Contribution of carotid chemoreceptors to mesenteric venoconstriction during acute hypercapnia in rabbits

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Tominaga, Masamune, Thomas A. Stekiel, Zeljko J. Bosnjak, and John P. Kampine. Contribution of carotid chemoreceptors to mesenteric venoconstriction during acute hypercapnia in rabbits. Am. J. Physiol. 277 (Heart Circ. Physiol. 46): H2305–H2310, 1999.—The contribution of carotid chemoreceptors to hypercapnia-induced mesenteric venoconstriction was examined in 12 α-chloralose-anesthetized rabbits (1.0–1.6 kg). Surgical preparation consisted of a tracheotomy, femoral arterial and venous cannulation, and a midline laparotomy through which a 13-cm loop of ileum was exteriorized and superfused with physiological salt solution. Mesenteric vein diameter and intravenous pressure (using a servo-null measurement system) were measured in 500- to 1,000-µm mesenteric veins during 40-s periods of 15%, 20%, and 25% CO2 inhalation. Measurements were then repeated following bilateral ablation of the carotid chemoreceptors. Before denervation, mesenteric vein diameter constricted 6.5 ± 1.1%, 11.9 ± 1.1%, and 17.9 ± 2.2% during the 15%, 20%, and 25% CO2 inhalation, respectively. After denervation, these values were reduced to 5.0 ± 0.9%, 6.9 ± 1.2%, and 8.4 ± 1.3%, respectively. We conclude that activation of the carotid chemoreceptors by hypercapnia induces active mesenteric venoconstriction. After denervation of the carotid baroreceptors and chemoreceptors, there was also a small decrease in venule diameter proportional to the level of inspired CO2. We further conclude that noncarotid body chemoreceptor activation contributes to mesenteric venular constriction.

Methods

Animal preparation. After obtaining approval by the Animal Care and Use Committee, we used 12 New Zealand White male rabbits (1.0–1.6 kg) for this study. The animals were anesthetized with thiamylal (10–20 mg/kg iv) via a 22-gauge cannula in the ear vein. Anesthesia was maintained by continuous infusion of α-chloralose (12.5–25 mg/h iv). Surgical preparation consisted of a tracheotomy, femoral arterial and venous cannulation, and a midline laparotomy. Local infiltration with 3–5 ml of 1% lidocaine was also administered (via a 25-gauge needle) in the surgical incision sites. Ventilation was controlled with a Harvard model 665 animal respirator (Harvard Apparatus, South Natick, MA). Between experimental periods of hypercapnia, the animals breathed a 70% nitrogen and 30% oxygen gas mixture at 2.5 l/min. The use of 30% fractional inspired oxygen concentration (FIO2) rather than room air was to avoid any unanticipated hypoxia between and especially during the periods of imposed hypercapnia. Hypoxia itself causes reflex venoconstriction (19), and if present, it would interfere with the reliability of the data in the present study. Intravenous injections of vecuronium bromide (0.1 mg/kg) were administered to prevent spontaneous ventilatory efforts and to eliminate the effects of hyperventilation on the cardiovascular system during the imposed hypercapnia. Inspired and endtidal CO2, O2, and N2 were continuously measured by a Perkin-Elmer medical gas analyzer mass spectrometer (model 1100, Perkin-Elmer, Pomona, CA). Arterial blood gas values were measured intermittently throughout each experiment using a Radiometer Copenhagen model ABL-1 blood gas device (Radiometer Copenhagen, Copenhagen, Denmark). Between periods of experimentally imposed hypercapnia, pH

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and arterial PCO2 (Paco2) were maintained within the physiological range (pH 7.35–7.45 and Paco2 30–40 mmHg). All rabbits received a basal infusion of 1 meq/h of NaHCO3 to offset the metabolic acidsosis that tended to develop during chloralose administration. Rectal temperature was monitored with a thermistor probe and maintained at 36.5–37.5°C throughout the experiment using a water-perfused heating mat. In six rabbits, bipolar stainless steel electrodes were fixed to a postganglionic splanchnic nerve trunk using a small amount of Wacker Silgel (Wacker-Chemie, Munich, Germany). In each animal studied, a 13-cm segment of ileum was exteriorized and mounted in a temperature-regulated superfusion chamber (13). The exteriorized intestine and intact mesentery were continuously superfused with physiological salt solution (PSS) (37–38°C) with the following concentrations (in mM): 118.4 NaCl, 5.9 KCl, 3.3 CaCl2, and 25.0 NaHCO3. The solution was aerated continuously with 5% O2-5% CO2-90% N2 gases to maintain pH between 7.35 and 7.45. This medium reproduces the ionic and gaseous environments found in the peritoneal cavity (3). The mesentery was pinned down to the Silastic chamber floor with 75-μm-diameter stainless steel pins to prevent movement artifact. Short segments of mesenteric veins (500–1,000 μm in diameter) were cleared of excess fat tissue when necessary and used for diameter measurement.

