Why do we have both peripheral and central chemoreceptors?

The peripheral chemoreceptors, the carotid (and aortic) bodies, detect arterial hypoxemia and stimulate breathing. At normal arterial PO2 (Pao2) values, they provide a tonic excitatory input to the brain stem (6), and with hypoxia they respond dramatically as PaO2 falls below 70 Torr. Thus, for oxygen, they provide an emergency detection system not a breath-by-breath measure of gas-exchange success.

Both the carotid bodies and the central chemoreceptors detect changes in CO2/pH and affect breathing. What are their respective roles and importance? These are old questions (see Refs. 3, 5, and 7 for historical perspective), but the paper by Smith et al. (9) in this issue of the Journal of Applied Physiology provides unique data obtained from conscious dogs that support clear answers to them. The dogs have one carotid body denervated (the main peripheral chemoreceptor in the dog) and the other perfused with blood under the control of the investigators. This maintains the tonic input from the carotid body and avoids any time-related changes that occur in animals after denervation (6). They induce a near-square-wave increase in end-tidal PCO2 of 10–12 Torr at J both the carotid body and the central chemoreceptors (“CB + Central”) or 2) only the central chemoreceptors (“Central Only”) with the carotid body simultaneously perfused with blood of normal Po2, PCO2, and pH. There are two protocols: 1) several 1- to 2-min exposures, and 2) 5-min steady-state exposures. The results are remarkable and clear. With Central Only stimulation, the response (a value of ventilation >3 SDs above baseline) was delayed by 11.2 s in the rapid transient protocol, but in the steady-state experiments the response was 63% of that with CB + CCR stimulation. The carotid bodies provide the rapid response, but the central chemoreceptors provide most of the steady-state response.

The greater role of the central chemoreceptors in the steady-state response is in good agreement with a large number of previous studies that used either acute carotid body denervation or separate perfusion of the two sites in anesthetized animals. The greater role of the carotid bodies in the rapid response complements their earlier results linking transient hypocapnia or separate perfusion of the two sites in anesthetized animals. This view gives serious purpose to central chemoreceptors and may explain why they are located at many sites (8). They are chemoreceptors detect arterial CO2, cerebral blood flow, and cerebral metabolism. This view gives serious purpose to central chemoreceptors and may explain why they are located at many sites (8). They are not a mere back-up for the carotid body, which seems perfectly adequate as a detector of arterial CO2 in physiological conditions, are especially suited for rapid responses (3, 4, 9), and provide a needed tonic drive under normal CO2/pH conditions (6).

REFERENCES


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