et al. (*15*), they are space-variant single- and multiple-channel models. Both models allow for increases in receptive field sizes with increasing eccentricity, but they differ in their allotment of the number of receptive field sizes at any one particular eccentricity; the single-channel model allows for one and the multiple-channel model for several sizes.

Our data show unequivocally that at any one area of retina, brisk ganglion cells of the same kind are closely matched in their spatial properties. It is only when we consider cells of different classes, that we see a range in sizes of neighboring concentric receptive fields with the upper and lower bounds being determined by brisk-transient and brisk-sustained units, respectively (*7*).

It seems reasonable to suggest that visual stimuli used in detection tasks may preferentially stimulate one kind of ganglion cell rather than another and therefore lead to unique thresholds. For example, Wilson (*16*) has shown in humans that by presenting line stimuli with temporal modulations which maximize either sustained or transient responses, two neural mechanisms can be detected at each retinal position. The mechanism characterized by the sustained response property responds to higher spatial frequencies than does the mechanism characterized by transient response properties, a finding in agreement with our neurophysiological findings in the cat.

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10. A hyperbolic function of the form $y - y_0 = c/(|x| - x_0)$, although somewhat arbitrarily selected, describes both the decline in cut-off frequency and the increase in the size of the receptive field center with increasing eccentricity.
11. As the bars of a coarse grating are moved across the receptive field of a ganglion cell, a variation in the response can be heard in the spike discharge as amplified through a loudspeaker. As the grating frequency is increased, but with the velocity adjusted to give a constant temporal modulation, the amplitude of the response grows weaker until an auditory threshold is reached. We found this threshold when averaged responses showed a peak-to-peak amplitude of about 25 spikes per second.
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The Central Origin of Efferent Pathways in the Carotid Sinus Nerve of the Cat

Abstract. *The application of horseradish peroxidase to the central cut end of the carotid sinus nerve of the cat produced retrograde labeling of neurons in the ipsilateral medulla in the region of the nucleus ambiguus at anterior-posterior coordinates −8 to −10.5. These data coupled with previous electrophysiological observations suggest that the nucleus ambiguus may be the origin of an efferent inhibitory pathway to the carotid body.*

The carotid sinus nerve contains efferent fibers as well as chemoreceptor and baroreceptor afferents (*1*). Activation of the efferent pathway inhibits chemoreceptor firing (*2*). These observations prompted the hypothesis that efferent axons make excitatory synaptic contacts in the carotid body with type 1 cells, which in turn release an inhibitory substance that depresses afferent activity (*3*). It has also been suggested that efferent activity produces in the carotid body vascular changes that indirectly alter chemoreceptor firing (*4, 5*).

The origin of the sinus nerve efferent pathways is uncertain. Initial electron microscopy (*3*) revealed that efferent contacts with type-1 cells degenerate after intracranial section of the glossopharyngeal nerve, which indicates that the efferent fibers originated in the brainstem. However, subsequent investigations could not confirm these findings (*6*).

We used the horseradish peroxidase (HRP) tracing technique to study the origin of efferent fibers in the carotid sinus nerve of the cat. Experiments were conducted on 12 adult cats anesthetized with Dial-urethan. The carotid sinus nerves were unilaterally or bilaterally exposed through a midline incision in the neck. The nerve was sectioned distally near the carotid sinus and placed in a short length (10 mm) of polyethylene tubing, which was filled with HRP solution (Sigma type IV, 25 percent solution in distilled water) and then sealed with petrolatum. The nerve remained in contact with HRP for 4 to 6 hours, after which the solution was removed and the incision closed. After a transport time of 24 to 40 hours, the animal was deeply anesthetized and perfused first with saline and then fixative (1 percent glutaraldehyde and 1 percent paraformaldehyde in 0.05*M* phosphate buffer). The brainstem and petrosal ganglia were removed and stored for 12 to 24 hours in the fixative and then transferred to 5 percent sucrose in phosphate buffer for at least 48 hours before being sectioned. The brainstem was cut on a freezing microtome in 42-μm sections, mounted serially on slides and processed for HRP according to the benzidine method (*7*). In some experiments sections were also counterstained with thionin. Sections were examined with darkfield illumination or with Nomarski optics.

Labeled cells were found in 7 of 12 experiments. They were located in the medulla ipsilateral to the site of HRP application between 10.5 and 8.0 mm posterior to bregma. In individual experiments the cells were distributed over a range from 0.75 to 1.6 mm. The number of labeled cells varied from 3 to 66 ($\bar{X} = 22$, $N = 7$). The cells were oval or spindle-shaped, 10 to 20 μm in diameter and lo-

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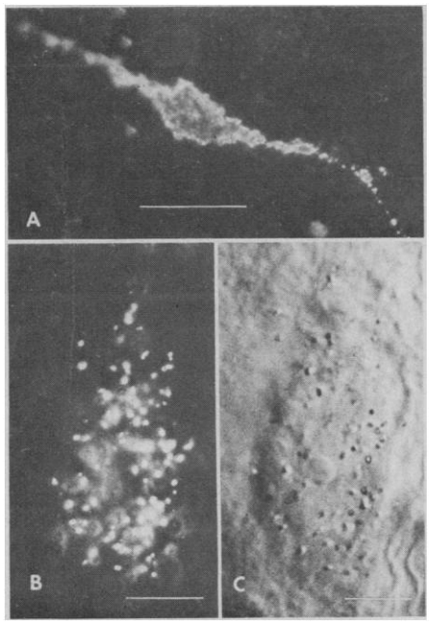

