Carotid Sinus Baroreceptors Modulate Tracheal Smooth Muscle Tension in Dogs

H.D. Schultz, T.E. Pisarri, H.M. Coleridge, and J.C.G. Coleridge

Arterial baroreceptors are known to influence airway smooth muscle tone. Thus, increasing carotid sinus pressure from 20 to 200 mm Hg causes reflex tracheal dilation. However, the effects of changing carotid sinus pressure around a normal arterial pressure set-point of 100 mm Hg have not been examined. In anesthetized, artificially ventilated dogs, we distended the vascularly isolated carotid sinuses with a pulsatile pressure and recorded isometric tension in an upper tracheal segment. The aortic nerves were cut. Increasing mean carotid sinus pressure in steps between 100 and 200 mm Hg decreased tracheal tension, heart rate, and arterial blood pressure; decreasing sinus pressure between 100 and 25 mm Hg had the opposite effect. Changing carotid sinus pressure still evoked tracheal responses when systemic arterial pressure was held constant. Increasing and decreasing carotid sinus pulse pressure around a constant mean pressure evoked similar changes in tracheal tension. All reflex effects were abolished by cutting or cooling (0°C) the carotid sinus nerves; tracheal responses were abolished by cutting the laryngeal nerves or administering atropine. When carotid sinus pressure was held at 100 mm Hg, cooling the sinus nerves increased tracheal tension. Changes in tracheal tension evoked by the carotid baroreflex were of comparable magnitude to those triggered by stimulating pulmonary stretch receptors, laryngeal receptors, and pulmonary C-fibers. Our results indicate that carotid sinus baroreceptors exert a tonic influence on the upper airways by a vagal cholinergic pathway, increasing and decreasing tracheal smooth muscle tension as blood pressure varies around the normal set-point. (Circulation Research 1987;60:337–345)

The reflex effects of stimulating carotid sinus baroreceptors are not confined to the cardiovascular system but also involve breathing and bronchomotor tone. In general, increasing carotid sinus pressure inhibits breathing and decreasing sinus pressure stimulates it (for references see Heymans and Neil and Brunner et al). There is less agreement about the effects of baroreceptors on bronchomotor tone. Some investigators found that bronchomotor responses were difficult to elicit from the carotid sinus; others reported that baroreceptor stimulation produced bronchoconstriction and concluded that baroreceptors take part in the reflex maintenance of bronchomotor tone in normal conditions (for references see Widdicombe). However, several of these studies did not allow changes in airflow resistance to be distinguished from changes in lung compliance or lung volume. Nadel and Widdicombe therefore reexamined the effects of carotid baroreceptors on bronchomotor tone, varying pressure in the perfused carotid sinuses in dogs and measuring changes in tracheal volume and total lung resistance. Increasing sinus pressure in a single step from 20 to 200 mm Hg caused a slight tracheal dilation, while reducing pressure had the opposite effect; total lung resistance was not changed significantly. The functional significance of these results is difficult to assess, however, because the changes in baroreceptor firing resulting from such large alterations in sinus pressure are very different from those occurring during normal baroreceptor operation when blood pressure varies around a set-point of about 100 mm Hg.

We undertook the present study to examine the reflex influence of carotid sinus baroreceptors on the smooth muscle tone of an innervated segment of the upper trachea, varying carotid sinus perfusion pressure above and below a baseline (set-point) pressure of 100 mm Hg.

Materials and Methods

General

Eleven dogs (14–25 kg) were given promazine HCl (Sparine, Wyeth, 50 mg i.m.); 30 minutes later they were anesthetized with α-chloralose (80 mg/kg i.v.). Supplemental doses of chloralose (10 mg/kg i.v.) were given hourly to maintain anesthesia. The trachea was cannulated low in the neck, and the lungs were ventilated with 50% oxygen in air by a Harvard Model 613 respirator whose expiratory outlet was placed under 2–3 cm of water. Tidal CO2 was monitored by a Beckman LB-1 gas analyzer, and end-expiratory Pco2 was kept at about 35 mm Hg by adjusting the ventilatory rate. Periodically, arterial Po2, Pco2, pH, and base excess were measured with a Corning 175 blood gas/pH analyzer. Sodium bicarbonate was infused i.v., when necessary, to correct metabolic acidosis.