Measurements. Arterial pressure was measured directly through the femoral arterial catheter. The interval between sequential systolic arterial pressure signals was measured in conjunction with a timer module, and a digital-to-analog converter was used to produce a continuous measurement of heart rate. A model 900 Servo-null (World Precision Instruments, New Haven, CT) pressure-measuring system was used to determine the intravenous pressure at the same site where the vein diameter was measured (21). Glass micropipettes were beveled to 5–7 μm in outer diameter, filled with 2 M NaCl, and used as the sensing electrodes.

Continuous measurements of mesenteric vein diameter were obtained with an on-line videomicroscopy system utilizing a model 2011 video camera (RCA Lancaster, PA) mounted on the side arm of a modified Reichert Stereo Star Zoom stereo microscope (Cambridge Instruments, Buffalo, NY). The system was described in detail by Bell et al. (2).

Sympathetic nerve activity (SENA) from a postganglionic splanchnic nerve was recorded via the bipolar electrodes referred to in Animal preparation. A moving time average technique was used (10). It involved full-wave rectification of the differentially amplified (1,000×) nerve activity signal, which was band pass filtered (4th-order Butterworth set at 0.1–1 kHz). This smoothed the spikes into a time-varying voltage that was representative of the neurogram (10) every 2 s (which represented the averaging interval). The amplitude of this varying voltage was proportional to the summation of changes in amplitude, frequency, and duration of individual depolarization bursts within that segment of multifiber nerve tissue. A zero level of activity was referenced to the baseline SENA level elicited after hexamethonium infusion (10 mg/kg) at the end of the experiment to quantify background "noise" in the recording system.

Aortic pressure, heart rate, vein diameter, intravenous pressure, and SENA were recorded on a Vetter model 620 digital videocassette recorder (A. R. Vetter, Rebersburg, PA) and subsequently printed on a model MT9500 eight-channel recorder (Astro-Med, West Warwick, RI) for later analysis. Experimental protocols. Changes in heart rate, aortic pressure, mesenteric intravenous pressure, mesenteric vein diameter, and sympathetic efferent nerve activity were recorded in response to 40-s periods of 15%, 20%, and 25% inspired CO2 administered in random order. The 30% Fio2 was maintained throughout each experiment, including during periods of hypercapnia. Each period of imposed hypercapnia was separated by a 10-min reequilibration period during which time all measurements returned to baseline values. Subsequently, carotid chemoreceptor denervation was produced as follows. The carotid bifurcations were dissected free from excess fat tissue with forceps, and the area was ablated with electric cautery at low currents. Carotid body ablation was accomplished by denervation of carotid baroreceptors as well. Therefore, the adequacy of the denervation process was confirmed by verifying the elimination of a baroreflex-mediated blood pressure increase in response to a 30-s period of bilateral carotid occlusion, which was present before denervation. After carotid sinus denervation, changes in heart rate, blood pressure, intravenous pressure, mesenteric vein diameter, and sympathetic efferent nerve activity were again recorded in response to sequentially administered 15%, 20%, and 25% inspired CO2 as described above.

Arterial blood could not be sampled each time hypercapnia was administered, because blood sampling from the arterial cannula interfered with heart rate, blood pressure, intravenous pressure, and mesenteric vein diameter measurements. Therefore, the same sequence of acute graded hypercapnia was repeated at the end of each experiment, and arterial blood was sampled at each of the three levels of inspired CO2. This produced a record of the pH and Paco2 that existed at each level during the experiment.

