Reflex vascular responses in the anesthetized dog to large rapid changes in carotid sinus pressure

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DOE, C. P. A., D. A. SELF, M. J. DRINKHILL, N. McMAHON, D. S. MYERS, AND R. HAINSWORTH. Reflex vascular responses in the anesthetized dog to large rapid changes in carotid sinus pressure. Am. J. Physiol. 275 (Heart Circ. Physiol. 44): H1169–H1177, 1998—This study examined reflex vascular responses to large rapid increases and decreases in carotid sinus pressure to determine whether delayed or inappropriate vascular responses might be obtained if, they occurred in people, could lead to hypotension during exposure to rapidly alternating gravitational forces. In chloralose-anesthetized open-chest dogs, a perfusion circuit controlled carotid sinus and thoracic aortic pressures and blood flows to both the vasculally isolated abdominal circulation and a hindlimb (perfusion pressure changes denoted resistance). When carotid pressure was increased and decreased over the range of 60–180 mmHg, the resulting reflex vasodilatation occurred significantly more rapidly than the vasoconstriction (P < 0.001). In the abdominal vascular bed, time constants for vasodilatation and vasoconstriction were 4.2 ± 0.5 and 7.5 ± 1.0 s, respectively. Decreases in carotid pressure in pulses of 10-s duration or less failed to elicit maximal vasoconstriction, whereas increases in carotid pressure lasting as little as 5 s did elicit maximal vasodilatation. “Square-wave” alternations in carotid pressure with periods of 10 s or less (5 s high, 5 s low) resulted in attenuation of the vasoconstriction, and at a 4-s period, both vascular beds remained almost maximally vasodilated throughout. The failure of vascular resistance to follow carotid pressure changes was not due to a failure of the response of sympathetic efferent activity, since the time constants for the reduction and increase in discharge were much shorter at 0.56 ± 0.13 and 0.43 ± 0.10 s, respectively. These results indicate that rapid changes in carotid pressure could result in inappropriate vasodilatation and hypotension and might, in some circumstances, such as in pilots flying high-performance aircraft, predispose to syncope.

baroreceptors; vasodilatation; gravitational effect; syncope

ALTHOUGH BARORECEPTORS are known to exist at various sites of the body, including the aortic arch (18) and, from recent studies, also the coronary arteries (1, 11), it is those in the carotid sinuses that have received most attention (21). Carotid baroreceptors are strategically placed to protect cerebral perfusion pressure, and because of their position in relation to the heart, they are subjected to the greatest changes in pressure during postural alterations and particularly during high levels of acceleration stress (28).

Both the afferent discharge from carotid baroreceptors and the resulting reflex responses are influenced by the amplitude and frequency of the pulse pressure distending them. In part, this is a consequence of nonlinearities of the static pressure-response relationship, particularly when pressure falls below threshold or increases to above saturation level. However, provided pressure excursions remain within the operating range of the receptors, most investigators have found that the overall discharge from the receptors per unit time is little affected by the pulsatility (2, 13, 23), although the reflex response can be greatly enhanced. Ead et al. (12) showed that simply reducing the pulse amplitude in the carotid sinus by use of small damping chambers caused large increases in arterial pressure, indicating that the inhibitory effect of the reflex was reduced. Several subsequent studies have examined the responses to carotid baroreceptor stimulation using mathematical modeling approaches applied to the baroreceptors themselves (6, 7, 13, 29), to the efferent sympathetic discharge (19, 20, 22), or to the resulting responses of vascular resistance and blood pressure (10, 19, 22, 24, 27). Essentially, these studies have shown that the baroreceptors themselves can faithfully respond to very rapid changes in distending pressures, and the response of efferent sympathetic discharge also follows the changes in frequency of pulsatile baroreceptor pressures up to 0.8 Hz. However, the response of arterial blood pressure becomes attenuated as the frequency of stimulation changes from 0.01 to 0.1 Hz.

Although the previous studies have established that the amplitude of oscillation of the reflex vascular responses becomes attenuated at high frequencies of oscillation of baroreceptor stimulus, generally only small sinusoidal pressure perturbations were applied. Furthermore, most previous studies have not compared the relative time courses of the vasoconstrictor and vasodilator responses resulting from respective decreases and increases in baroreceptor stimulation. None has compared the time courses of the vasoconstrictor and vasodilator responses to square-wave supramaximal alterations in baroreceptor pressure. This could be of importance, since situations may exist where there may be very large and unpredictable changes in baroreceptor stimulation, possibly with a periodicity of <10 s. One such situation occurs when flying a high-performance aircraft. This is the so-called “push-pull” effect (4, 26), where sudden large alternations in gravitational thrust in the vertical plane (−Gz and +Gz) may predispose to syncope. The purpose of this investigation, therefore, was to determine whether large changes
in carotid sinus pressure at various periodicities could result in apparent inappropriate or inadequate reflex vascular responses.