Femoral arterial blood pressure was measured by a Statham P23Gb strain gauge, and heart rate was measured by a cardiotachometer triggered by an electro-
cardiogram (lead II). The signals representing tidal CO₂, femoral arterial blood pressure, heart rate, and other variables described below (transverse tension in the tracheal segment, blood pressure in the vascularity isolated carotid sinuses, and temperature of the carotid sinus nerves) were recorded by a Grass polygraph.

**Tracheal Smooth Muscle Tension**

We recorded transverse smooth muscle tension in an innervated segment (4–6 cm long) of the upper trachea immediately caudal to the larynx. The segment was stretched initially to a baseline tension of 25 g/cm; tension was measured with a force-displacement transducer (Grass model, FT03C, Quincy, Mass.). Details of the preparation have been described previously.⁶

**Control of Carotid Sinus Pressure**

Both carotid sinuses were isolated by ligating major branching arteries in the carotid sinus regions; small unnamed arterial branches were left intact to preserve a small flow of blood through the carotid sinuses. Heparin (200 mU/kg) was injected i.v., and the cephalad ends of the common carotid arteries were cannulated low in the neck and connected to a pressurized reservoir initially containing 500 ml of 6% dextran in Kreb's Henseleit solution, the temperature of which was kept at 37°C. As solution passed from the perfusion reservoir to the carotid sinuses, it was replaced by arterial blood, delivered by a Sarns pump connected to a catheter in the caudal end of a common carotid artery. The volume of blood in the perfusion reservoir was held constant by means of a float connected to a microswitch that turned the Sarns pump off or on.

Mean pressure in the perfusion reservoir was regulated by an inflow of compressed air and a variable leak and was set initially at 100 mm Hg. Pressure pulsations (100 pulses/min) were produced by a Harvard pulsatile blood pump connected by a side port on the inflow line between the perfusion reservoir and the carotid sinuses. Carotid sinus pressure was measured by a Statham strain gauge (P23Gb) connected to a catheter in the left lingual artery.

The occipital arterial branch to the carotid body was not ligated, and we did not intentionally interfere with the blood supply to the carotid body. Hence, we assume that the carotid body chemoreceptors were exposed to the changes in sinus pressure. To determine whether the carotid body chemoreceptors were functional, we injected sodium cyanide (100–200 µg) into the perfusion circuit immediately upstream to the common carotid arteries.

**Protocol**

**Stimulus-response characteristics.** We distended the carotid sinuses with a pulsatile pressure (pulse pressure, 30–50 mm Hg) and examined the changes in tracheal tension, heart rate, and arterial blood pressure evoked by stepwise alterations (usually multiples of 25 mm Hg) in mean sinus pressure above and below a base-line (set-point) of 100 mm Hg, each step usually lasting 1 minute. Tracheal tension, heart rate, and arterial blood pressure were measured during the control period (averaged over 1 minute) and after sinus pressure had been maintained at each level for 1 minute. We also examined the effects of varying pulse pressure at a constant mean sinus pressure.

**Interruption of reflex pathways.** Before examining the tracheal responses to changes in carotid sinus pressure, we always cut the right and left aortic (depressor) nerves to eliminate aortic baroreceptor input. The nerves were freed from the vagal sheath near the nodose ganglion and were identified by the characteristic arterial baroreceptor pattern of their afferent discharge, which was recorded using conventional techniques.