Statistics. Hypercapnia-mediated changes in measurements of heart rate, arterial pressure, intravenous pressure, mesenteric vein diameter, and sympathetic nerve activity were determined by comparing control measurements (before hypercapnia) with corresponding measurements during hypercapnia. The changes were compared with each other by a multiple analysis of variance for repeated measures using the Super ANOVA statistical software (Abacus Concepts, Berkeley, CA). Data reported as percent changes were first transformed to arcsin √x before analysis to avoid a nonnormal distribution (17). All data are reported as means ± SE. Differences with a P value ≤ 0.05 are considered to be significant.

RESULTS

Blood gases. Table 1 illustrates the means ± SE blood gas values for each level of acute hypercapnia. The blood samples for arterial blood gas analyses were taken 40 s after initiation of CO2 inhalation. The pH and Paco2 values were significantly different among the three levels of hypercapnia and changed proportionally with the imposed levels of CO2 in the breathing mixture. There was no apparent difference in the observed end-tidal CO2 readings or measured blood gas values between intact and denervated animals. Accordingly,

<table>
<thead>
<tr>
<th>CO2 %</th>
<th>pH</th>
<th>Paco2, mmHg</th>
<th>Paco2, mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline (0%)</td>
<td>7.41 ± 0.01</td>
<td>32.2 ± 1.6</td>
<td>123.4 ± 3.2</td>
</tr>
<tr>
<td>15%</td>
<td>7.13 ± 0.01*</td>
<td>76.4 ± 2.3*</td>
<td>145.0 ± 4.2*</td>
</tr>
<tr>
<td>20%</td>
<td>7.06 ± 0.02**</td>
<td>96.5 ± 1.6**</td>
<td>151.0 ± 4.7**</td>
</tr>
<tr>
<td>25%</td>
<td>6.99 ± 0.02**</td>
<td>114.5 ± 1.5**</td>
<td>161.1 ± 3.2**</td>
</tr>
</tbody>
</table>

Table 1. Arterial blood gas analyses
the data for Table 1 were pooled from both animal types.

Mesenteric vein diameter. Figure 1 illustrates the transient mesenteric venoconstriction in response to acute CO₂ inhalation. The peak constriction occurred between 60 and 70 s after CO₂ inhalation was initiated, despite the fact that the increased concentration of inhaled CO₂ lasted only 40 s. Inhalation of 15% CO₂ tended to produce less mesenteric venoconstriction after denervation than before. However, there were no significant differences at any of the time intervals (Fig. 1). In contrast, mesenteric venoconstriction in response to both 20% and 25% inhaled CO₂ was significantly attenuated following denervation compared with the corresponding response before denervation. The absolute and percent mesenteric vein diameter changes in response to hypercapnia were as follows (means ± SE): 15% CO₂ resulted in a constriction of 38 ± 5 µm (6.5 ± 1.1%) in the intact preparation compared with 28 ± 5 µm (5.0 ± 0.9%) following denervation; 20% CO₂ resulted in a constriction of 73 ± 9 µm (12.0 ± 1.5%) before denervation and 38 ± 6 µm (6.9 ± 1.2%) following denervation; and 25% CO₂ resulted in a constriction of 115 ± 20 µm (18.0 ± 2.2%) before denervation and 47 ± 9 µm (8.4 ± 1.3%) after denervation. In the neurally intact preparation, the percent venoconstriction during 20% CO₂ was significantly greater than that during 15% CO₂. Likewise, the 25% CO₂ response was greater than the 20% response (Table 2). Thus there was a graded response such that the percent change in vein diameter was proportional to the level of hypercapnia. However, after denervation there was no significant difference in the percent change in vein diameter between the 15% and 20% levels of CO₂ or between the 20% and 25% levels. The percent contribution toward hypercapnia-mediated mesenteric venoconstriction by the peripheral chemoreceptors was determined by calculating the percent reduction in peak diameter change resulting from chemoreceptor ablation. These values were as follows: 23.0 ± 4.7% for 15% CO₂, 44.6 ± 5.5% for 20% CO₂, and 54.9 ± 3.7% for 25% CO₂. Again, this represented a graded response such that the percent contribution of the chemoreceptors was proportional to the level of imposed hypercapnia, with the contribution resulting from 20% inspired CO₂ being significantly greater than that for 15% and the

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**Fig. 1.** Time course changes in mesenteric vein diameter (MVD) expressed as percent change from control. Data are expressed as means ± SE; n = 12 rabbits. During 15% CO₂ inhalation (A), there was no difference in percent change between intact and denervated conditions. During 20% (B) and 25% CO₂ (C) inhalation, there was significant attenuation of mesenteric venoconstriction after denervation. Intact, condition before carotid chemoreceptor denervation; Denervated, condition after carotid chemoreceptor ablation. *P ≤ 0.05 vs. baseline (prehypercapnia condition, i.e., 0 s); †P ≤ 0.05 vs. before denervation.

