Gold Standard in Assessing Baroreceptive Function

To the Editor:

In their recent article1 Lipman et al pursue the ambitious goal of defining, beyond any doubt, the “gold standard” for evaluating baroreflex gain. In their hands, the active engagement of the system accompanying the intravenous injection of a pressor drug was more closely related to carotid distensibility, considered as the input of an open-loop system, than any other index derived from spontaneous oscillations of heart period and arterial pressure. This appeared particularly evident in the presence of a low baroreflex gain. Unfortunately this mechanistic certainty does not reflect the physiological complexity of neural modulation of heart period.

The aortic arch and the carotid sinus do not represent the only reflexogenic areas, but only the ones projecting to the dorsal medulla (and, also in this regard, we would add the cardiac chambers). In addition, there is an entire population of afferent fibers, innervating the cardiovascular system and projecting to the spinal cord, usually referred to as “afferent sympathetic fibers.” These afferent fibers, erroneously considered for numerous decades to transmit only signals related to pain perception, are spontaneously active in normal hemodynamic conditions, are strongly excited by stretch, and mediate reflexes mainly excitatory in nature with positive feedback characteristics.2,3

By now it appears more than plausible that a normal baroreflex event is the result of an interaction of central integration, negative-feedback and positive-feedback reflex modulations.2,4 As an experimental proof, the baroreflex bradycardia induced by an aortic pressure rise is increased after section of thoracic dorsal roots, interrupting the course of afferent sympathetic fibers and thus abolishing the excitatory reflex component participating in the baroreflex.5 Negative-feedback mechanisms seem to normally prevail in conditions of quiescence, while positive-feedback mechanisms may become stronger or even predominant during physical exercise, emotion, or in some pathophysiological conditions such as arterial hypertension or acute myocardial ischemia.2

It is quite obvious that we do not yet have adequate tools to discriminate the play of the various components of this complex interaction in normal closed-loop conditions. However, it is likely that the various experimental approaches provide complementary information on baroreflex function and that each of them suffers from some shortcoming.

It may be that an artificial pressor stimulus, in resting conditions, may solicitate more effectively the supraspinal negative feedback mechanisms while the normal oscillations may be more strictly linked to respiration and reflect more adequately the normal interaction of inhibitory and excitatory components. But the question is: given this complexity which quality of gold is that provided by forcing one mechanism probably beyond its normal play? And which quality of general view is that requiring to disregard acquired notions of anatomy and physiology? Perhaps the wiser attitude in front of complexity is to attempt to evaluate its multifarious patterns rather than performing over-simplifications that would only jeopardize future research and creative thinking.

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