In some experiments, we cut the left and right carotid sinus nerves or cooled them to 0°C. In others, we blocked conduction in myelinated fibers selectively by cooling the sinus nerves to 6°C. The carotid sinus nerves were exposed and placed on the platform (5 mm wide) of a silver cooling device through which alcohol of different temperatures was circulated. The sides and bottom of each platform were insulated with a layer of silicone elastomer (Dow). To reduce thermal gradients, the nerve and adjacent surface of the platform were covered with 4% agar in saline, which upon gelling formed a semisolid layer. The temperature of each platform was measured with a thermistor (Model 729, Yellow Springs Instruments) and was recorded by the Grass polygraph.

In some experiments, vagal afferent pathways to the tracheal segment were interrupted by cutting the superior, recurrent, and pararecurrent laryngeal nerves. We also examined the contribution of parasympathetic cholinergic pathways to the baroreflex-induced tracheal responses by administering atropine sulphate (1 mg/kg i.v.). Because atropine markedly reduced baseline smooth muscle tension, it was impossible to examine the potential ability of noncholinergic mechanisms (engaged by the baroreflex) to further decrease tracheal tension.⁷ We therefore restored baseline tracheal tone to the level existing before atropine by placing a narrow strip of gauze soaked in serotonin solution (100–500 µg/ml serotonin in saline) on the posterior wall of the segment.

**Effect of changes in systemic arterial pressure.** Changing carotid sinus pressure evoked reciprocal changes in systemic arterial blood pressure, which secondarily may have contributed to the observed changes in tracheal tension. Therefore, in some experiments we stabilized systemic arterial blood pressure by connecting a pressurized reservoir of oxygenated blood to a femoral artery. Pressure in the reservoir was set at the dog's control systemic arterial pressure.

Systemic arterial P0₂ and P0₅ in the carotid sinus perfusion reservoir and in the arterial blood pressure stabilizing reservoir were always greater than 100 mm Hg.

**Tracheal responses to stimulation of other afferent inputs.** We compared the reflex changes in tracheal tension evoked by varying carotid sinus pressure with those initiated by stimulating other afferent
endings. The larynx was stroked gently with a fine curved probe inserted through the tracheal opening; pulmonary stretch receptors were stimulated by briefly hyperinflating the lungs (3 \( V_T \)); pulmonary C-fibers were stimulated by injecting capsaicin (10 \( \mu g/kg \)) into the right atrium through a catheter inserted via an external jugular vein; and hind limb muscle afferents were stimulated by injecting capsaicin (2 \( \mu g/kg \)) into a femoral artery through a catheter in the central end of the gracilis artery.  

Analysis of Data

Student's paired \( t \) test was used to determine statistical significance; differences were considered significant if \( p < 0.05 \). Data for groups are expressed as mean ± standard error of the mean.

Results

Stimulus-Response Characteristics

In experiments on 6 dogs, step changes in mean carotid sinus pressure above and below the set-point of 100 mm Hg, while pulse pressure was held constant, evoked reciprocal changes in tracheal smooth muscle tension, heart rate, and arterial blood pressure that were proportional to the change in pressure (Figures 1 and 2; Table 1). Increases in sinus pressure evoked tracheal relaxation; decreases in pressure evoked contraction. The latency of the tracheal response to an increase in sinus pressure was significantly shorter than that to a decrease, relaxation and contraction occurring 4 ± 2 seconds and 8 ± 2 seconds, respectively, after the onset of the pressure steps (\( p < 0.001 \)).