METHODS

Experiments were undertaken using dogs (11–20 kg) of either sex. Animals were premedicated with a subcutaneous injection of morphine sulfate (0.5 mg/kg) and anesthetized with 13 mg/kg pentobarbital sodium (intravenously) followed by 80 mg/kg α-chloralose (intravenously). A stable anesthetic level was maintained by constant infusion throughout the experiment of α-chloralose (0.5 mg·kg⁻¹·min⁻¹).

The neck was opened at the midline, the trachea was cannulated, and the lungs were ventilated by a Starling “ideal” pump using 40% oxygen-enriched air at a rate of 18 strokes/min with a stroke volume initially of 17 ml/kg. When the pleura was opened, an end-expiratory resistance, equivalent to 3 cmH₂O, was used to prevent collapse of the lungs. Arterial Pₐ, P₃, and pH were determined frequently and adjusted to normal limits (>100 mmHg, 35–45 mmHg, and 7.30–7.40, respectively) by addition of oxygen to the inspired gas, adjustments to the respiration pump, and administration of molar sodium bicarbonate solution as necessary.

Both carotid sinus regions were vascularly isolated, and the left hindlimb was isolated and prepared for perfusion as previously described (16). Briefly, the femoral nerve and vessels and the sciatic nerve were dissected free, and nylon cords, for subsequent tightening, were placed around the main muscle groups avoiding these nerves and vessels.

The chest was widely opened in the fifth left intercostal space using an incision that crossed the midline by dividing the sternum and dividing the fifth and sixth left ribs near their vertebral attachments. The descending aorta above the diaphragm was mobilized by dividing four pairs of intercostal arteries between ligatures, and the pericardium was opened to allow access to the left atrial appendage.

The animal was given heparin sodium (500 IU/kg) and was connected to the perfusion circuit (Fig. 1), which had a capacity of ~2 liters and was filled with a solution of equal parts of mammalian Ringer solution and dextran (Sigma, St. Louis, MO) in saline. A cannula in the central end of the descending aorta conveyed blood to a large arterial reservoir that was maintained at a constant pressure, and from which it was pumped to the various parts of the circulation as follows: at constant flow (603U pump, Watson-Marlow, Falmouth, UK) into the distal end of the aorta, at constant flow (505U pump, Watson-Marlow) into the femoral artery of the isolated limb, and into a constant-pressure reservoir at a flow (505 U pump) automatically controlled to maintain a constant level of blood from which the carotid sinuses were perfused.

Fig. 1. Diagram of perfusion circuit. Cannula in descending aorta conveys blood to main arterial reservoir from which it is pumped 1) into pressurized carotid reservoir at a rate controlled automatically to maintain a constant blood level in that reservoir, 2) at constant flow into aorta immediately above diaphragm, and 3) at constant flow into vascularly isolated hindlimb. Blood from carotid sinuses (through lingual arteries), femoral vein, and inferior vena cava drains into a venous reservoir and is pumped back into external jugular veins. Some of left atrial blood drains into an open reservoir from which it is pumped into main arterial reservoir. P, pump; SG, strain gauge transducer; S, snare for limb isolation.
Blood from the inferior vena cava, the femoral vein draining the perfused limb, and the carotid sinuses via the lingual arteries passed into an open venous reservoir from which it was pumped (603U pump) back into the dog through the external jugular veins. The speed of this pump was controlled automatically to maintain a constant level in the venous reservoir.

A wide-bore (8-mm) cannula, inserted into the left atrium through its appendage and connected to an open reservoir, allowed control of left atrial pressure. The level of blood in this reservoir was held constant by pumping (603U pump) from it to the main arterial reservoir.

The hindlimb was vascularly isolated by tightening the three nylon cords previously placed round the muscle groups, using ratchet devices. The effectiveness of the isolation of both the limb and abdominal circulations was confirmed by stopping the relevant perfusion pump and observing that the arterial perfusion pressure fell to <30 mmHg. The animals' temperature was maintained at 37–39°C with the use of heating elements under the table and heat exchangers in the circuit.

