertion that we have not identified specific components of the CGM is wrong, a red herring. Dr. Ekblom’s group did not show that “EMG activity and the work of the heart continue to increase” at supramaximal work rates. Instead subjects terminated exercise at <100% skeletal muscle activation in their exercising limb muscles. This is the key finding supporting the CGM.

Dr. Marcara (see Ref. 7) argues that exercise is regulated by the conscious rather than the subconscious brain: the truth will become apparent in the fullness of time. Dr. González-Alonso (see Ref. 8) believes that a plateau or drop in cardiac output and blood flow to active muscles and brain precedes the attainment of $V\dot{O}_{2\text{max}}$ in trained humans. If true the question remains: What causes the termination of exercise if not a regulated neural control whose function is to limit the extent of skeletal muscle recruitment and therefore to prevent excessive demands on a failing heart and circulation? What is the purpose of continuing to exercise with the same amount of active skeletal muscle while cardiac output and blood flow are declining and myocardial ischemia is developing? The critical question is whether there is evidence for an anticipatory regulation of homeostasis as predicted by the CGM or whether the human body always exceeds its homeostatic capacity before exercise terminates as Dr. González-Alonso believes. Evidence for the former interpretation exists (5, 7, 8) but other than the studies of Dr. González-Alonso and colleagues, we are not aware of evidence supporting the latter belief.

We find much agreement with Dr. Cheung’s (see Ref. 8) enlightened views. The CGM does not exclude the central circulation as a source of sensory feedback to the CGM, a point not properly understood by Dr. Kemi (see Ref. 8). Dr. Calbet (see Ref. 8) has provided some of the best evidence that exercise performance cannot be regulated exclusively by the extent of oxygen delivery to the exercising limbs. Thus increasing oxygen delivery to muscle does not improve maximal exercise performance either at altitude (4) or at sea level (3); hence the termination of maximal exercise cannot be due to an inadequate oxygen delivery to muscle. We cannot force Dr. Calbet to comprehend that which he still cannot see (6).

REFERENCES