<table>
<thead>
<tr>
<th>15% CO₂</th>
<th>20% CO₂</th>
<th>25% CO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MVD, µm</strong></td>
<td><strong>Baseline</strong></td>
<td><strong>Peak</strong></td>
</tr>
<tr>
<td>Intact</td>
<td>614 ± 28</td>
<td>576 ± 30*</td>
</tr>
<tr>
<td>Den</td>
<td>555 ± 41†</td>
<td>527 ± 40‡</td>
</tr>
<tr>
<td><strong>MAP, mmHg</strong></td>
<td><strong>Baseline</strong></td>
<td><strong>Peak</strong></td>
</tr>
<tr>
<td>Intact</td>
<td>84.7 ± 5.0</td>
<td>78.1 ± 4.4*</td>
</tr>
<tr>
<td>Den</td>
<td>99.7 ± 5.8†</td>
<td>92.4 ± 5.4†^</td>
</tr>
<tr>
<td><strong>HR, beats/min</strong></td>
<td><strong>Baseline</strong></td>
<td><strong>Peak</strong></td>
</tr>
<tr>
<td>Intact</td>
<td>270 ± 5</td>
<td>193 ± 9*</td>
</tr>
<tr>
<td>Den</td>
<td>290 ± 8†</td>
<td>237 ± 9†^</td>
</tr>
<tr>
<td><strong>MVP, mmHg</strong></td>
<td><strong>Baseline</strong></td>
<td><strong>Peak</strong></td>
</tr>
<tr>
<td>Intact</td>
<td>6.9 ± 0.4</td>
<td>6.6 ± 0.3</td>
</tr>
<tr>
<td>Den</td>
<td>7.9 ± 0.4†</td>
<td>7.5 ± 0.5†</td>
</tr>
</tbody>
</table>

Data are means ± SE; n = 12 rabbits. Intact, intact condition before carotid chemoreceptor denervation; Den, denervated condition; MVD, mesenteric vein diameter; baseline, resting prehypercapnia condition; peak, maximum constriction in response to hypercapnia; MAP, mean arterial blood pressure; HR, heart rate; MVP, mesenteric intravenous pressure. *P ≤ 0.05 vs. baseline; †P ≤ 0.05 vs. before denervation; ‡P ≤ 0.05 vs. 15% CO₂; §P ≤ 0.05 vs. 20% CO₂.
contribution for 25% CO₂ being significantly greater than that for 20%.

Sympathetic efferent nerve activity. Figure 2 illustrates changes in filtered, rectified SENA during hypercapnia, before and after chemoreceptor denervation. In the intact preparations, each level of CO₂ significantly increased peak efferent nerve activity compared with baseline. After denervation, the magnitude of baseline activity and peak activity during hypercapnia both increased significantly compared with the intact preparation. However, the increase in sympathetic activity during hypercapnia (i.e., peak vs. baseline) was significantly less in the denervated versus the intact preparation. Before denervation, the peak increases in the filtered, rectified sympathetic efferent nerve activity in response to hypercapnia were 33.5 ± 6.4%, 36.6 ± 8.3%, and 40.6 ± 12.9% during the 15%, 20%, and 25% CO₂ inhalation periods, respectively. After denervation, each of these values decreased significantly to 4.8 ± 1.6%, 8.4 ± 2.0%, and 13.1 ± 4.6%, respectively. After hexamethonium administration (at the end of the denervation studies), there was no recognizable pattern in the sympathetic nerve activity, and it was considered to represent the background signal in the recording system.