Blood pressures were recorded using saline-filled nylon catheters attached to strain gauges (Gould-Statham P23 Id) from the carotid perfusion cannula (carotid sinus pressure), the descending thoracic aorta, the perfused abdominal aorta (via the right femoral artery), the perfused left femoral artery, and inferior vena cava (inserted through a femoral vein). Signals were amplified (EMMA system, SE Laboratories, Feltham, UK) and recorded on an electrostatic recorder (Gould ES1000) and on VHS tape (Racal V-store; Racal Recorders, Southampton, UK). The taped signals were subsequently digitized at a rate of 2,000 samples/s for subsequent computer analysis (Fastdaq, Lectromed, Letchworth, UK).

Nerve recording experiments. In some experiments, small strands were dissected from either renal or lumbar sympathetic efferent nerves. The strands were placed under warm mineral oil and laid across bipolar silver electrodes. Efferent activity was amplified and filtered (Neurolog System, Digitimer, Welwyn Garden City, UK). A window discriminator was used to count the discharge frequency.

Experimental procedure. After the perfusion circuit was connected, flows were adjusted to set pressures to approximately the same values as before cannulation. The flows to the abdominal region and the limb then remained unchanged throughout the remainder of the experiment. The animal was then allowed to stabilize for 30 min before starting experimental interventions.

Initially, carotid pressure was changed in 30-mmHg increments between 60 and 180 or 210 mmHg to determine the threshold and saturation pressures (operating range) for the reflex responses of perfusion pressures in both the limb and the abdominal circulation. Carotid pressure was then changed over this operating range with single pressure steps, either increases or decreases, until steady-state vascular responses were obtained. Carotid pressure was then changed over the same range in single pulses in both directions and with durations of 30, 20, 10, 5, and 2 s. We also determined the responses to alternating carotid pressures in a square-wave fashion, with equal intervals at the high and low pressures, and with total cycle lengths of 60, 40, 20, 10, and 4 s.

These experiments were carried out in accordance with current United Kingdom legislation [Animals (Scientific Procedures) Act 1986] and in conformity with the National Institutes of Health Guide for the Care and Use of Laboratory Animals [Department of Health and Human Services Publication No. (NIH) 85–23, Revised 1985].

Fig. 2. Traces of abdominal aortic perfusion pressure (Abd AoP), carotid sinus pressure (CSP), isolated hind-limb perfusion pressure (LPP), and inferior vena caval pressure (IVCP) during a large step increase and decrease in CSP. Note rapid decrease in LPP and Abd AoP to increasing CSP and slower increase in these pressures after a decrease in CSP.
RESULTS

Results reported are calculated from the averages of values calculated for each animal and are given as means ± SE. Significance levels unless otherwise indicated were assessed using the Student’s t-test for paired data.

Steady-state responses to changes in carotid sinus pressure. In nine dogs, carotid pressure was increased in single steps from 71 ± 9.7 to 177 ± 6.5 mmHg with thoracic aortic pressure held constant at 96 ± 7.0 mmHg. This resulted in decreases in abdominal perfusion pressure from 170 ± 10.9 to 102 ± 12.8 mmHg (−40 ± 5.5%, P < 0.001) and limb perfusion pressure from 159 ± 11.4 to 99 ± 14.1 mmHg (−38 ± 5.3%, P < 0.001). Perfusion pressures returned to levels not significantly different from initial control levels when carotid pressure was decreased again.

The decreases in perfusion pressure in both vascular beds to increases in carotid pressure occurred more rapidly than the corresponding increases in perfusion pressure to decreases in carotid pressure (Fig. 2). To quantify the time courses of the vascular resistance responses in the two beds to increases and decreases in carotid pressure, monoexponential curves (Graph Pad) were fitted to the changes in perfusion pressure, and the time constants were calculated. Figure 3 shows a curve fit for changes in abdominal perfusion pressure to increases and decreases in carotid pressure in one dog. The data derived from all animals are listed in Table 1. The time constants, for both vascular beds, for the vasoconstrictor responses (7.5 ± 1.0 and 11.4 ± 2.2 s for abdomen and limb responses, respectively) were approximately double those for the vasodilator responses (4.2 ± 0.5 and 5.5 ± 0.6 s).

Vascular responses to single pulse changes in carotid pressure of various durations. In five dogs, carotid sinus pressure was changed from a low (54 ± 3.2 mmHg) to a high value (192 ± 12.3 mmHg) for durations of 30, 20, 10, 5, and 2 s before returning to the low value. Thoracic aortic pressure was held constant at 98 ± 6.2 mmHg. The reverse procedure was also carried out in each dog whereby carotid pressure was decreased from the high to the low pressure for the same durations. Figure 4 compares the responses in one dog to these step changes in carotid pressure. In all dogs, there were larger changes in both perfusion pressures to increases in carotid pressure of 2-, 5-, and 10-s duration compared with the responses to the corresponding decreases in pressure.