In each of the 6 dogs, tracheal responses were evoked by changes in sinus pressure of 25 mm Hg above and below the set-point (Table 1). In some dogs, effects could be evoked by smaller changes in pressure. Thus, in 2 of 5 dogs, tracheal tension decreased by about 9 g when sinus pressure was increased by 10 mm Hg; in 1 of 3 dogs it increased by 5 g when pressure was reduced by 10 mm Hg. At sinus pressures above 175 mm Hg and below 50 mm Hg, the stimulus.
FIGURE 1. Average changes in tracheal tension (ΔTT, g), heart rate (ΔHR, b/min), and systemic arterial blood pressure (ΔABP, mm Hg) evoked by increasing and decreasing mean carotid sinus pressure (CSP) in 6 dogs (carotid sinus pulse pressure was held constant). Baseline values for tracheal tension, heart rate, and arterial blood pressure at a mean carotid sinus pressure of 100 mm Hg were, respectively, 120 ± 12 g, 178 ± 13 b/min, and 132 ± 5 mm Hg.

Figure 2. Average changes in tracheal tension (ΔTT, g), heart rate (ΔHR, b/min), and systemic arterial blood pressure (ΔABP, mm Hg) evoked by increasing and decreasing mean carotid sinus pressure (CSP) in 6 dogs (carotid sinus pulse pressure was held constant). Baseline values for tracheal tension, heart rate, and arterial blood pressure at a mean carotid sinus pressure of 100 mm Hg were, respectively, 120 ± 12 g, 178 ± 13 b/min, and 132 ± 5 mm Hg.

Table 1. Tracheal Tension, Heart Rate, and Arterial Blood Pressure at Various Levels of Carotid Sinus Pressure

<table>
<thead>
<tr>
<th>CSP (mm Hg)</th>
<th>ΔTT (g)</th>
<th>ΔHR (b/min)</th>
<th>ABP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>30 ± 3</td>
<td>198 ± 14</td>
<td>177 ± 6</td>
</tr>
<tr>
<td>50</td>
<td>27 ± 4</td>
<td>197 ± 15</td>
<td>174 ± 7</td>
</tr>
<tr>
<td>75</td>
<td>10 ± 1</td>
<td>187 ± 15</td>
<td>152 ± 7</td>
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<tr>
<td>100</td>
<td>0</td>
<td>178 ± 13</td>
<td>132 ± 5</td>
</tr>
<tr>
<td>125</td>
<td>-14 ± 4</td>
<td>165 ± 16</td>
<td>109 ± 8</td>
</tr>
<tr>
<td>150</td>
<td>-32 ± 6</td>
<td>150 ± 17</td>
<td>85 ± 9</td>
</tr>
<tr>
<td>175</td>
<td>-50 ± 7</td>
<td>128 ± 19</td>
<td>71 ± 8</td>
</tr>
<tr>
<td>200</td>
<td>-54 ± 8</td>
<td>123 ± 19</td>
<td>64 ± 8</td>
</tr>
</tbody>
</table>

Data (means ± SE) in 6 dogs after carotid sinus pressure (CSP) had been maintained at each level for 1 minute; CSP increased and decreased around a set-point of 100 mm Hg. ΔTT, change in tracheal tension (g) around baseline value (0 g); HR, heart rate; ABP, arterial blood pressure. All changes in tracheal tension were significant (p < 0.05).

(carotid sinus pressure) response (tracheal tension) curve tended to flatten (Figure 2; Table 1).

The changes in tracheal tension invariably persisted until, after 1 minute, carotid sinus pressure was restored to the initial set-point of 100 mm Hg. In most experiments, tracheal tension reached a steady state within 30–40 seconds of the change in sinus pressure (Figure 1A and E); in 2 dogs tension reached a steady state within 1 minute in response to all but the largest step increases and decreases in sinus pressure (e.g., Figure 1C). Tracheal responses did not appear to adapt in 3 dogs even when the duration of the carotid sinus pressure step was increased to 4 minutes (Figure 3).

Reflex changes in tracheal smooth muscle tone were evoked not only by varying mean sinus pressure at a constant pulse pressure but also by varying pulse pressure at a constant mean pressure, the magnitude of the tracheal response being proportional to the change in pulse pressure (Figure 4). Thus, in experiments on 3 dogs, tracheal tension decreased by 12 ± 2 g when a steady (nonpulsatile) sinus pressure was changed to a pulsatile one with a pulse pressure of 30 mm Hg, and it decreased by 26 ± 3 g when a steady pressure was changed to a pulsatile one with a pulse pressure of 60 mm Hg; mean pressure was held at 100 mm Hg throughout. The trachea contracted when the nonpulsatile pressure was restored.

