The following letters are in response to the Point:Counterpoint series “The muscle metaboreflex does/doe not restore blood flow to contracting muscles” that appeared in the January issue (vol. 100: 357–361, 2006; http://jap.physiol.org/content/vol100/issue1).

To the Editor: Although we agree with O’Leary and Joyner (5) that “...no study has quantitatively addressed the above question in humans...” and that “...an unambiguous experimental model in humans is needed...” we think it worthwhile to discuss further the possible mechanisms underlying the discrepancy between the results previously obtained in humans and dogs. In most of those earlier studies, the arterial baroreflexes (ABRs) were intact, which means that results obtained during activation of the muscle metaboreflex (MMR) were certainly affected by the ABRs and require careful interpretation.

In dogs, sinoaortic denervation reportedly shifts the mechanism of MMR-induced pressor responses from mainly an increase in CO to increases in both CO and peripheral vasoconstriction (3), suggesting ABRs preferentially inhibit MMR-induced vasoconstriction. During postexercise ischemia (PEMI) in humans, by contrast, there are no increases in stroke volume or HR, but an index of cardiac contractility and arterial pressure are both increased (4), suggesting ABR-mediated suppression of HR may inhibit increases in CO during MMR. Moreover, the period of MSNA suppression evoked by carotid baroreflex loading (neck suction) was shortened (2) and the increase in leg vascular conductance was diminished (1), suggesting that ABR-induced suppression was overridden by MMR-induced vasoconstriction. Taken together, these findings suggest that the influence of ABRs on cardiovascular responses during MMR differs in dogs and humans. We therefore suggest that the discrepancy seen between dogs and humans reflects at least in part the difference in the interactions between ABR and MMR.

REFERENCES


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To the Editor: This argument could well hinge on species difference: the affirmative presentation by Professor O’Leary (3) used dogs and the negative presentation used humans during exercise, both undergoing partial occlusion of the flow to the skeletal muscles of one leg. In the dog, there was a secondary increase in cardiac output resulting in a partial restoration of flow to the occluded, exercising leg. In the human model by Professor Joyner (3), the secondary response was vasoconstriction without increased cardiac output and no restoration of flow to the occluded, exercising leg.

I suggest that a reflex induced by the arterial occlusion—not necessarily the metaboreflex—produces a general sympathetic response that includes contraction of the dog’s muscular spleen (1). We used awake, intact dogs, and induced splenic contraction by epinephrine, fright, and hemorrhage. The spleen contracted within 7 s of the stimulus; flow in the splenic vein increased within 12 s, associated with an increase of 16 points in hematocrit (45–66%). The hematocrit of a systemic vessel increased 24 s later by 5 points (45% resting to 50%); see Fig. 1 and Table 1.

Humans do not have this perfect venous reservoir (2) and lack the means of restoring blood flow to ischemic, contracting muscles by increasing cardiac output. So, both Point and Counterpoint were correct for the species they used. The resolution of this Point:Counterpoint could come from a repeat of the dog experiments in chronically splenectomized dogs.

REFERENCES


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To the Editor: At low dynamic workloads, when cardiac output (CO) reserve is high, the pressor response induced by experimentally restricting blood flow to working muscles is mediated by an increase in CO (3). In this situation, the increase in CO may increase active muscle blood flow and improve oxygen delivery (5). However, at low dynamic workloads, a large reduction in blood flow is required to elicit a pressor response. Thus the metaboreflex is not tonically active at low dynamic workloads. At higher dynamic workloads, when CO reserve is low, smaller reductions in muscle blood flow are required to elicit a pressor response. However, in this situation, the pressor response is mediated by an increase in vascular resistance. The vasoconstriction must occur in the active skeletal muscle because this is the area of highest vascular conductance. Vasconstriction to active muscle likely exacerbates the blood flow-metabolism mismatch. Thus the metaboreflex could, if it were active at low dynamic workloads, improve oxygen delivery. However, it is not active at low dynamic workloads. Furthermore, the metaboreflex likely does not correct for the blood flow-metabolism mismatch at higher workloads when the pressor response is mediated by vasoconstriction in the active skeletal muscle.
Restricting blood flow to active muscles may alter the effort required to perform work (1, 2). Subjects reported that the sense of effort required to produce the target force during rhythmic forearm exercise increased when forearm blood flow was occluded. Thus the role of central command in mediating the pressor response should also be acknowledged (2, 4).

REFERENCES


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