Table 1. Time constants for vasodilation and vasoconstriction determined from changes in abdominal aortic and limb perfusion pressures

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Aortic Perfusion Pressure, mmHg</th>
<th>Vasodilation</th>
<th>Vasoconstriction</th>
<th>Δ</th>
<th>Limb Perfusion Pressure, mmHg</th>
<th>Vasodilation</th>
<th>Vasoconstriction</th>
<th>Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5.8 (0.98)</td>
<td>9.3 (0.88)</td>
<td>3.5</td>
<td></td>
<td>2.6 (0.87)</td>
<td>7.9 (0.87)</td>
<td>5.3</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>3.8 (0.90)</td>
<td>7.1 (0.91)</td>
<td>3.3</td>
<td></td>
<td>5.9 (0.92)</td>
<td>15.9 (0.95)</td>
<td>9.5</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2.8 (0.88)</td>
<td>5.3 (0.89)</td>
<td>2.5</td>
<td></td>
<td>9.5 (0.85)</td>
<td>14.3 (0.91)</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>5.7 (0.92)</td>
<td>7.1 (0.88)</td>
<td>1.4</td>
<td></td>
<td>4.8 (0.91)</td>
<td>10.7 (0.85)</td>
<td>5.9</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>4.2 (0.88)</td>
<td>7.7 (0.91)</td>
<td>3.5</td>
<td></td>
<td>5.00 (0.83)</td>
<td>5.9 (0.83)</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>2.6 (0.91)</td>
<td>3.5 (0.95)</td>
<td>0.9</td>
<td></td>
<td>5.9 (0.81)</td>
<td>8.7 (0.89)</td>
<td>2.8</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>3.4 (0.86)</td>
<td>5.7 (0.81)</td>
<td>2.3</td>
<td></td>
<td>3.6 (0.96)</td>
<td>4.5 (0.83)</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>6.7 (0.88)</td>
<td>14.2 (0.87)</td>
<td>7.5</td>
<td></td>
<td>6.6 (0.88)</td>
<td>26.7 (0.91)</td>
<td>20.1</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>2.9 (0.91)</td>
<td>7.3 (0.84)</td>
<td>4.4</td>
<td></td>
<td>5.1 (0.91)</td>
<td>8.5 (0.88)</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>Mean ± SE</td>
<td>4.2 ± 0.50</td>
<td>7.5 ± 1.00</td>
<td>3.3 ± 0.64</td>
<td></td>
<td>5.5 ± 0.65</td>
<td>11.4 ± 2.25</td>
<td>6.0 ± 1.98</td>
<td></td>
</tr>
</tbody>
</table>

Data in parentheses indicate coefficients of determination ($r^2$) of fit of data to a monoexponential curve. Δ, Change.
The results from all experiments are summarized in Fig. 5. This shows that for both vascular beds the reflex decreases in perfusion pressures (vasodilatation) were near maximal for stimulus durations of 10 s or more and were only slightly reduced at 5 s. The reflex increases in perfusion pressures (vasoconstriction) to decreases in carotid pressure, however, were significantly reduced when the pulse duration was 10 s or less. The changes in perfusion pressures (vasodilatation) at these durations were significantly larger than the corresponding increases in pressures.

Vascular responses to alternating carotid pressures at various periods. In seven dogs in which thoracic aortic pressure was held at 95 ± 7.1 mmHg, carotid pressure was changed between 75 ± 4.6 and 176 ± 7.9 mmHg, with equal intervals at the high and low pressures and periods of 60 s (30 s each at high and low), 40, 20, 10, and 4 s. Figure 6 illustrates the effects of these different periods. At the period of 20 s (second block), the peak perfusion pressures to both regions were less than those at the 40-s cycle length. These were reduced further at the 10- and 4-s periods. It can be seen that while the maximum perfusion pressures were reduced by shortening the cycle length, the minimum values were little changed, and this consequently resulted in a overall decrease in pressure. The results for all seven animals are summarized in Fig. 7, and this emphasizes the decline in the peak pressure at the low carotid pressure. The net effect of this is that at the shorter cycle lengths, perfusion pressures were almost constant and remained near their minimum values.

