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 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 
nervated 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 trans-
ducer (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. Hepe-
arin (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 reser-
vior 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 perfu-
sion reservoir to the carotid sinuses, it was replaced by 
blood arterial, delivered by a Sarns pump connected to 
a catheter in the caudal end of a common carotid ar-
tery. 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 regu-
lated 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 Har-
vard 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 as-
sume that the carotid body chemoreceptors were ex-
posed to the changes in sinus pressure. To determine 
whether the carotid body chemoreceptors were func-
tional, we injected sodium cyanide (100–200 μg) or 
nicotin bitartrate (10–20 μg) into the perfusion circuit 
immediately upstream to the common carotid arteries.

**Protocol**

**Stimulus-response characteristics.** We distend-
ed 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 arte-
rial blood pressure were measured during the control 
period (averaged over 1 minute) and after sinus pres-
sure had been maintained at each level for 1 minute. 
We also examined the effects of varying pulse pressure 
at a constant mean sinus pressure.

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

In some experiments, we cut the left and right carot-
id 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 gradi-
ents, the nerve and adjacent surface of the platform 
were covered with 4% agar in saline, which upon gell-
ing 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 efferent pathways to the 
tracheal segment were interrupted by cutting the supe-
rior, recurrent, and pararecurrent laryngeal nerves. We 
also examined the contribution of parasympathetic 
cholinergic pathways to the baroreflex-induced trache-
al responses by administering atropine sulphate (1 
mg/kg i.v.). Because atropine markedly reduced base-
line smooth muscle tension, it was impossible to exam-
ine the potential ability of noncholinergic mechanisms 
(engaged by the baroreflex) to further decrease trache-
al 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 pres-
sure.** Changing carotid sinus pressure evoked recipro-
cal changes in systemic arterial blood pressure, which 
secondarily may have contributed to the observed 
changes in tracheal tension. Therefore, in some experi-
ments 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 P₀₂ 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 pres-
sure 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 V̇); pulmonary C-fibers were stimulated by injecting capsaicin (10 μg/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 μg/kg) into a femoral artery through a catheter in the central end of the gracilis artery.8

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 ± 1 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](http://circres.ahajournals.org/) Changes in tracheal tension (TT), heart rate (HR), and systemic arterial blood pressure (ABP) evoked by graded alterations in mean carotid sinus pressure (CSP) above (Parts A–C) and below (Parts D–F) 100 mm Hg in one dog. Carotid sinus pulse pressure was unaltered.
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>HR (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>
</tr>
<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).

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 $P_{CO_2}$. Small and varied changes in $P_{CO_2}$ were
sometimes observed as sinus pressure was increased or
decreased, but $P_{CO_2}$ 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 pres-
sure, 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 ($25 \pm 6$ 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 cool-
ing the sinus nerves to 6°C (Figure 5). Tracheal relax-
ation could still be evoked by raising sinus pressure
from 100 mm Hg to 200 mm Hg ($p<0.05$), the resul-
tant 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 superi-
or, 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 de-
creased by $58 \pm 8$ g after transection of the laryngeal
nerves or cholinergic blockade with atropine. The de-
crease in baseline tension after atropine was not re-
sponsible, per se, for the failure of the tracheal seg-
ment to respond to changes in carotid sinus pressure.
Topical application of serotonin (100–500 $\mu$g/ml) to
the posterior wall of the tracheal segment restored
baseline tension to the level before atropine in each of

![Figure 3](http://circres.ahajournals.org/)

Sustained changes in tracheal tension, heart rate, and systemic arterial blood pressure evoked by a prolonged increase (Panel A) and decrease (Panel B) in mean carotid sinus pressure. $P_{CO_2}$, tidal $CO_2$; other abbreviations as in Figure 1.
Circulation Research  Vol 60, No 3, March 1987

**ABP** (mm Hg)

**HR** (b/min)

**TT** (g)

**CSP** (mm Hg)

**FIGURE 4.** Changes in tracheal tension, heart rate, and arterial blood pressure evoked when a constant carotid sinus pressure was replaced by a pulsatile pressure, mean carotid sinus pressure being held constant at 100 mm Hg. Pulse pressure in carotid sinus: Part A, 30 mm Hg; Part B, 60 mm Hg. Abbreviations as in Figure 1.

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 VT) 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.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,10,11 and their stimulation evokes reflex bronchoconstriction,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 Hg10,11 and is unlikely to have decreased significantly in our experiments when sinus pressure was increased above 100 mm Hg because Po2 in the perfusion circuit was always kept above 100 mm

Figure 6. Increase in tracheal tension produced by cooling the carotid sinus nerves to 0°C. CSNT, carotid sinus nerve temperature. Other abbreviations as in Figures 1 and 3.

Figure 7. Reflex changes in tracheal tension and heart rate could still be evoked by changing mean carotid sinus pressure when systemic arterial blood pressure was stabilized by a compensator. Parts A and C, before, and Parts B and D, after a pressurized reservoir of blood was connected to a femoral artery; reservoir pressure was set at the initial systemic arterial blood pressure. Abbreviations as in Figure 1.
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 efferent vagal studies in dogs and cats. Thus, activity in vagal efferent fibers to the heart varies directly with blood pressure, whereas activity in vagal bronchomotor fibers varies inversely. The reciprocal engagement of these two vagal efferent 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 bronchodilatation — an association reflecting the close interaction of the central mechanisms controlling breathing and airway caliber.

Acknowledgments

We thank Albert Dangel and Ronald Brown for technical assistance and Rolinda Wang for typing the manuscript.

References


**Key Words** • arterial baroreceptors and airways • blood pressure influence on airways • carotid sinus reflexes • reflex control of upper airways
Carotid sinus baroreceptors modulate tracheal smooth muscle tension in dogs.
H D Schultz, T E Pisarri, H M Coleridge and J C Coleridge

Circ Res. 1987;60:337-345
doi: 10.1161/01.RES.60.3.337

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circres.ahajournals.org/content/60/3/337

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation Research is online at:
http://circres.ahajournals.org/subscriptions/