The reflex tracheal responses were accompanied by changes in heart rate and systemic arterial pressure characteristically evoked by changing arterial baroreceptor input (Figures 1–3; Table 1). Tracheal and cardiovascular effects exhibited similar stimulus-response characteristics.

Although we did not attempt to exclude the carotid bodies from the sinus perfusion circuit, in 3 of the 6 dogs injection of cyanide or nicotine into the perfusion circuit was without effect, presumably because blood flow to the carotid bodies had been inadvertently interrupted. In the other 3 dogs, the carotid bodies were assumed to be functional because injection of cyanide or nicotine evoked tracheal contraction, bradycardia, and systemic hypertension. The effects of varying sinus pressure on tracheal tension, heart rate, and systemic arterial pressure were similar in the two groups of dogs.
Changing carotid sinus pressure had little effect on end-tidal PCO₂. Small and varied changes in PCO₂ were sometimes observed as sinus pressure was increased or decreased, but PCO₂ was not significantly different from control once sinus pressure had reached its new steady level (Figure 3).

** Interruption of Reflex Pathways **

The changes in tracheal tension, arterial blood pressure, and heart rate evoked by altering sinus pressure were abolished by cutting the carotid sinus nerves (3 dogs) or by cooling them to 0°C (4 dogs) (Figure 5). Responses abolished by cooling were restored by rewarming the nerves. Baseline tension in the tracheal segment increased in each of 4 dogs when the carotid sinus nerves were cooled while sinus pressure was held at 100 mm Hg (Figures 5 and 6). Contraction began when the nerve temperature reached 7–10°C and was maximal (25 ± 6 g above control) at about 2°C (Figure 6). Heart rate and arterial blood pressure also increased during sinus nerve blockade.

To determine whether carotid baroreceptor (non-myelinated) C-fibers contributed to the baroreflex changes in tracheal tone, in 4 dogs we selectively blocked conduction in (myelinated) A-fibers by cooling the sinus nerves to 6°C (Figure 5). Tracheal relaxation could still be evoked by raising sinus pressure from 100 mm Hg to 200 mm Hg (p < 0.05), the resultant decrease in tracheal tension at 6°C being 30% of that at 37°C; reducing sinus pressure below 100 mm Hg had no effect.

The tracheal responses to changes in sinus pressure were abolished in each of 3 dogs by cutting the superior, recurrent, and pararecurrent laryngeal nerves and in each of 3 dogs by administering atropine (1 mg/kg i.v.). Baseline tension in the tracheal segment decreased by 58 ± 8 g after transection of the laryngeal nerves or cholinergic blockade with atropine. The decrease in baseline tension after atropine was not responsible, per se, for the failure of the tracheal segment to respond to changes in carotid sinus pressure. Topical application of serotonin (100–500 µg/ml) to the posterior wall of the tracheal segment restored baseline tension to the level before atropine in each of
the 3 dogs but did not restore the tracheal responses to changes in sinus pressure. Hence, the changes in tracheal tension evoked by the carotid sinus baroreflex before administration of atropine were mediated by a cholinergic pathway.

It is conceivable that baroreflex-induced changes in pressure or volume in the pulmonary circulation or heart caused changes in firing of pulmonary and cardiac vagal afferents that contributed secondarily to the tracheal responses. Cutting the lower cervical vagus nerves in 2 dogs reduced, but did not abolish, the tracheal responses. After vagotomy, the baroreflex-induced changes in tracheal smooth muscle tone in these 2 dogs were 66% and 53% of those when the vagi were intact, a reduction that can reasonably be attributed to interruption of vagal bronchomotor fibers destined for the recurrent and pararecurrent nerves supplying the upper trachea.