Responses of efferent sympathetic nervous discharge. Six sympathetic efferent units were recorded from five dogs. In three dogs, recordings of renal nerve activity were made (4 units), and in the other two, activity from lumbar nerves was recorded. Carotid sinus pressure oscillations with a period of 4 s, over the predetermined operating range (59 ± 1.7 to 176 ± 0.4 mmHg), always resulted in reciprocal changes in nerve discharge. The efferent discharge before an increase in carotid sinus pressure was 3.46 ± 0.82 impulses/s, and this decreased to 0.08 ± 0.03 impulses/s at the high carotid pressure. The time constants for the responses of efferent sympathetic nerve discharge were 0.59 ± 0.13 s.

Fig. 4. Responses in 1 dog to step decreases (top) and to step increases (bottom) of various durations in CSP. CSP was changed over entire baroreceptor sensitivity range in a square-wave fashion and maintained at each level for intervals of 2–20 s. Note that decreases in Abd AoP and LPP to increases in CSP were nearly maximal for pulse widths of only 5 s, whereas responses to decreases in CSP required longer periods of stimulation.
to increases and 0.43 ± 0.10 s to decreases in carotid sinus pressure, and these were not significantly different from each another (P > 0.5). Figure 8 shows recordings of lumbar sympathetic discharge from one dog showing the rapid response of the efferent neural activity to the changes in carotid pressure.

DISCUSSION

The main purpose of this study was to examine the time courses of the reflex vascular responses to large and rapid increases and decreases in carotid sinus pressure. The main impetus to this work was the observation that, in pilots of high-performance aircraft, sudden vertical plane acceleration (+Gz) after deceleration (−Gz) was often associated with hypotension and syncope. This is the so-called push-pull effect (4, 26).

To allow us to study responses to discrete stimulation of carotid baroreceptors, we needed to devise a preparation that would confine the stimulus to the carotid sinuses and would prevent secondary modification of the responses due to changes in the inputs from other reflexogenic areas. One technique frequently used to prevent changes in input from uncontrolled reflexogenic areas is to denervate the regions. However, we preferred not to do this because it would have resulted in abnormal levels of vasomotor activity. Instead, we aimed to hold the stimuli to the other regions at constant physiological levels. Changes in inputs from aortic and coronary artery receptors were minimized by controlling thoracic aortic pressure. A left atrial reservoir with partial left heart bypass minimized changes in left heart filling and controlled the stimuli to atrial and ventricular receptors. Although using this technique we cannot be certain that there were no changes at all in the stimuli to intrathoracic receptors, any changes that might have occurred would have been very small in relation to the maximal stimuli applied to the carotid receptors. Furthermore, it is known that significant vascular responses occur only when large pressure changes are applied to these regions (14, 15).

We assessed the responses of vascular resistance in two independently perfused vascular beds by determining changes in perfusion pressures during constant-flow perfusions. Vascular isolations of both regions were confirmed by observing that pressures fell rapidly to below 30 mmHg when the pumps were stopped. We used constant-flow perfusions rather than constant pressures, even though this imposed pump pulsations on the pressure, because the constant flow should allow neurally mediated responses to be studied without metabolic autoregulatory effects, which might have resulted if flow were allowed to change.

There are three main findings from this investigation. First, we have shown that when carotid sinus pressure is changed rapidly over the entire baroreceptor sensitivity range, the vasoconstriction occurring in response to the decrease in carotid pressure develops more slowly than the dilatation resulting from carotid hypertension. The time constant for the vasodilatation was only about one-half of that for the vasoconstriction. The second observation was that when we compared the responses to brief pulses of either large increases or decreases in carotid sinus pressure, the magnitude of the decrease in vascular resistance when carotid pressure briefly increased was greater than the vasoconstriction to brief decreases in carotid pressure. These differences were significant for pulse durations of 10 s or less. Finally, we showed that when alternating square-wave pulses were applied to the carotid baroreceptors and...
the period of alternation decreased below 10 s, not only were the responses of perfusion pressures unable to follow these pulses, but the vascular beds remained vasodilated. This was shown to be a failure of the vessels to constrict because efferent sympathetic nerve activity was able to respond completely to pressure alternations with periods as short as 4 s.

Some of these results are extensions of the findings of others. It was already known that the baroreceptor reflex has a rapid neural response but a much slower vascular response (19, 22, 29). We have confirmed this and have made important further observations relating to the differences in the rates of development of the reflex vasodilatation and vasoconstriction and to the maintained vasodilatation when carotid pressure alternates rapidly.