Hemodynamic changes. Table 2 illustrates mean arterial pressure, heart rate, and intravenous pressure responses to hypercapnia in intact and denervated preparations, which were measured concurrently with diameter changes. Significant decreases were observed in heart rate and blood pressure in response to each level of hypercapnia. In addition, mesenteric intravenous pressure decreased significantly in response to the 20% and 25% (but not the 15%) CO₂ levels. The data reported as “peak” values in Table 2 refer to the value corresponding to the maximum change (i.e., decrease) observed during the sequence of CO₂ inhalation. Thus the “peak” of any of the hemodynamic responses did not necessarily correlate where the “peak” venoconstriction was recorded. Rather, in most cases, maximum changes were usually observed after termination of the 40-s period of CO₂ inhalation. After denervation, the same pattern of hypercapnia-mediated decreases in mean arterial pressure, heart rate, and mesenteric intravenous pressure were observed as in the intact preparations. However, in the denervated preparations, the magnitude of both the baseline as well as the peak values for these measurements were increased compared with the intact condition.

DISCUSSION

The results of the present experiments indicate that 1) acute hypercapnia caused mesenteric venoconstriction, 2) this venoconstriction was significantly attenuated following carotid denervation, and 3) the percent contribution by carotid chemoreceptors toward this venoconstriction was proportional to the level of imposed hypercapnia.

Whereas many studies have examined the effects of hypercapnia on the cardiovascular system, few have focused specifically on the effects of increased PaCO₂ on mesenteric capacitance veins. The net response of these vessels to hypercapnia is a result of the interaction between a direct vasodilatory effect (4, 7), a reduction in vessel distending pressure, and an indirect (sympathetically mediated) vasoconstriction (6, 16). The data in the present study suggest that mesenteric venoconstriction predominates in response to acutely administered (i.e., short duration) systemic hypercapnia.

It is recognized that such mesenteric venoconstriction may be the result of the following: 1) active constriction of the venous smooth muscle via the sympathetic efferents and the chemoreceptors, 2) constriction of the veins by sympathetic activity induced by the carotid baroreceptor denervation and modified by noncarotid body chemoreceptors, and 3) a passive reduction in venous pressure (9). The two factors that influence mesenteric vein diameter by passive effects are the arterial inflow of blood into the mesenteric bed and the venous outflow from the bed. Both of these factors are dependent on intraluminal pressure and tone of vessels leading to and from the bed of interest. There is evidence in the present study for a passive reduction in mesenteric vein diameter because there is a small but significant decrease in mesenteric intravenous pressure in response to each level of hypercapnia before and after carotid denervation. This decrease occurred concurrently with a similar decrease in mean arterial pressure. Hemodynamic effects of hypercapnia have been shown to produce negative myocardial chronotropic and inotropic effects (11, 15, 20). Possible causes of such hypercapnia-induced heart rate changes could include direct effects on the myocardium, baroreflex-mediated responses (20), or bradycardia resulting from increases in intracranial pressure during hypercapnia-mediated cerebral vasodilation. Thus, despite reflex sympathetic activation that appears to result from hypercapnia, the severe bradycardia that occurs during
the acute stimulus may decrease cardiac output and produce a temporary net decrease in mean arterial blood pressure.

However, there is also evidence suggesting that such vasoconstriction was a reflex sympathetically mediated response to chemoreceptor activation by hypercapnia. At each level of CO₂, mesenteric vasoconstriction was accompanied by simultaneous increase in postganglionic sympathetic efferent nerve activity. Furthermore, denervation resulted in simultaneous attenuation of both the reflex increase in sympathetic efferent nerve activity and the vasoconstriction response. Whereas the present study does not provide evidence as to the relative contribution of active versus passive forces affecting the mesenteric vein diameter, similar vasoconstriction in response to acute intervals of systemic hypercapnia has been abolished by the simultaneous administration of tetrodotoxin (a known neuronal inhibitor) (8) locally in the PSS superfusate to the vessel under study (18). Because the tetrodotoxin was administered locally to the vessel, all other systemic hemodynamic responses to the hypercapnia were unchanged. Thus the fact that local sympathetic neural blockade can virtually eliminate the hypercapnia-mediated vasoconstriction without affecting systemic hemodynamics strongly suggests that this response is primarily the result of sympathetic activation. Furthermore, in the present study, inspection of the data in Table 2 and Fig. 1 indicate that, before denervation, intravenous pressure decreased by −4%, 6%, and 13% in response to 15%, 20%, and 25% inspired CO₂, respectively, compared with corresponding mesenteric vein diameter decreases of 5%, 10%, and 16% under the same conditions. In contrast, following denervation (during the same sequence of hypercapnia), intravenous pressure decreased by −5%, 9%, and 13%, respectively, compared with venous diameter decreases of 4%, 6%, and 7.5%. Thus denervation had relatively little effect on intravenous pressure, whereas vasoconstriction decreased to the point that the percent change in pressure was actually more than that for diameter. This suggests less active and greater relative passive influences on the vessels studied following denervation.