Fig. 1. Neurons in the nucleus ambiguus labeled after an application of HRP to the central cut end of the ipsilateral carotid sinus nerve. (A) An intensely labeled cell under darkfield illumination. A lightly labeled cell is shown twice in (B) under darkfield illumination and in (C) with Nomarski optics. Calibration bars: 50 μm in (A) and 10 μm in (B) and (C).

cated in the rostral nucleus ambiguus (retrofacial nucleus) (*8*) and the adjacent reticular formation (nucleus parvocellularis) (Figs. 1 and 2). A few cells were also observed in a more dorsal position in the reticular formation. Usually only one or two cells were noted in one 42-μm section.

Application of HRP to the glossopharyngeal nerve peripheral to the site at which the carotid sinus nerve emerged resulted in the labeling of a larger number of cells ($\bar{X}$ = 160, N = 2) which occurred between 9 and 7.4 mm posterior to bregma and which were more widely distributed in the transverse plane than cells labeled from the carotid sinus nerve. There appeared to be at least two groups of glossopharyngeal efferents. One group of multipolar cells (20 to 50 μm), which had the appearance of motoneurons, were located in the region of the rostral nucleus ambiguus. Smaller oval or spindle-shaped cells (10 to 25 μm in diameter) were located in the more dorsal part of the reticular formation (Fig. 2). A similar distribution of labeled cells was noted when the entire glossopharyngeal nerve (including the carotid sinus nerve) was exposed to HRP.

After HRP was applied to the carotid sinus nerve, the number of labeled afferent neurons in the petrosal ganglion varied in different preparations from 175 to 613 ($\bar{X}$ = 367, N = 5). Considerably larger numbers of labeled petrosal ganglion cells were noted after HRP was applied to the glossopharyngeal nerve, but the exact cell counts were not obtained. Axons and afferent nerve terminals in the medulla were also labeled with HRP. Axons were evident as lines of fine granules within the projections of the carotid sinus nerve and glossopharyngeal nerve (Fig. 3). The HRP reaction product, presumably contained in afferent terminals, was also present in the nucleus tractus solitarius.

Thus, we have demonstrated, for what we believe to be the first time, axonal projections from neurons in the central nervous system to the carotid sinus nerve of the cat. Another recent HRP study failed to detect neurons in the brain stem after HRP was applied to the carotid sinus nerve (*9*). This negative result probably resulted from the use of the diaminobenzidine method for HRP, which, in comparison with the benzidine procedure we used, produces a less intense reaction product. The benzidine procedure was also sufficiently sensitive to detect anterograde transperikaryal transport of HRP in the central projections of sinus nerve afferents (Figs. 2 and 3). Similar anterograde transport of HRP has been demonstrated in the spinal cord, where it has been used to study the central projections of afferents in the pelvic nerve (*10*) and sciatic nerve (*11*).

One possible interpretation of our findings is that the labeled cells were centrally located afferent neurons analogous to sensory cells in the mesencephalic nucleus of the trigeminal nerve. The labeled cells, however, did not have the typical unipolar appearance of sensory cells, so it seems reasonable to assume that they are efferent neurons. Although the termination and function of this efferent system remains to be established, we hypothesize that it represents the efferent inhibitory pathway to the carotid body identified in previous physiological experiments (*2*).

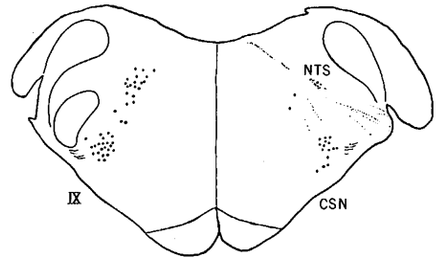

Fig. 2. Location of HRP-labeled neurons and axons in the medulla after application of HRP to the central cut end of the carotid sinus nerve (*CSN*) and the cut end of the glossopharyngeal nerve (*IX*). Data for each nerve were obtained in different experiments. The CSN side shows 17 labeled neurons observed in a series of 30 sections (1.2 mm) located primarily in the region of the nucleus ambiguus. Labeled axons and axon terminals were found, respectively, in the central projection of the CSN and the nucleus tractus solitarius (*NTS*). The IX side shows the distribution of 43 labeled neurons observed in a series of eight sections (336 μm) occurring in the region of the nucleus ambiguus and in the dorsal reticular formation.

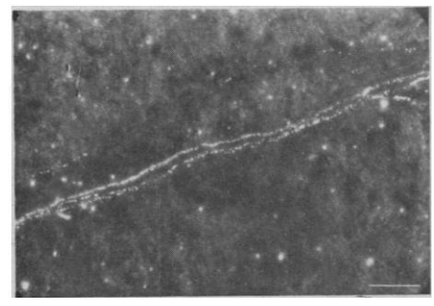

Fig. 3. Darkfield photomicrograph of HRP reaction product in axons in the medulla after the application of HRP to the ipsilateral carotid sinus nerve. Calibration bar, 40 μm.

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