The Ultimate Goal in Neural Regulation of Cardiovascular Function Revisited

TO THE EDITOR: This letter is sequel to an article (8) previously published by me and my colleagues in this journal. In that article (8), the following multiple-choice question (MCQ) was raised:

In health, neural cardiovascular reflex mechanisms are geared toward maintaining:
A. Cardiac output
B. Total peripheral resistance
C. Arterial blood pressure
D. Tissue blood flow

Using a couple of simple examples, the point made was that, in health, the ultimate goal in the neural regulation of cardiovascular function is the maintenance of arterial blood pressure (BP) within a range in which tissues are able to maintain their blood flows by autoregulation. I now believe that this notion is simplistic. For instance, during exercise, products of metabolism in active skeletal muscle initiate activity in sensory neurons, leading to increases in efferent sympathetic outflow and BP. There is experimental evidence that this reflex, called the muscle metaboreflex, restores oxygen delivery and blood flow to exercising skeletal muscle (5, 6). Although cardiovascular regulation during exercise was discussed in our article (8), this point was missed. Viewed philosophically in terms of the ultimate goals of mechanisms, it would thus be more appropriate to conclude that the main purpose of cardiovascular mechanisms is to ensure adequate blood flows to all tissues commensurate with their metabolic requirements. The rise in sympathetic activity that was missed. Viewed philosophically in terms of the ultimate goals of mechanisms, it would thus be more appropriate to conclude that even neural mechanisms are eventually oriented toward enabling tissues to maintain their blood flows commensurate with their metabolic requirements. The rise in sympathetic activity that produces vasoconstriction in nonexercising muscle and viscera effectively serves to redistribute flow to exercising skeletal muscle. Second, the vasoconstricting effect of the rise in muscle sympathetic nerve activity in active skeletal muscle is attenuated by the local vasodilatory effect of hypoxia (4) and other products of metabolism that accumulate in active exercising skeletal muscle.

The conclusion stated above is even more obvious when a wider spectrum of pathophysiological states is considered, and such consideration is in fact necessary to speculate upon the ultimate purposes of mechanisms. For example, the response to intense cerebral ischemia consists of an increase in sympathetic outflow to resistance vessels, thereby elevating arterial BP and cerebral perfusion pressure and consequently serving to restore cerebral blood flow. This is the central nervous system (CNS) ischemic pressor response (3). This mechanism may also be responsible for the acute BP elevation following stroke (7). When intracranial tension is elevated, ischemia of the vasomotor center leads to a reflex increase in sympathetic activity, and if baseline BP were normal, this would result in acute hypertension (Cushing’s reflex). Evidently, the CNS ischemic pressor response and Cushing’s reflex (which is a special case of the CNS ischemic pressor response induced by raised intracranial tension) are concerned with restoring cerebral blood flow and not maintaining arterial BP. It is possible for me to recall that in the classroom session that led to the writing of our 2004 article (8), my student Bharathi Balachander spoke about Cushing’s reflex as an example to consider when we discussed the MCQ mentioned in that article.

In 2005, Fink (1) put forth the hypothesis that the long-term level of arterial BP may just be an emergent parameter in a mainly decentralized control system oriented toward maintain-
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Bharathi Balachander is currently a final-year undergraduate medical student at Jawaharlal Institute of Postgraduate Medical Education and Research (Pondicherry, India).

REFERENCES


E. S. Prakash
School of Medicine
Faculty of Medical and Health Sciences
Asian Institute of Medicine, Science and Technology
Bedong 08100, Kedah Darul Aman, Malaysia
E-mail: dresprakash@gmail.com
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