In the present study, the conclusions about the relative contribution of the peripheral chemoreceptors in mediating reflex mesenteric vasoconstriction in response to hypercapnia are based on the attenuation of the diameter response following chemoreceptor and baroreceptor denervation. Several species, including rabbits, are devoid of aortic bodies (1). Thus cauterization of the carotid chemoreceptor in the current preparation should have abolished all peripheral neural sensory input resulting from hypoxia and hypercapnia. Ablation of the tissues in the carotid bifurcation should have effectively eliminated the carotid sinus as well as the carotid body (because of their close proximity), and it is unlikely that one would be eliminated but not the other. Therefore (as indicated), elimination of the baroreceptor response to bilateral carotid occlusion following the denervation procedure was considered to represent effective ablation of both the carotid sinus and the carotid body sensory receptors. After such ablation, the remaining hypercapnia-mediated mesenteric vasoconstriction in the denervated versus the innervated condition should represent the relative graded contribution of all noncarotid body chemoreceptor input that regulates mesenteric VSM tone.

One major consideration in interpretation of the sympathetic efferent nerve activity data is that baseline (i.e., prehypercapnia) levels of this activity were greater in the denervated compared with the intact preparations. In addition, baseline heart rates, mean arterial pressures, and mesenteric intravenous pressures were also all increased in denervated compared with innervated animals. These responses are likely the result of baroreceptor deafferentation, which is known to produce an enhanced resting sympathetic tone. It could be argued that such an increase (resulting from baroreceptor unloading) might alter interpretation of the results by masking a transient sympathetically mediated response to hypercapnia. However, in other studies using artificially imposed hypotension to produce baroreflex unloading, such maneuvers did not result in a supramaximal sympathetic response, and other stimuli (under these conditions) have been shown to produce significantly greater responses than those resulting from baroreceptor unloading alone (12). Clearly, prehypercapnia mesenteric vein diameters were all constricted in denervated preparations relative to corresponding intact controls (Table 2). Furthermore, in contrast to the intact condition, the vein diameter response to 25% CO₂ was unchanged from that of the 20% CO₂ response in the denervated preparations. This is certainly indicative of an “elevated level of sympathetic tone.” This might also suggest that less additional sympathetic tone was available to be recruited in denervated preparations and that hypercapnia-mediated vasoconstriction was attenuated only because the vessels were “preconstricted” to start with. However, this does not appear to be the only explanation, at least in the case of the (most severe) 25% level of CO₂. During this response the minimum diameter was actually greater in the denervated (versus the intact) preparation despite starting from a lower baseline. A higher resting sympathetic tone would not be expected to alter the maximum (sympathetically mediated) constriction attainable. Thus this provides support for a mechanism that also includes elimination of (as well as masking of) the peripheral chemoreceptor response to hypercapnia. Moreover, even if the sympathetically mediated response to hypercapnia were merely masked (by baroreceptor unloading) rather than actually eliminated (by electrocautery ablation), the differences in measured responses would still be expected to represent the contributions that the peripheral chemoreflex stimulation would have made.

In summary, acute graded hypercapnia produced corresponding increases in sympathetic efferent nerve activity and mesenteric vasoconstriction and decreases in heart rate and mean arterial blood pressure. Other studies have reported that increased CO₂ produces reflex increases in sympathetic activity, direct relax-
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Regulated by chemoreceptor activation during in-deafferentation procedure likely represents the contribution of the diameter change during hypercapnia following the denervation of the peripheral chemoreceptors in the current study produced a significant attenuation of this hypercapnia-mediated venoconstriction. Such attenuation may have resulted from direct ablation of chemoreceptors (thus eliminating all reflex response to hypercapnia) or a masking of chemoreceptor response due to baroreceptor deafferentation, or a combination of the two. Nevertheless, the reduction of the diameter change during hypercapnia following the deafferentation procedure likely represents the contribution toward control of the capacitance veins that is regulated by chemoreceptor activation during increased CO2.

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