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 hypoxic/ischemic 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 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 produces vasoconstriction in nonexercising muscle and viscera to maintain their blood flows commensurate with their metabolic needs.

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 maintaining the blood flow demands of various tissues. Although this theory was put forth with the idea of suggesting a means of understanding of what determines the long-term level of BP, it may well be carefully applied to understand the short-term regulation of BP as well, for instance, BP changes during exercise.

I recently administered the case given below (to second-year undergraduate medical students in our university doing the Bachelor of Medicine and Bachelor of Surgery course) to determine if they understood what the ultimate goal of all cardiovascular regulatory mechanisms is. These students had had a series of lectures on the cardiovascular, respiratory system, and gastrointestinal system. The case reads as follows:

A 40-yr-old apparently healthy man suddenly loses consciousness. During this episode, his brachial artery BP is 120/80 mmHg, average heart rate is 72 beats/min, average cardiac output is 5 l/min, blood hemoglobin concentration is 15 g/dl, arterial blood O2 content is 200 ml/l, and plasma glucose concentration is 100 mg/dl. He did not sustain any head injury. During the episode or following it, he did not have convulsive movements, and his ECG was essentially normal. Doctors rule out psychological causes for symptoms. They are completely puzzled. Every time such an episode occurs, fortunately, the patient regains consciousness within a minute or so.

I am aware that this is a low-fidelity problem, but this is intentional as well as necessary to make a point; for example, it is implied that one of these episodes was witnessed, and cardiac output was measured during the episode. I then invite students to ask me if they need any more information on this patient in order to arrive at a generic solution. And when I have them wondering what the mechanism of transient loss of consciousness in this patient may be, I present the following straightforward MCQ.

Select the single best response:

The ultimate goal of all cardiovascular regulatory mechanisms would be to maintain:
A. Arterial blood pressure
B. Cardiac output
C. Total peripheral resistance
D. Tissue blood flow

Six students chose A, 9 students chose B, 3 students chose C, and 36 students chose D, the answer I contend is the most appropriate. When I asked what they thought was the most likely basis for syncope in this patient, they gave the correct answer at once: a rise in cerebral vascular resistance in the face of apparently normal BP leads to cerebral hypoperfusion and transient loss of consciousness. This entity is called cerebral syncpe (2). Furthermore, one may reasonably speculate that the CNS ischemic pressor response is recruited to restore cerebral blood flow when syncope occurs.

A distinct advantage of this case is that it could be used right at the start of a lecture or a series of lectures on cardiovascular regulation because it requires little prior knowledge of the actual mechanisms themselves. I believe (although I did not pretest this group of students with this MCQ) that the case presented here would be useful to enable students construct the concept that the ultimate goal of all cardiovascular regulatory mechanisms is to ensure adequate blood flows to all tissues commensurate with their metabolic requirements.
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REFERENCES


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