**Effect of Changes in Systemic Arterial Pressure**

We examined the possibility that the reflex changes in systemic arterial pressure evoked by altering sinus pressure may have been responsible, at least in part, for the observed changes in tracheal tension. In 3 dogs, we stabilized systemic arterial blood pressure by connecting a femoral artery to a pressure compensator. The tracheal responses under these conditions (Figure 7B and D) were not significantly different from those when systemic pressure was allowed to vary (Figure 7A and C).

**Tracheal Responses to Stimulation of Other Afferent Inputs**

The changes in tracheal tension evoked by the carotid baroreflex (Table 1) were of a comparable magnitude to those produced by stimulating a number of other afferent inputs that are known to influence airway smooth muscle tone. In experiments on 6 dogs, stimulating pulmonary stretch receptors by hyperinflating the lungs (3 V T) decreased tracheal tension by 51 ± 9 g; stimulating hindlimb muscle afferents by injecting capsaicin (2 μg/kg) into a femoral artery decreased tension by 33 ± 12 g; stimulating pulmonary C-fibers by injecting capsaicin (10 μg/kg) into the right atrium increased tension by 34 ± 6 g; and stimulating the larynx with a fine probe increased tension by 38 ± 9 g.

**Discussion**

Our results demonstrate that graded changes in carotid sinus perfusion pressure evoke reciprocal changes in tracheal smooth muscle tension, increases in sinus pressure producing tracheal relaxation, and decreases in pressure tracheal contraction. By blocking the appropriate nervous pathways, we established that the tracheal responses were dependent on a reflex, the afferent and efferent arms of which were in the...
carotid sinus and laryngeal nerves, respectively. The relation between sinus pressure and tracheal tension, like that between sinus pressure and heart rate and between sinus pressure and systemic arterial blood pressure, displayed the characteristic sigmoidal baroreflex stimulus-response curve operating around a normal arterial pressure set-point of 100 mm Hg and tending to reach a plateau at sinus pressures above 175 mm Hg and below 50 mm Hg — a relation generally similar to that between sinus pressure and baroreceptor impulse frequency.\textsuperscript{9} Tracheal responses were not transitory but persisted for the duration of the imposed change in sinus pressure. In some dogs, the reflex appeared to have a low threshold, the trachea responding to changes in sinus pressure as small as 10 mm Hg. For these and other reasons (see below), we conclude that carotid sinus baroreceptors were responsible for the tracheal responses observed.

The possibility that the carotid bodies played a part in the reflex responses at low sinus pressures must be considered. Because carotid chemoreceptors in cats are stimulated when perfusion pressure falls below 60–65 mm Hg,\textsuperscript{10,11} and their stimulation evokes reflex bronchoconstriction,\textsuperscript{4,12} they may have contributed to the effects of reducing sinus pressure from 75 to 50 mm Hg in our experiments — at least in those dogs with functional carotid bodies. Carotid chemoreceptor firing is unaltered by changing blood pressure between 100 and 75 mm Hg\textsuperscript{10,11} and is unlikely to have decreased significantly in our experiments when sinus pressure was increased above 100 mm Hg because Po\textsubscript{2} in the perfusion circuit was always kept above 100 mm Hg.
Hg, and firing would already be very low. Carotid chemoreceptors clearly played no part in the tracheal responses evoked either by changing a pulsatile sinus pressure to a nonpulsatile one at the same mean pressure or by changing sinus pressure in the dogs whose carotid bodies were inactive. Aortic chemoreceptors undoubtedly had little part in the effects described here. The aortic nerves were cut, and alterations in the surviving aortic chemoreceptor input secondary to the reflex changes in arterial blood pressure would be likely to evoke tracheal responses opposite in sign to those observed.