The only work comparable to ours was by Levison et al. (24), who also noted that, in response to square-wave stimuli to carotid baroreceptors, the rate of vasoconstriction was approximately one-half that of the vasodilatation. However, in that study, pressure pulses of only 2–20 mmHg were applied rather than changes over the entire baroreceptor range as were applied in our study. A major advantage to the use of supramaximal stimuli is that it should provide sufficient stretch to the baroreceptors to ensure that they remain maximally excited for the duration of the stimulus. Smaller carotid pressure changes result initially in large responses that then tend to adapt toward smaller steady-state levels. We observed this effect in preliminary experiments, and it has also been reported by Chapleau and co-workers (7, 8), who regarded it as a form of acute resetting. With the use of pressures in excess of saturation levels, any adaptation or resetting of the receptors would be unlikely to influence the discharge rate of the baroreceptors at either supramaximal or subthreshold stimuli.

The possible roles of properties of the baroreceptors themselves or central nervous pathways in causing the differential rate sensitivity were examined by recording activity in sympathetic efferent nerves. This activity followed the carotid pressure changes faithfully and rapidly with time constants for increases and decreases not significantly different from each other and much shorter than those for the vascular responses. This demonstrates that neither the baroreceptors themselves nor the central pathways could have been responsible for the reduced vascular response. Because the baroreceptors and the neural pathways involved cannot explain the differences between the rates of vasoconstriction and vasodilatation, the explanation must re-

Fig. 6. Responses in 1 dog to stepwise alterations in CSP with periods of between 40 and 4 s with perfusion pressures remaining near dilated levels. All pressures are in mmHg.
side somewhere within the effector mechanism. Possible explanations are that it could be because of differences in the rates of release and removal of synaptic norepinephrine, or because of the mechanical properties of the vascular smooth muscle. Norepinephrine is released in response to stimulation from adrenergic vesicles and diffuses to the adjacent vascular smooth muscle. Its removal is mainly by neuronal reuptake but also by metabolism and diffusion (5). The concentration at any time is dependent on the balance between release and removal, and a more rapid removal than release could explain our results.

Fig. 7. Values of Abd AoP and LPP when CSP was low (○, 75 ± 12.2 mmHg) and high (●, 176 ± 7.9 mmHg) and period of alternation varied between 60 and 4 s. Note attenuation of vasoconstrictor responses when periods were 10 and 4 s. Results are means ± SE from 7 dogs. *P < 0.05, **P < 0.01 compared with corresponding values at 60-s period.

An alternative explanation may be related to the architecture of the structures within the blood vessel walls. Models of vascular smooth muscle have the contractile element coupled to a series elastic component (SEC), and this may extend by up to 20% during maximal contraction (25). The compliance of the SEC decreases as it is stretched (9, 17, 25), and this could explain the differential rates of vasoconstriction and vasodilatation because, as the contractile element shortens from the relaxed state, this initially would extend the compliant SEC rather than reduce the lumen. However, in the active state, the tension in the SEC would be high and its compliance low so that lengthening of the contractile element would lead to an immediate dilatation of the vessel.

Whatever the explanation for the different rate sensitivities for the reflex vasodilatation and vasoconstriction, these experiments are likely to be relevant to situations in which there are large and rapid changes in baroreceptor stimulation. If responses occur in people in a similar way to those seen in these dogs, a transient perturbation of baroreceptor stimulus lasting perhaps only 2 s may lead to a relatively prolonged vasodilatation. A relevant study was carried out by Banks et al. (3), who subjected human volunteers to decreases and increases in gravitational forces in the vertical plane (+Gz and −Gz) and found that applications of a −Gz force of −2 Gz for only 2 s caused blood pressure to fall and to impair the vasoconstrictor response to subsequent application of an acceleration force at +2.25 Gz. The results of the present study may offer an explanation for the inappropriate vasodilatation when blood pressure changes rapidly and point to possible hazardous situations that may arise when baroreceptor stimulation is abruptly changed.

In addition to the effects that might occur when flying high-performance aircraft, it is not inconceivable also that even some terrestrial activities could cause similar rapid changes in baroreceptor input. One example might be by bending down low and suddenly standing straight up when the necessary vasoconstriction might be delayed and dizziness and even transient loss of consciousness might occur.

Fig. 8. Responses of lumbar efferent sympathetic nerve discharge in 1 dog to changes in CSP with a period of 4 s. Efferent activity changes with each carotid pulse with no discernible lag. LSNA, lumbar sympathetic nerve activity.
REFERENCES