Stimulation of central chemoreceptors by local changes in PCO₂ or pH secondary to changes in cerebral blood flow could not account for the tracheal responses observed. Carotid baroreceptor stimulation does not alter total or regional cerebral blood flow when cerebral perfusion pressure is held constant, and in our experiments, the tracheal responses observed when systemic blood pressure was controlled were not different from those when systemic pressure was allowed to vary. Certainly, although autoregulation maintains cerebral blood flow constant over a range of perfusion pressures between approximately 70 mm Hg and 150 mm Hg, systemic arterial pressure passed out of this autoregulatory range in our experiments when sinus pressure was raised to 200 mm Hg and reduced to 50 mm Hg (Table 1). Hence, cerebral blood flow may have decreased in the former instance and increased in the latter. However, the resultant local changes in PCO₂ and pH acting on the central chemoreceptors would be likely to cause tracheal responses opposite in sign to those actually observed in our experiments (see Mitchell et al). Impulses arising from carotid sinus baroreceptors travel in myelinated (A-) and nonmyelinated (C-) fibers. A-fiber baroreceptors are active at a sinus pressure of 100 mm Hg, and their firing increases and decreases as pressure is varied around the set-point. C-fiber baroreceptors have a much higher threshold and are virtually inactive at a pressure of 100 mm Hg. Hence, both the tracheal contraction produced by decreasing sinus pressure and that produced by cooling the sinus nerves when sinus pressure was held at 100 mm Hg can be attributed to reduction of A-fiber baroreceptor input. However, C-fiber baroreceptors may have contributed to the tracheal relaxation produced by increasing sinus pressure, and, indeed, relaxation could still be evoked after the sinus nerves had been cooled to 6°C, a temperature at which conduction in carotid baroreceptor A-fibers is blocked selectively.

We do not know whether aortic baroreceptors modulate tracheal smooth muscle tone, but it seems reasonable to assume that their effects, if any, would be similar to those of carotid baroreceptors. In our experiments, most aortic baroreceptor input was abolished by cutting the aortic nerves, and changes in firing of baroreceptor fibers in the main vagus nerve, resulting from the reflex changes in systemic arterial pressure, would be likely to oppose the primary reflex effects of the carotid baroreceptors. Probably the influence of arterial baroreceptors on the airways would be even greater if both carotid and aortic baroreceptors were functional and subjected to the imposed changes in blood pressure.

The reciprocal effects of baroreceptor stimulation on vagal cholinergic control of the heart and airways — increasing cardioinhibitory drive and decreasing bronchoconstrictor drive — correlate well with the results of effenter vagal studies in dogs and cats. Thus, activity in vagal effenter fibers to the heart varies directly with blood pressure, whereas activity in vagal bronchomotor fibers varies inversely. The reciprocal engagement of these two vagal effenter pathways by the carotid baroreflex is in contrast to their simultaneous engagement by stimulation of carotid body chemoreceptors, laryngeal receptors, and pulmonary and bronchial C-fibers.

Although Nadel and Widdicombe found the carotid baroreflex to have only a minor influence on tracheal smooth muscle, our results indicate that it is capable of evoking tracheal relaxation and contraction comparable in magnitude to the changes triggered, in the same dog, by stimulating laryngeal receptors, pulmonary stretch receptors, pulmonary C-fibers, and hind limb muscle afferents — afferent inputs that are generally agreed to have conspicuous bronchomotor effects.

In conclusion, our results provide strong support for the notion that arterial baroreceptors exert a tonic influence on the upper airways, increasing and decreasing baseline smooth muscle tension as arterial blood pressure varies around the normal set-point. These effects parallel the ventilatory responses induced in spontaneously breathing dogs by changing baroreceptor input. Changes in breathing and bronchomotor tone usually go hand in hand, stimulation of breathing being associated with bronchoconstriction and inhibition of breathing with bronchodilation — an association reflecting the close interaction of the central mechanisms controlling breathing and airway caliber.

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References


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